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SECTION OF NEUROLOGY.

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Section of Neurology.[1]

President—WILLIAM ALDREN TURNER, C.B., M.D.

PRESIDENT'S ADDRESS.

The Influence of Psychogenic Factors in Nervous Disorders.

By WILLIAM ALDREN TURNER, C.B., M.D.

IT is not my purpose on taking over the honourable office of President of the Section of Neurology for the current Session to give you in the address, which it is customary for the President to deliver on this occasion, a retrospective account of the progress of Neurological Science or to refer to the part which members of this Section have taken therein. We cannot fail, however, to honour the names of many members of the Section who are no longer with us, nor withhold our tribute of respect to the memory of those whom we have come to regard as amongst the great masters of British neurology.

EPOCHS IN NEUROLOGY.

It might not, however, be without interest or profit to reflect for a few moments on three outstanding circumstances which have determined a notable advance in the progress of our specialty.

The localization of cerebral function began to attract general attention both within and outside the profession some years before I became a student of medicine. The researches were epoch-making and prepared the way to a great outburst of enthusiasm for neurological investigation and research. The impetus thus given to neurology by the new methods of work may be likened to the discovery of a rich mineral region of the earth's surface. As the prospectors rush for gold so numbers of well-trained and enthusiastic investigators set to work to ascertain the structure and functions of the brain and spinal cord. In a few years the anatomy of the central nervous system became unfolded in its complexity and richness of structure. Clinical observations were correlated with anatomical and pathological knowledge, whereby the organic diseases of the brain and spinal marrow became based on sure and certain foundations. The final decade of last century and the early years of the present one were the most fertile and prolific in work of this nature, and it is not improbable that the high water mark of anatomico-pathological investigation into nervous diseases was reached at this time.

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[1] At a meeting of the Section, held October 9, 1919.

Although the operation of lumbar puncture was described so long ago as 1891, there was no general resort to its practical application in clinical work until comparatively recent years. Its present day extensive and routine employment in nervous and mental diseases, providing thereby the means of a bacteriological and serological examination of the cerebro-spinal fluid, as an aid to diagnosis, is one of the most valuable later day advances in neurological medicine. Largely in consequence of this, the infective or toxic origin of many organic nervous diseases is now well-established. There is, however, one chronic progressive disease, very common in this country, which for some reason has resisted efforts made to ascertain its causation. I refer to disseminated sclerosis, the pathogenesis of which is still as obscure as when its symptoms and clinical course were described more than thirty years ago.

It seems hardly necessary for me to do more than mention the value of the researches, which have been made during later years upon the functions of the endocrine glands and the important part played by their secretions in the ætiology of disease, and more especially of those disorders standing in close relationship to the nervous system. The influence of the emotions in stimulating, arresting or modifying endocrine secretion, and the bearing of this on the production of functional disorder, has been brought into special prominence by the circumstances of the War. It is not unlikely, however, that the study of the war neuroses from the psychological view-point, which has been the practice of our neurologists, may have obscured the value of the influence of endocrine agency in the origin and maintenance of many of their symptoms.

In the few remarks which I shall address to you this evening, the psychological basis of functional nervous disorder will be outlined. Although trained in the physical side of nervous diseases, and slow perhaps to adjust my outlook to a serious modification of it, I venture to think that another advance is in progress in the psychological study, examination and treatment of the functional or psychogenic disorders of the nervous system. The pioneers have been at work for some years and the ground has been prepared for the benefit of those who follow. The initiative has come largely from Continental and American workers, but the War has been the means of introducing a number of British neurologists and others into this most interesting and fascinating branch of neurology.

Clinically-applied Psychology.

One obvious outcome of the War has been to diffuse an interest, both medical and lay, in the psychogenic origin of the neuroses and psychoses arising under war conditions. A large number of medical officers, while serving in the Army, received an *ad hoc* training in the principles of psychology with a view to their practical application in the treatment of the psychoneuroses of war. Many neurologists have awakened to find that the study of the physical side only of functional nervous disorder is not sufficiently satisfying or productive. Scores of victims of hysteria and allied disorders have found that the treatment of their disability by psychological methods has given greater and more permanent relief than happened by physical means alone.

In ways such as these the War has given a stimulus to the development of a school of clinically-applied psychology—a semi-dependent specialty which will play in the future an active and (under suitable conditions of study and practice) wholesome part in the elucidation and treatment of functional nervous disorders. We require for this purpose specialist physicians, who have had also a training in nervous and mental diseases, so that their psychological knowledge may be used in treatment after a careful differential diagnosis has been made.

We want the subject studied in a scientific spirit and by scientific methods, so that all factors bearing upon causation may be defined and their influence determined. We want the proper value and usefulness of all methods of psychological treatment tested and proved, so that any stigma or suspicion of their legitimacy may be removed or corrected.

It is not improbable that fresh light will be thrown upon the causation of the psychoneuroses and that problems surrounding their origin may be released from that close association with psychopathia sexualis, which has been so minutely sought for and found by its more enthusiastic advocates—a relationship which recent experiences have not entirely confirmed.

It seems to me that it will be incumbent upon the existing hospitals for nervous diseases and upon neurological sections in general hospitals, to consider this subject as an integral part of their work and to make provision for the treatment of their patients upon these lines.

The experience of the War has shown the great value of segregation in the treatment of the psychoneuroses, which should be in the hands of specially qualified physicians. Treatment by psychical measures goes far back in the history of human ailments, and the influence of some kind of suggestion in the treatment of nervous disorder has been admitted from time immemorial. "In a sense mystic medicine is psychical medicine, though the converse is not true. The psychical element is discoverable in the incantations of medicine men, in the appeals to omens and oracles, in the resort to healing shrines, and in the ministrations of the Christian Scientist. The psychical medicine of to-day, however, is that in which the use of mental influence is resorted to on the same scientific lines as is the use of water, medicines, electricity or the surgeon's knife" (C. K. Mills).[1]

FAILURE OF PSYCHOLOGICAL ADAPTATION.

Contemporary psycho-pathologists maintain, and their view generally is accepted, that the psychoneuroses and some of the psychoses are "reactive" disorders, that is to say, they are the reactions or responses to a failure of psychological adaptation or re-adjustment to environment. Associated and intimately blended with the process of re-adjustment is repression usually of a painful emotion or instinctive tendency arising from a conflict with antagonistic motives, such as honour, conscience, duty, or the traditional sentiments and ideals of society.

Mental conflict and repression are in themselves not harmful if re-adjustment takes place normally. It is only under conditions in which repression fails to adapt the individual to his surroundings that harm results, and this is more likely to occur in those whose nervous or mental make-up favours abnormal resolution, or whose nervous and mental health has been lowered by fatigue, exhaustion or illness.

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[1] C. K. Mills, *Monthly Cyclopedia*, July, 1908.

The psychological history of each one of us will reveal just how far we have been able to adapt our mental outlook to the commonplace and accidental circumstances of our lives. The occurrence of a nervous or mental breakdown is no mere haphazard circumstance. It occurs on the occasion when a new psychological adaptation is required. It is the individual's response or reaction to some experience with which he is confronted or to a difficult situation in which he finds himself. Abundant illustrations of this teaching have been provided by the circumstances of the War, and most of us can recall from experience confirmatory evidence of this doctrine.

WAR AND PEACE-TIME NEUROSES.

In the comparative study of the psychoneuroses of war and of peace-time, the observed reactions have shown no material qualitative difference unless it be some exaggeration of the symptoms or an enhanced picturesqueness in the clinical picture of the war hysterias. In the anxiety conditions and in some of the psychoses a very definite war colouring has been given to the symptoms. Under the conditions of military service the mental conflicts are relatively simple. Personal feelings, opinions and habits are at variance with disciplinary and other methods contingent upon training and camp-life. In the field the instinct of self-preservation comes into immediate and urgent conflict with ideas of competency, honour and duty. Those writers who have studied the subject without prejudice have shown that there is little evidence in favour of the production of the war neuroses by the activity of repressed sexual complexes.

In civilian life the circumstances under which the patient has broken down may appear trivial or insufficient to account for the failure of adaptation and the development of a neurosis or mental disorder. But the mental conflict may be of a deep-seated and absorbing kind, and in some cases is associated with painful emotions of an ethical, social or moral character, not infrequently associated with the instinct of reproduction and repressions connected therewith.

CONTRIBUTORY PHYSICAL CAUSES.

Although the psychogenic element is far reaching and all important in the direct causation of the psychoneuroses, the contributory influence of physical causes should not be overlooked or neglected. Adverse physical conditions, such as fatigue, overwork, loss of sleep, debilitating illness or fever, or even a surgical operation prepare the ground by impairing the normal metabolic processes. They lead to loss of body weight, and make the patient less able to repress or withstand the encroachments of morbid ideas and feelings of incompetency, unworthiness and self-reproach.

The additional influence of a strong emotional shock and, in the case of many of the war neuroses, of a cerebral concussion or commotion from shell explosion, is sufficient to determine the more acute and disabling symptoms of the breakdown.

FAILURE OF ADAPTATION—TYPES OF REACTION.

We may now consider briefly the *varieties of reaction*, which attend upon a failure of psychological adaptation. They are of several kinds —some are purely neurological (using this term in the sense of a functional nervous disorder or psychoneurosis); others cross the threshold into the sphere of psychiatric medicine.

Hysterical Reactions.

Probably the most common, because the least complex, is the hysterical type of reaction. The person of hysterical temperament solves his mental conflict by a crude dissociation, in which (according to the Freudian doctrine) the emotional element of a painful idea is repressed into the subconscious and becomes "converted" into a sensori-motor symptom, such as a paralysis, contracture, anæsthesia or tic-like movement. This solution of a difficulty would appear to be simple; forgetting being easy, the painful memories are actively put aside, repressed and split off from the main personal consciousness.

The experience of the War, however, would appear to show that all cases of hysterical sensori-motor symptoms are not due to repression and conversion. Some would seem to have arisen directly under the influence of suggestion and to be capable of cure by a well-directed and convincing psychotherapy consisting in the main of suggestion, persuasion and re-education. It would appear, moreover, as if too much importance has been placed by some medical officers upon the therapeutic influence of the "lightning cure" as a means of permanent restoration of health. There is a freely expressed opinion that many cases of hysterical paralysis and contracture have been treated as if they were examples of "suggestion hysteria," when in essence they were examples of the "conversion" disorder.

In place of a sustained sensori-motor abnormality, the hysterical reaction may be of a convulsive character, and may exhibit all grades of severity from a simple hysterical attack up to the complex seizure of major hysteria. These attacks are due to repressed memories, which possess an intensely emotional colouring. They come up into consciousness from time to time in the form of an apparently irrational emotional discharge. Their recurrence in some cases and on some occasions, is due probably to an association often unrecognized by the patient. In other cases they may be a purely automatic discharge, as in the case of many epileptic seizures. The mental content of the attack may be brought to the surface by light hypnosis under the influence of which it may be analysed and cleared up with the subsequent cure of the fits.

Functional *amnesia*, or its substitutes, stupor, confusion and fugue, form the most profound degree of hysterical dissociation. The frequency with which it preceded and out of which emerged the common forms of hysterical reaction, was one of the most remarkable associations of the more severe and persistent types of war psychoneurosis, especially in cases which had been buried or submitted to the influence of serious shell explosion or shock.

A type of case, very common under war conditions, is one in which the physical manifestations of fear-emotion persist with apparent loss of memory of the events which originated and accompanied the emotion. This type is one in which disturbances of speech, tremors, tachycardia and symptoms of a reaction of the sympathetic nervous system may persist for an indefinite time. As Myers[1] has pointed out, the claim may be fairly put forward that when a patient has recovered from a state of stupor, but is still amnesic in regard to some of the experiences through which he has passed, the restoration of the normal personality is apparent rather than real.

W. Brown[2] states that in these cases the symptoms may be cured rapidly by bringing up the lost memory with hallucinatory vividness under light hypnosis. The revived memory then automatically links up with the physical symptoms of the emotion, at first intensifying the symptoms (abreaction), which then disappear quickly.

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[1] Myers, *Lancet*, September 9, 1916.

[2] W. Brown, *Lancet*, May 17, 1919.

Light Hypnosis.

It has been argued that by means of hypnotic suggestion or even definite hypnosis painful experiences and their memories may be driven below the threshold of consciousness, and that in this way these agents may accentuate rather than relieve the suffering of the patient. By the use of hypnotism in the manner just described, no such outcome is anticipated, as by this means the memory is restored and integrated with the existing physical symptoms. This method of treatment has been extensively carried out in some of the neurological hospitals with such satisfactory and apparently permanent results, that the practice would appear to be of great therapeutic value. The type of case referred to would seem to differ from the true conversion hysterias to the extent that there is no real conversion of emotion into physical innervation; on the contrary, the psychical side of the emotional experience disappears from consciousness while the physical side persists in diminished intensity.

The recognition of functional amnesia is the key to the whole question of hysterical dissociation. To permit of full recovery, it is essential that it should be cleared up, either by light hypnosis as a form of analysis or by the longer process of psychological analysis. It is a matter of practical experience that patients presenting an hysterical amnesia are easily hypnotized and suffer no ill-effects therefrom. Hypnotism also is a ready means of distinguishing amnesia of an hysterical character from that due to physical concussion of the brain (concussio cerebri). In the latter condition it is improbable that the memory can be recalled, while in cases in which a mixed type of amnesia has been present, the functional variety only has been cleared up under hypnosis.

Reactions Characterized by Anxiety.

States of anxiety or anxiety neurasthenia constitute another type of reaction following upon a failure of psychological adaptation. In this condition, according to the Freudian doctrine, the emotional side of a painful idea or experience is repressed and dissociated as in hysteria, but is not converted into a physical symptom. On the contrary, it is shifted along association paths and becomes linked up with an apparently innocuous idea or system of ideas. In this way the anxieties, phobias and obsessions are developed.

Anxiety states and anxiety depression were amongst the most common varieties of the war neuroses. They occurred alone or in conjunction with hysterical symptoms. Their psychopathology has been variously described, but the picture drawn by many writers upon the war neuroses of the development of anxiety amongst officers on active service, do not fail to impress one with the nature and variety of the mental conflicts and repressions underlying the onset and cause of the disability. In mild types of the disorder, the anxiety is diffused and leads to symptoms of mental pre-occupation and worry. In the more severe types, the anxiety takes the form of a compulsion neurosis, in which there is a tendency for the patient to act or think in an unreasonable way.

The common prominent symptoms are a feeling of anxiety, insomnia associated with unpleasant dreams and mental depression coloured by underlying fears and dreads. There are periods in which the patient is relatively free from anxiety, but these periods of intermission may be broken by apparently causeless waves of depression and often associated with the intrusion into consciousness of the memory of a particular and unpleasant incident.

Rivers[1] has pointed out that many of the most trying and distressing symptoms of the war neuroses are due to attempts to banish from the mind distressing memories or painful affective states; and Rows[2] has shown how the memories of painful experiences in earlier life, with which a strong emotional tone is connected, may be an important means of maintaining anxiety symptoms in soldiers after the more acute effects of the war shock have passed away.

For the treatment of these conditions a mental analysis should be made and therapeutic conversations carried on. Instead of encouraging repression, the unpleasant emotional experience should be talked over, ventilated and brought into line with the patient's personality. He should be made to understand the nature of his trouble; the manner of its origin should be explained to him, and he should be shown how best to readjust his outlook to his symptoms.

It was a common observation made quite early in the war that symptoms of anxiety were more usual in officers, and that the physical symptoms of hysteria—paralysis, contractures, mutism, astasia-abasia—were more frequent in the men. In a general way this was true, but a combination of anxiety-depression and gross hysterical symptoms was by no means uncommon. The most likely explanation of this difference is found in the position of greater responsibility in which the officer is placed, and to the mental conflict or struggle which he undergoes to banish from his mind doubtings as to his adequacy or competency for his work. In the case of the men, the attitude of antagonism to the fighting, accompanied by a wish for release from an unbearable situation (which it is alleged forms the basis of the mental conflict), may be converted readily into a sensori-motor symptom by such circumstances as a slight concussion, a physical shock, partial burial, or a minor and not necessarily incapacitating wound.

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[1] Rivers, *Proc. Roy. Soc. Med.*, 1918, xi (Sect. Psych.), pp. 2, 3.

[2] Rows, *Brit. Med. Journ.*, March 25, 1916.

Mental Reactions.

I do not propose to refer, other than quite shortly, to the *mental reactions* following upon failure of psychological adaptation. Many contributory influences, some of them of a deeply-rooted and complex character, are at work in the causation of the psychoses, but the trend of recent investigation and research into the origin of these disorders, emphasizes the importance of mental conflict, as the fundamental ætiological factor in their production.

The doctrine of the psychogenic origin of mental disease is replacing the old materialistic, physical or "toxic" view.

It is a point, moreover, whether some mental "symptom-complexes" may not have both an organic and functional origin, similar to the epileptic "symptom-complex," where we have long recognized, on the one hand, an organic or symptomatic epilepsy, and on the other hand, a functional or idiopathic disorder.

In the psychoses, as in the psychoneuroses, the experience of war has thrown much light upon the psychological mechanisms underlying their origin. We have seen in the observation wards and mental war hospitals, numbers of soldiers, especially when below the normal standard of mental development, suffering from a mental breakdown during the early months of military training due to an inability on the part of the soldier, with his individualistic up-bringing and education, to adapt himself to the novel and unusual circumstances of camp life with its many uncongenial associations. Although a large number of men with definitely neuropathic and psychopathic tendencies may pass through the period of training successfully, a further liability to break down occurs when the soldier finds himself under the unnatural and exacting conditions of active service.

There is another aspect of this subject which is apt to be overlooked by the medical mind, but which is of great importance from the military side. One of the most remarkable facts in this connexion was the very large number of men who were able within a short time to adapt themselves and maintain good nervous and mental health for long periods under the most stressful conditions of modern warfare. There is also trustworthy evidence to show that numbers of feeble-minded soldiers, who, it might be supposed, would be liable to adaptational failure, were trained by Army methods and discipline to successful readjustment towards those difficulties which service overseas demands.

The conditions of military service are such that symptoms of mental alienation may be detected in the earliest possible stage. Abnormal behaviour or disciplinary delinquences are observed and noted, and these are in many cases the first indication of a mental estrangement.

According to Stanford Read[1] the most prominent feature of the war mental reactions was the prevalence of a paranoid colouring of the underlying condition, whether this was the feeble-minded, the manic-depressive or the dementia præcox type of mental reaction. It may consist of any degree from a vague feeling of suspicion or an indefinite sense that everybody is against him up to a brief unsystematized persecutory psychosis.

Army life, especially on active service, favours the development of suspicious and persecutory ideas. The massing of large numbers of men with temperamental differences, uncongenial habits and incompatible tendencies, a harsh and unsympathetic management on the part of superior officers and a rigid discipline, tend to create in certain temperaments asocial habits and morbid introspection. Hence the soldier comes to believe that his comrades are making critical and unpleasant remarks about him, and eventually imagines that he is regarded as a spy and "voices" call him names and threaten him.

A confusional psychosis was probably the most common type of mental reaction observed in soldiers coming from overseas. In some cases it formed the basic reaction, and was attributable to a cerebral commotion or shell shock. In other cases it was the early phase or the onset of an underlying and more deeply-rooted disorder, such as dementia præcox or general paralysis of the insane. In some of these confusional cases, exhaustion and toxic causes (especially malaria and alcohol) may have been contributory factors, but there is reason to suppose that many were purely psychological in origin and were due primarily to a maladaptation or inability on the soldier's part to cope with a new and altogether intolerable situation (Stanford Read).

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[1] Stanford Read, *Proc. Roy. Soc. Med.*, 1919, xii (Sect. Psych.), p. 45.

EPILEPSY.

The psychological investigation, or mental analysis, of patients suffering from functional nervous disorders, which has proved so fruitful in the study of the psychoneuroses, anxiety states and allied conditions, has been applied recently to the study of idiopathic or essential epilepsy. As the observations on this disorder are little known in this country, I have thought it desirable to refer to them, so as to indicate the lines along which progress would appear to be made possible in this most ancient and intractable of nervous ailments.

The study of epilepsy heretofore has been conducted mainly from the physical, physiological and biochemical aspects, and what may be termed the life-history of the disease is well known in all its manifestations, phases and complications.

As a result of much investigation into convulsive disorders the frequency of idiopathic epilepsy has been reduced by the more general recognition of symptomatic epilepsy, as a common variety of the disease not only in children, but in adults also. Notwithstanding this, when all possible causes have been investigated and the symptomatic type eliminated, the number of cases of essential, genuine or idiopathic epilepsy remains large.

The psychological study of idiopathic epilepsy includes the recognition of the temperamental make-up of the epileptic and the analysis of the mental content accompanying the convulsive or other characteristic reaction.

Epileptic Temperament and Make-up.

The epileptic temperament has been described by many writers on this disease. It is rare indeed to find an epileptic who does not present some form of mental obliquity. Epileptics are usually self-opinionated, egotistical, and possess a conceit and assurance out of all proportion to their achievements. We find them moody, with periods of laziness and lethargy, alternating with outbursts of hastiness and pugnacity. They are difficult to live with and are often useless as workers. The majority are unable to adapt themselves to social conditions and as a result become self-centred, morbid and asocial.

Pierce Clark,[1] to whom we are indebted for the most complete and exhaustive clinical analysis of epilepsy from the psychological point of view, defines the epileptic constitution as characterized by a personality defect of egotism, morbid sensitiveness, hypochondriasis and poverty of ideas. In consequence the epileptic is incapable of social adaptation and is rendered inadequate to lead a normal adult life. This temperamental make-up is inherited. It exists from earliest childhood and may be ascertained by a careful and detailed study of the life-history antecedent to the onset of the fits. When present in an outstanding degree, its possessor may develop into an established epileptic, the fit reaction becoming a necessary corollary of the inheritance. It may be accompanied by evidence of maladaptation, which takes the form of day-dreams, outbursts of temper, irascibility or depressive phases for many years before the convulsive reactions occur.

The Onset of Epilepsy.

The epochs of new adaptations, which entail fresh and more elaborate forms of stress, are those periods of life in which epilepsy most usually begins. Such periods have long been recognized in puberty and adolescence. These epochs, which are by far the most common for the onset of the idiopathic disease, correspond to the development and maturation of the reproductive instincts with all the novel adjustments which these entail. The adaptations desirable and necessary in connexion with school, university or apprenticeship life, and later on with professional or business careers, may be accompanied by the onset of epilepsy in those who are potentially prone to it, as a result of their temperamental make-up or from a lack of proper educational training in adjustment to stress.

Epileptic Reactions.

In Clark's opinion, the convulsive reactions of epilepsy are the direct outcome of the inability of the epileptic to subordinate individualistic tendencies to social demands and the reaction is shown in a loss of consciousness. Put in another form "the seizure is a reaction away from stressful reality and may serve some purpose by enabling the patient to escape from responsibility or avoid some necessity for adaptation."

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[1] Pierce Clark, *Psychiatric Bulletin*, New York, January and April, 1916, and January and October, 1917.

The epileptic has certain inherent traits which hinder natural development and retard and weaken the normal capacity to adjust to adolescent life. The reaction may assume divers forms other than the convulsive seizure with which we are all familiar. These are the various forms of *petit mal*, probably the most frequent of all epileptic reactions, as well as day-dreams, abstractions, lethargies, dream episodes and other psychical equivalents. The *grand mal* seizure is naturally the most severe reaction but "the imaginings and day-dreams of epileptics may be so vivid and real, that reality is easily cast off in a lapse of consciousness" (Clark).

Detailed mental analysis of epileptics may bring to the surface all kinds of mental conflicts and repressions, which favour the onset of epileptic reactions in consequence of an inability on the part of the patient to adjust his outlook to particular circumstances, such as periods of stress or disappointment. I would recommend a study of Clark's work on this subject to those who are interested especially in the psychological investigation of epilepsy. One feature stands out with notable prominence—viz., the value of a meticulous examination of the psychological history and make-up of the epileptic, both actual and potential.

It is obviously impossible to conduct a psychological analysis during the period of unconsciousness, but an examination of this character may be made during the milder types of seizure, in the transitory confusion accompanying *petit mal* attacks, and in the post-convulsive dream states of the major attack. A study of the epileptic deliria and the spontaneous remarks which are made by patients in these conditions, if noted and analysed, may throw light upon the mental conflict underlying a particular epileptic reaction. Clark points out that the real motivation of the fit may be unconscious, while that which would appear to be sufficient may not be followed by an attack. It would appear also "that the ordinary immediate stimulus of the fit is not the irritation itself, but the effort of repression." "Repression may increase nervous tension to the point that a convulsive reaction may be the means of temporarily withdrawing the person from a too demanding environment."

The approach to a fuller understanding of the fit in individual cases would seem to be a thorough study of the aura, combined with an analysis of the elements of the seizure and the mental make-up in the interparoxysmal period. "It may be assumed that the disorder in genuine epilepsy has to do with adjustments at deep instinctive levels, that they are profoundly biological in character and that the fit as a reaction to failure may be so severe as to threaten even life itself. . . . Hence the epileptic type of reaction tends to cripple, and to terminate in a large number of cases in that permanent renunciation to efficient adjustments to reality—viz., epileptic dementia."[1]

Psychological Treatment of Epilepsy.

The bearing of what has been said upon the psychological foundation of epilepsy and the importance of the study and analysis of the mental conflict of particular seizures, raises the question of the treatment of epilepsy, as at present conducted. A reaction to the almost universal therapy by means of the bromides was bound to come, especially in view of the fact that large numbers of epileptics (rather more than 50 per cent.) derive no benefit from bromide medication.

In the hospital class of chronic epileptic, it is obviously impossible to examine or treat large numbers of these patients along individualistic lines. In badly adapted persons, as most chronic epileptics are, with well-marked temperamental defects, a steadily progressive epilepsy may develop with (and in some cases even without) a minimum of environmental stress. In these persons epilepsy attains its maximum frequency and severity. The social position of the epileptic outside an institution is unsatisfactory and unfavourable for adaptation. He becomes the victim of "social repression" which tends to aggravate his disability. He loses spontaneity and contact with the world, while interest in himself increases. He becomes apathetic, dull, anergic, and may lie in bed for long periods. More institutions for epileptics might be established with benefit to the community, and more might be effected in these institutions by individual attention and effort towards training the patient to accustom himself to types of stress, if he is to make a proper adaptation in life. Amongst those whose adaptational disability is less pronounced, or the high-grade type of epileptic, are found the cases of epilepsy which are cured, or in whom the fits are arrested for a sufficiently long time to encourage the hope of a cure. In these cases, the temperamental make-up is only slightly developed, and the disease usually does not appear until late in puberty or adolescence or even later in life, when under conditions of stress a full adaptive demand may be required, but cannot be adequately met.

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[1] White and Jelliffe, "Diseases of the Nervous System," 1917, Art., "Epilepsy."

In private practice much more can be done by training the young epileptic along suitable physical, educational and psychological lines to meet the fundamental demands of his life.

A rational therapy, therefore, should be based upon psychological lines and efforts should be made to educate the epileptic to adapt himself to the stresses and difficulties of life, whether these are met with in his home, at school, college, work or business; or whether they are social, ethical or moral.

I have sketched here in outline, mainly from the recent studies of Pierce Clark, the psychological attitude towards epilepsy, which has revived interest in this disease during recent years. There is much in it which may stimulate to further study of the malady. It brings epilepsy into line with other convulsive and allied disorders.

The experience of the war has thrown a somewhat remarkable light upon this subject. The psychoneuroses and psychoses have shown a largely increased frequency under conditions of mal-adjustment and mental conflict in war experiences. On the contrary, genuine epilepsy arising *de novo* under war conditions has been rare. The majority of epileptic soldiers were epileptic before joining the Army and many so-called epileptic fits in soldiers were in reality hysterical convulsive reactions. In order to establish the existence of epilepsy, we have to look beyond the seizure or convulsive reaction and to find it in the characteristic temperament or mental make-up.

I do not propose to offer, on this occasion, any criticism of the psychogenic origin of epilepsy, which has just been sketched. I believe that the experience of most of us is that genuine epilepsy differs very widely from the common psychoneuroses in that it is based upon deeply-rooted and complex foundations. The main point of difference in the fully developed and chronic disease is the tendency towards dementia, which invariably follows.

The value of psychotherapeutic measures in the treatment of epilepsy has yet to be proved. It is obvious that any method or remedy, which will strengthen the adaptive power of the epileptic to the strains and stresses of life is to be sought out and made available, but nothing which has been mentioned has shown the need for discarding the use of small and carefully regulated doses of the bromides in suitable cases.

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Section of Neurology.

President—Dr. W. ALDREN TURNER, C.B.

Some Lessons of the War applied to Spinal Surgery.[1]

By PERCY SARGENT, C.M.G., D.S.O., F.R.C.S.

SYLLABUS OF PAPER.

GUNSHOT Injuries compared with those of Civil Practice. Nature of the Anatomical Lesions (1) of the Cord, (2) of the Membranes and Bones. Comparison of the Conditions found at Early and Late Operations, in Fracture-dislocation and Gunshot Wounds respectively. Views as to Advisability of Laminectomy in Gunshot Injuries applied to Civilian Cases. Lessons bearing upon Treatment; the Automatic Bladder; the Significance of Bedsores; Treatment of Spasticity; Re-education of Function; the Swimming Bath.

FEW lessons of the war stand out more clearly than the elimination of the unhappy and wholly artificial division of diseases into medical and surgical. Almost every problem presented by the wounds of warfare has required for its elucidation the co-operation of a number of workers—pathologists, physiologists and chemists—as well as clinicians.

This is no less true of wounds involving the nervous system than of any other group, and if there is one general lesson concerning spinal injuries more important than another which ought to be taken to heart in our dealings with the accidents of civil life, it is that their treatment should not be solely in the hands of either a neurologist or a surgeon. Thus on the one hand we should no longer see cases of hysterical paraplegia provided with a spinal jacket, nor, on the other, patients abandoned to their fate who might be benefited by operation.

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[1] At a meeting of the Section, held November 13, 1919.

In some important respects gunshot injuries of the spine differ from those usually met with in civil practice. With the latter must be included the many examples of spinal injury which occurred during the war from accidents other than the impact of a missile, such as resulted from men being flung about violently by an explosion, or buried by the collapse of a trench.

The primary outstanding difference is that of *wound infection.* The gunshot wound is invariably compound, whilst the fracture dislocation is not. On this account the opportunities afforded for observing the state of the cord in gunshot injuries in the early stages were greatly curtailed, for the septic state of the damaged tissues prohibited any operations other than those directed to the treatment of the wound. The majority of wounds in which the cord was directly or indirectly implicated did not lend themselves to early radical excision. Whether or not a patient with a gunshot injury of the spine died of meningitis depended almost wholly upon whether or not the dura mater had been torn, and under no circumstances was it justifiable to open the intact theca, in order to ascertain the state of its contents, in the presence of a septic wound. It became evident in the earliest days that the escape of cerebro-spinal fluid almost certainly meant death from meningitis unless the leak could be stopped by operation, and this, in an otherwise favourable case, I always regarded as one of the few definite indications for early operation. In many cases the theca had been punctured by a fragment of bone which, remaining *in situ*, plugged the hole which it had made, and prevented both the escape of cerebro-spinal fluid and the ingress of micro-organisms. An operation which uncorked the theca in the presence of a septic wound enormously decreased the chances of recovery. I need not here enter further into this question, and I only mention it in order to emphasize the handicap imposed by the condition of the wounds, not only in preventing more frequent early explorations, but also because the results of infection rendered most of the cords obtained post mortem useless for neuro-histological purposes.

It is possible, and even probable, that a mild infection of the spinal membranes from which the patient recovered would produce intrathecal adhesions such as give rise later to that loculation of cerebro-spinal fluid which so closely resembles the meningitis circumscripta serosa described by Horsley.

The second outstanding difference concerns the manner in which the cord is damaged. Most of the injuries of civil life are produced either by the massive displacement of one part of the vertebral column upon another, or by a crushing fracture of the vertebral bodies whereby part of the comminuted bone is squeezed backwards so as to contuse and compress the cord. Momentary displacements of two vertebræ upon one another, with immediate rebound, may also injure the cord. In any case the damage is most commonly produced by direct violence, and the result may vary from a slight contusion up to a complete shearing across of the cord. The cases of gunshot injury comparable with these were those in which a spent bullet or shrapnel ball, impinging upon some part of the spine, entered the vertrebal canal driving fragments of bone before it. But when a missile of high velocity struck some part of a vertebra without entering, or directly violating the contents of, the vertebral canal, effects were commonly produced which are rarely seen in the spinal injuries of civil life. The resulting changes in the cord are usually described as concussion phenomena, and the lesions vary from transient œdema up to a total disintegration of the cord at the point of maximum intensity. These cases have helped to answer the much debated question as to the occurrence of temporary loss of function in the cord from concussion, comparable with that so commonly met with in the brain. Momentary or very short-lived functional loss has been observed many times, not only from direct hits, but also as the result of the bursting of a high explosive shell close to the patient.

Gunshot wounds provided many instances of the Brown-Séquard syndrome, and Roussy has drawn attention to a group of partial lesions in which the cord was hemisected, as it were, not in the lateral, but in the antero-posterior direction.

MORBID ANATOMY.

The excellent description of the morbid anatomy and histology of the damaged spinal cord given by Gordon Holmes in his Goulstonian Lectures (1915) is too well known to require more than a passing mention here. One point, however, requires special notice. In the surgical text-books the term "hæmatomyelia" finds a prominent place, but in the light of Holmes' observations it would appear that the pre-war conception of hæmatomyelia needs revision. In the sense that minute hæmorrhages are present in every cord which has been at all severely damaged, the term might be allowed to stand, were it not that it lays too great an emphasis upon this relatively unimportant aspect of the cord changes. In the sense of a hæmorrhage of any notable size spreading up and down in the cord comparable with the hæmorrhages of pathological origin, no instance has come under our observation, and it would seem likely that in fracture-dislocation it is an occurrence of great rarity. Most, if not all, of the cases of cervical hæmatomyelia, are probably instances of concussion.

Similarly, whilst a slight extramedullary and extrathecal hæmorrhage is commonly met with in recent examples of gunshot wound, a hæmorrhage of sufficient extent to cause symptoms of compression is rarely encountered. Holmes and I met with only one such case, and that in the post-mortem room. On the other hand a small amount of effused blood within the theca may perhaps give rise later to adhesions, and loculation of the cerebrospinal fluid.

Although the functions of the damaged cord can only be re-established to the extent to which the nervous elements have not been actually destroyed, yet the cord is, fortunately, to use Henry Head's picturesque expression, so "over-wired," that quite a gross degree of destruction is not incompatible with a useful amount of recovery. Surgery can only indirectly influence the return of function in the injured cord by placing it in the conditions most favourable for recovery by removing, as thoroughly as may be practicable, any actual or potential cause of compression.

I shall now very briefly compare the gross extramedullary lesions which are found at early and late operations in gunshot injuries and fracture-dislocations respectively.

(I) CONDITIONS FOUND AT AN EARLY DATE IN GUNSHOT INJURIES.

The cases selected by Holmes and myself for laminectomy within a few days after the wounding were those which we considered suitable, judged, broadly speaking, by the following criteria :—

(1) Absence of serious injury elsewhere.

(2) Leakage of cerebro-spinal fluid.

(3) Radiographic evidence of the presence of a missile or fragments of bone within the spinal canal.

(4) Some evidence, however slight, that the conductivity of the cord was not wholly abolished.

To this list might perhaps be added *pain*. It is true that in some cases the pain caused by damage to nerve roots is of an agonizing character, whether spontaneous, or elicited by touching hyperæsthetic root areas. A few such cases came under our observation in France, and some were operated upon. But on the whole the necessity for surgical intervention on the ground of pain alone is, in my experience, much rarer than the text-books would lead one to suppose. The first twenty-four cases which we subjected to early operation, two to ten days after being wounded, can be grouped, according to the gross anatomical features which they presented, into :—

(1) Nine cases. Those with a missile and bony fragments within and narrowing the spinal canal, but without laceration of the dura.

(2) Six cases. Those similar to the foregoing, but with leakage of cerebro-spinal fluid.

(3) Five cases. Those with a missile within the theca, mostly with gross destruction of cord, and all with leakage of fluid.

(4) Four cases. Those in which no evidence of injury could be seen within the spinal canal. In such cases the injury to the cord, whatever its degree, represented a pure concussion effect.

Of these twenty-four, sixteen were followed for periods of from twelve months to four years. Only eight show any considerable degree of recovery; two are *in statu quo* and six patients are dead. Of the eight definitely improved, neurologically speaking, only four can walk, but in all of them partial or complete voluntary vesical control has been regained. All but one had a piece of metal and bony fragments removed from the spinal canal, whilst in four cases cerebro-spinal fluid was leaking from the wound. It is difficult to believe but that the early operation had some favourable influence upon these recoveries.

(II) CONDITIONS FOUND AT A LATER DATE IN GUNSHOT INJURIES.

Superficially no two cases are exactly alike; fundamentally they have much in common as regards the immediate surroundings of the cord. There is dense scar tissue, often with bony fragments embedded in it, adherent to the cord or to the nerves of the cauda equina, and this scar may form a veritable annular stricture. There is also in some cases a definite cystic loculation of cerebro-spinal fluid. The cord itself may appear pale and shrunken, and in one case there was an intramedullary cyst; in the worst cases it can be seen to have been partly or wholly divided, whilst fragments of bone, and even of metal, may be found embedded in it.

(III) CONDITIONS FOUND AT EARLY OPERATION IN FRACTURE-DISLOCATION.

The few cases upon which I have operated within twenty-four hours of the accident have all shown the same features—namely, the unruptured theca tightly stretched over an anterior bony prominence, and devoid of pulsation below that point. In all of them the salience of the bony projection was reduced by traction upon the spine and manipulation whilst the theca and its contents were carefully protected from further injury. After a short time, probably only a few days, such reduction would no longer be possible, and then the salient ridge would have to be chiselled away. A similar decompressive result can be attained by the removal of two or three laminal arches both above and below the site of the dislocation, as this tends to straighten out the theca.

(IV) CONDITIONS FOUND AT LATE OPERATIONS IN FRACTURE-DISLOCATION.

In addition to the cord being angulated over a bony projection, as in the early cases, the lumen of the spinal canal may be still further encroached upon by callus. Scarring of the membrane may be found constricting the cord or cauda equina; meningitis circumscripta serosa is commonly present. In a recent case, operated upon four months after the accident, I found a pale, soft, swollen cord pressed against the dura mater by a definite cystic collection of cerebro-spinal fluid situated in front of it.

The conclusion to which one is drawn by a study of these four classes is that some of the extramedullary causes which must militate against recovery could be eliminated, or at least minimized, by early operation; and further, that, at a later date, there may exist conditions retarding recovery which are capable of being removed by operation.

Early exploratory operation should perhaps be more often undertaken in cases where there is no septic wound to contra-indicate it. If in any given case it were possible to ascertain by neurological examination that the cord had been completely trans-sected, or so seriously damaged as to preclude the possibility of any useful degree of recovery, then clearly it would be useless to operate. But the experiences of the war have shown that this is not possible. There is no sign or group of signs by which a complete lesion can be diagnosed with precision in the earliest stages. We can only say that the early appearance of a defence reflex from the soles, and the presence of an extensor response in the great toe, may probably be taken as favourable indications, though both have been observed in the presence of lesions subsequently found to be complete. The risks of an exploratory laminectomy are in reality very small, at least in the hands of those familiar with the operation, and it is my belief that its difficulties have been greatly exaggerated. The chief essential in any laminectomy is to secure a full and complete exposure of the spinal contents for some distance above and below, as well as actually at, the site of the injury. In this way alone can information be obtained and dangers avoided.

CYSTITIS.

Next in importance to the question of laminectomy comes the problem of preventing and treating cystitis and pyelonephritis. The work of Head and Riddoch has been of incalculable value in this respect. This is not the place to discuss the merits of vesical drainage, irrigation, and catheterization. These and other measures, carefully and thoroughly carried out, can do much to diminish infection of the bladder and to postpone the fatal issue; but no amount of care will either wholly prevent, or cure, cystitis in the absence of return of vesical function. If neither voluntary nor reflex function can be restored, the patient will sooner or later inevitably succumb to pyelonephritis.

During the interval which must elapse between the infliction of the spinal injury and the return of vesical function, whether voluntary or automatic, not only must every precaution be taken against infection, but over-distension of the bladder ought to be avoided. The exact path by which infection reaches the kidney from the bladder is uncertain, but there is some evidence that vesical distension at least plays a part. Further, a bladder which has habitually been subjected to over-distension becomes atonic, and this, apart from the condition of the spinal centres, delays that recovery of function upon which the cure of cystitis depends. Head and Riddoch emphasize the fact that toxæmia inhibits the reflex activity of the spinal cord. Absorption through the wall of any pus-containing cavity is accelerated when the contents are under pressure, so that in this manner also over-distension of the bladder will retard recovery. On these grounds any method of dealing with the temporarily paralysed bladder which encourages or permits its wall to be overstretched ought to be avoided. Vesical distension plays an important part in that common sequence of events which lead to pyelitis and death. Once the activity of the vesical reflex arc has begun—and this, according to Head and Riddoch, takes place on an average during the third week after the spinal injury—the automatic action of the bladder can be established by methods which these authors have described in detail. The amount of fluid constituting the adequate stimulus for detrusor action is determined in each individual case; twice a day this quantity is poured into the bladder until the reflex is evoked. The evacuation can be facilitated by various means such as deep breathing, hypogastric pressure, and by any cutaneous or other stimulus capable of evoking a flexor spasm of the legs.

The re-education of the bladder is one of the most important of all the lessons learnt from the study of gunshot wounds of the spine, and should prove of inestimable value in the treatment of those cases of spinal injury which are met with in civil practice, as well as in the management of many cases of paraplegia resulting from disease of the cord.

BEDSORES.

The work of Head and Riddoch has emphasized the importance of bedsores, and pointed out their real significance. They are no longer to be regarded merely as a reproach to nurses, nor do the general illnesses which may result from septic absorption constitute their chief danger. If the bedsore kills it does so most frequently by way of the kidneys, for it forms a link in a fatal sequence of events — namely, bedsore, toxæmia, depression of reflex spinal activity, cystitis, and pyelonephritis. It possesses also a little vicious circle of its own, for healing depends largely upon the recovery of the reflex functions of the cord which are inhibited by the action of circulating toxin absorbed from the bedsore. It is during the period of spinal shock that the skin is most vulnerable. Then it is that the traumatism produced by pressure, by heat and cold, and by rubbing, most easily starts inflammation, ulceration, and necrosis of the skin. Small wonder, then, that a paralysed man who, after lying out in the cold and wet, has been transported from hospital to hospital upon a rough stretcher, should arrive at the base with bedsores already well established. It is in the earliest stage that the greatest precautions should be taken, and in civil life the prevention of bedsores should be more easily ensured than in the often unavoidably adverse conditions of warfare.

THE PARALYSED LIMBS.

The spinal injuries of the war have provided ample material for determining the signs whereby complete and incomplete lesions of the cord may be distinguished from one another. Previous opinions upon this question have been most ably criticized, and present views very fully set forth by Riddoch, whose paper in vol. xl of *Brain* should be closely studied by all who have to deal with patients suffering from traumatic lesions of the spinal cord. From the purely practical point of view it is important to be able to ascertain not merely whether any minimal conduction persists through the damaged segments, but whether enough cord remains undestroyed to allow of a useful degree of recovery in the limbs. Many paretic patients who can produce quite good voluntary contractions of the muscles, are totally unable to put the legs to their primary and fundamental use as organs of locomotion. So much has been learnt in this regard from wounded soldiers, that we have even been able to review a number of old standing cases of spinal injury, and in some instances to benefit them. I have one patient, for example, who was previously bedridden and encumbered by a pair of useless, flexed, and spastic legs, but can now walk.

When any considerable degree of recovery has taken place in an injured cord, the usefulness of the legs may be impaired, not only to the extent to which sensory and motor paths have been permanently cut, but by other causes as well. A more general recognition of these causes of disability, and a wider knowledge of their treatment, may be reckoned amongst the most valuable of the practical lessons derived from the study of war injuries of the spine. These accessory causes, which in varying degree contribute to the disability, and which are largely remediable, are :—

(1) Inability to make proper and co-ordinated use of the recovered neuro-muscular mechanism, a power which can be restored by properly directed methods of re-education. Stated in the simple terms of common sense, the patient has forgotten how to use his legs, and must be taught afresh.

(2) *Hysteria.*—The existence of hysterical symptoms grafted upon organic lesions is now well recognized. Of the psychical treatment of such symptoms I am not competent to speak. But whilst recognizing its importance I should like to emphasize the value of what is in reality psychotherapy carried out by those who know nothing of psychology and have never even heard of Freud. It is no uncommon thing for a

paraplegic patient to lie in bed for months with no treatment beyond a little perfunctory massage. The mere fact of getting such a patient out of bed into a chair may have a strong psychical effect; the gradual systematic re-education of movements which have long been lost interests and encourages him; and every stage of recovery brought about by these and other means is a powerful stimulus to further effort. The swimming bath, the theraputic use of which we owe largely to Farquhar Buzzard, has been of incalculable value. Patients who can perhaps just stand with support, but who can make no movement of progression, are sometimes able to walk almost unaided in the bath, at their very first attempt. With his weak legs supported by the water, a patient very soon learns to perform the co-ordinated movements of swimming. At the same time his physical condition is improved by the exercise, whilst further effort is encouraged by the optimism which progress engenders.

(3) *Deformities.*—A third case of disablement in a recovering case is furnished by deformities due to neglect. I need only mention them here; they should never be allowed to develop, but when established, their correction by ordinary orthopædic measures constitutes an essential preliminary step in treatment.

(4) *Spasticity.*—Spasticity should be regarded from two aspects: first, as it affects those with total transverse lesions, or with lesions so severe as to preclude any possibility of recovery of function in the lower limbs; and secondly, in those who possess, or are on the way to regain, what would otherwise be a useful degree of voluntary power.

Spasticity is harmful in more ways than one. It not only conceals and renders useless any voluntary power that may exist, but the spasms are often so disturbing to the patient as to tell seriously upon his health. Further the constant friction against the bedclothes damages the skin and causes bedsores, particularly in those cases in which the cord lesion is so situated that outbursts of sweating accompany the flexor spasms; whilst splinting for the prevention of deformities is rendered extremely difficult. I can only mention very briefly some of the ways in which the difficult problem of spasticity may be dealt with. If we regard it quite simply as the manifestation of excessive and uncontrolled reflex action, we may attack either the afferent or the efferent side of the reflex arc; or else we may deal with the mechanical rather than the neural aspect. In other words we may take measures to restrict the output of energy, or else we may allow the power to run to waste, or we may divert it to a useful purpose. To take the mechanical aspect first, by dividing the attachments of muscles their effective action may be, temporarily at least, abolished or modified ; by transplanting the spastic hamstrings into the quadriceps tendon their energy can be utilized for correcting the very deformity which they would otherwise produce. Restriction of output of energy may be brought about by diminishing the afferent impulses which evoke reflex action. The avoidance of rough handling and of every other sudden and strong stimulus is important, and it is hardly necessary to point out the harmfulness of irritating spastic muscles by massage and electricity.

The operation suggested by Professor Foerster for the spastic diplegia of cerebral origin, namely, division of some of the posterior nerve-roots passing to the affected segments of the cord, known to those who like labels by the cacophonic term "rhizotomy," has proved of considerable value. The method which I adopt is to expose the conus medullaris and upper part of the cauda equina by removing the neural arches of the last two thoracic and the first two lumbar vertebræ, and then to divide alternate bundles of the lumbar and sacral posterior roots to the extent of about two-thirds of their total bulk. The actual number divided depends upon the degree of spasticity which it is desired to overcome. In the cases with complete division of the cord, or with such a severe lesion as to render any useful degree of recovery impossible, I have no hesitation in dividing anterior roots as well. Possibly in such cases, in order to preserve the power of facilitating automatic action of the bladder, it would be better to divide anterior roots alone. A useful minor operation for the temporary relief of spasticity, when it chiefly affects the adductor muscles, is to expose and crush the obturator nerves. In the hopeless cases these nerves should be divided so as to produce permanent paralysis.

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Section of Neurology.

President—Dr. W. Aldren Turner, C.B.

DEMONSTRATION OF CASES ILLUSTRATING THE LATE EFFECTS OF INJURIES OF THE NERVOUS SYSTEM.

Shell Wound of Head, Right Temporal Region, Sensory Paresis of Left Hand and Foot; Mental and Physical Symptoms due to Hole in Skull; Effect of Closure with Osteoplastic Graft.[1]

By Henry Head, M.D., F.R.S.

(1) Captain E. C. T. E., R.A.M.C., aged 29; wounded March 22, 1918, with the fragment of an aëroplane bomb. He did not realize he was hit, but continued to dress the wounded. Was wearing nothing on his head at the time. Then remembers nothing until he found himself in Rouen. He was admitted to the Casualty Clearing Station with a "small penetrating wound through outer and inner tables of the skull and a small piece of the inner table had been driven into the brain. There was a large extra- and intra-dural hæmorrhage." He arrived at Rouen on March 26, and the wound was explored two days later. A subdural hæmorrhage was found and the wound was drained. He made an uninterrupted recovery.

Admitted to the London Hospital on April 13, 1918, with paresis of left arm, leg and face. The wound had healed except for some granulating tissue over the centre of the pulsating portion. It was situated in the right temporal region 15 cm. to 19 cm. behind the nasion and 7 cm. to 10 cm. to the right of the middle line; total nasion-inion line, 35 cm. At this time he suffered greatly from headache. The lower portion of the left half of the face was weak and the tongue was protruded somewhat to the left. All the deep reflexes on the left side were brisker than those on the right, but there was no ankle clonus and the plantar gave a downward response; the abdominal reflex was absent on the left side. Grasp of the left hand was weak and isolated movements of the fingers were poorly performed. The lower extremity was not affected. The greatest change was in the sensations from the left hand and foot. Recognition of passive movement was diminished in the whole of the left hand and great toe, but not in elbow, shoulder or ankle. The compasses were badly discriminated over index, middle, ring and little fingers and over the left sole. Localization was poor over the four fingers but good over the sole. The tactile hairs showed loss of sensibility of these fingers and the sole, but there was no definite loss to temperature, and prick was unaffected. There was gross loss of recognition of shape and texture in the left hand.

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[1] At a meeting of the Section, held December 11, 1919.

In May, 1919, he was admitted under my care, at the Empire Hospital, for headache. The wound was deeply depressed, pulsating, and he was perpetually conscious of the opening. Stooping upset him; he could not travel in the train or play golf. Concentration and intellectual effort at once brought on a headache. The physical condition was almost exactly as before.

On May 28, 1919, Mr. Wilfred Trotter filled in the hole in the skull with a piece of bone taken from the right shin. The wound healed by first intention.

The skull now felt completely normal on palpation, and it would have been difficult to say where it had been repaired. All signs of pulsation had disappeared, and the scar was absolutely painless. He was no longer troubled with the feeling of an opening in the skull, but could stoop and make unexpected movements without any abnormal sensation. His power of intellectual effort increased, and he was again able to play golf.

[This patient was unavoidably prevented from attending the meeting, and was replaced by the following officer.]

(2) Captain E. N. C., Indian Army, on February 19, 1918, was serving in Mesopotamia; his saddle slipped, and he fell under his horse. He was kicked in the left parieto-occipital region, and sustained a compound fracture of the skull. He was operated upon within a few hours. "Bone fragments were removed; the brain below the injury

was pulped to a depth of about 3 in., and hernia cerebri developed, which subsided after three lumbar punctures." He was admitted to the Empire Hospital for Officers on September 6, 1918. The wound had healed completely and was covered with a deeply depressed pulsating scar. He showed definite aphasia and right hemianopia, with loss of central vision on the blind side. There were no other abnormal physical signs. He suffered from no headache, but was much troubled with consciousness of the opening in his skull. He could not stoop or make any sudden movement without the pulsation increasing considerably. He was easily tired, both by travelling in the train and intellectual effort. Physical exertion, such as walking, did not produce abnormal fatigue.

On October 17, 1919, Mr. Wilfred Trotter filled in the hole in the skull with three grafts from the tibia. The opening measured 11 cm. in length, and 5½ cm. vertically. The wound healed by first intention, and when the patient was shown at the meeting, it was impossible to make out with certainty where the opening had been.

The most striking change was the patient's recovery of confidence in himself; he no longer feared to stoop, or to make sudden movements, and lost the habit he had acquired of placing his hand over the opening when he sneezed or coughed. Associated with this recovery of self confidence, his power of speech distinctly improved.

Closure of Openings in the Skull by Bone Grafts.

By Wilfred Trotter, M.S.

Operation: The opening is exposed by the reflection of a large flap, not including the pericranium. If the scar of the original injury in the scalp is of doubtful vitality it should be excised and the opening in the flap stitched up. The gap in the skull is outlined by an incision down to the bone at its edge. The pericranium is reflected away from the opening. The dura and scar are freed from attachment to the bone, and the scar is pared down as much as is possible without opening the subdural space. No harm will result if the subdural space is accidentally opened. The rounded edge of the bone is cut away with a chisel so as to give a flat surface on which a graft can lie. Thin slices of adequate size to overlap the opening are then cut with a frame saw from the inner surface of the tibia, the periosteum being divided and reflected in such a way as to leave a fringe projecting all round the graft, but especially at its ends. The graft is laid on the opening and secured firmly in close apposition to the skull by numerous stitches between the fringe of periosteum surrounding the graft and the fringe of pericranium surrounding the hole. In this way absolute fixation can always be secured. Most openings left by war wounds can easily be closed by two grafts, but sometimes more are necessary. It is not difficult to reconstitute the skull—even though flat grafts alone are used—in such a way that after a lapse of some months it is scarcely possible to detect any abnormality in shape.

Indications for operation: It is rare for any patient who has an opening in the skull to be quite free from symptoms. The smaller the opening and the more rigid the scar by which it is occupied the less likely are the symptoms to be pronounced. The commonest symptom is tenderness of the scalp around the gap. When the latter is in a situation to be pressed on by a hat the inconvenience is serious. Consciousness, more or less unpleasant, of movement at the opening is also common. This of course varies with the area and the thinness of the scar. Every movement that causes even a slight variation in the venous pressure is felt at the opening. Thus when the patient stoops, coughs, sneezes, laughs, or makes any effort, he feels the brain bulge and the scar stretch. One patient, who had had an inguinal hernia, compared the feeling in the head when he coughed or laughed with that which the rupture had given him. Sometimes the arterial pulsations of the brain are a source of distress, especially in the recumbent posture. Actual headache comes next in the order of frequency. To be characteristic it should show evidence of a mechanical origin, being brought on either by the depressed or the expanded position of the brain. In the former case, the patient will say that he wakes in the morning without headache, but that this comes on after he has been on his legs some time, and is at its worst at bedtime. In the other case the patient will wake with headache, which improves after he has got up.

In certain cases of traumatic epilepsy there is evidence that the opening aids in maintaining the abnormal excitability of the brain. When the onset of the fits has a definite relation to posture, this possibility should especially be considered. Frequently it is obvious that the opening in the skull is a source of uneasiness and distress that the patient cannot put into definite words. He may complain of being worried by the unnaturalness of his condition, by a supposed danger of injury, or he may be able to do little more than convey a sense of anxiety, not the less distressing for being indefinable. Whether these are to be regarded as evidences of disturbed function of the brain or of the mind, they are invariably relieved by closure of the opening. Whenever headache is a pronounced symptom and the opening is small and the scar rigid, the possibility that there is an element of unresolved contusion in the case should always be considered. When there is little movement of the scar with changes in posture, the mere opening is unlikely to be a source of the symptoms. When there is any doubt as to the diagnosis, an exploratory operation to determine the condition of the brain should be done.

Shell Wound of Head, 1915; Persistent Headache Four Years; Operation; Free Opening of Skull and Dura in Region of Injury; Contusion of Brain Found; Relief of Headache.

By Wilfred Trotter, M.S.

J. W., aged 31. Shell wound of skull, 1915. Patient does not remember anything for several days after. He had two operations for removal of fragments of bone and metal. The wound healed in about ten weeks. As soon as he was allowed out of bed headache began and persisted up to the recent operation four years later.

Character of headache: A dull aching pain was continually present and was liable to severe exacerbations. These were brought on by exertion, fatigue, excitement, or worry. The headache when severe was accompanied by giddiness. On three occasions he "fainted" at his work. He was unable to continue his work as a gardener, as stooping was particularly apt to bring on the pain. The headache was felt over the vertex of the skull round about the scar.

Operation, November 11, 1919: Bone removed in region of the scar and dura incised freely. There was slight bulging of the brain. The arachnoid was opaque and slightly distended by fluid collected under it. The brain was stained an orange colour as the result of extravasation of blood. The conditions were typical of an old unresolved contusion. There has been a great improvement in the headaches since the operation.

This case is shown as characteristic of a common sequel of head injury due to war wounds. The pathological basis of the condition is an unresolved contusion of the brain substance; of its clinical manifestations, headache is by far the most conspicuous.

In a typical case the history will be that the patient has suffered a local injury to the vault of the head from a bullet or shell fragment. The injury may appear to have been a mere graze or to have involved the bone. It will be characteristic that there has been no great loss of bone substance. If there has been a perforation of the skull the opening will be quite small and usually closed by a rigid scar. The headache generally comes on, or is first seriously complained of, during convalescence, when the patient is beginning to get about. There may be more or less continuous moderate headache, but the chief feature is the liability to attacks of very great severity. Such attacks may last from a few hours to several days, and are apt to be brought on by physical exertion, by exposure to bright lights or loud noises and by mental excitement. In some cases stooping and even a brief physical effort may cause a throb of intense pain and, if often repeated, lead to a definite attack. During the attack the headache is of an intense throbbing, bursting character and often of unbearable severity. In many cases the patients declare that between attacks they feel perfectly well. Giddiness is often complained of and is brought on by the same causes as produce pain. If the injury is appropriately situated there may be signs of slight functional defect of the cortex, but commonly headache is the dominating symptom. When the brain in the region of the scar on the scalp is explored evidences of an old contusion are found. There may be more or less increase of tension, but this is so slight as often scarcely to be distinguishable from the effect of venous engorgement due to the anæsthetic; the arachnoid is opaque and its trabeculæ unduly visible, and it is distended by the local accumulation of fluid beneath it; the brain shows discoloration from old extravasation of blood. In the patient shown, the brain in the contused region was of a deep orange tint though four years had elapsed since the injury. The evidences of contusion vary in intensity, and in well marked cases amount to a definite meningitis serosa circumscripta. Occasionally a small amount of actual blood clot is found on the surface of the brain.

After free removal of bone and incision of the dura the headache is greatly relieved or completely cured. Some months later the opening in the skull should be closed.

On the theoretical aspects of the condition it may be desirable to call attention to two well established matters of principle. First, the resilience of the skull is such that it may be accepted as an invariable rule that whenever a missile travelling at high velocity touches the bone, the latter is driven in upon the brain with force enough to inflict a considerable bruise. It is quite unimportant whether the bone shows evidence of injury to ordinary clinical or X-ray examination. The bone may have recovered its shape completely and have sustained no fracture and yet have caused a distinct local lesion of the brain. If it can be shown, then, that such a projectile has touched the skull the existence of a local cerebral contusion at the point of contact may be assumed.

Secondly, the brain, being the only organ in the body with a capsule (the skull) completely rigid to all physiological forces, is unable to undergo the compensatory general swelling by which the circulation is maintained through any other bruised organ. Thus it is that a contusion of the brain is apt to remain unresolved much longer than a similar condition anywhere else. In the case shown, there were visible evidences of contusion no less than four years after the injury. This tendency of lesions that elsewhere would be trivial and passing to persist and cause grave symptoms is altogether peculiar to the brain. Like very many other phenomena special to cerebral pathology, it is a direct consequence of encapsulation by the skull.

Gunshot Wound of Head; Fracture of Left Frontal and Parietal Bones.

By OSWALD SHIELDS, Major R.A.M.C.

PENSIONER A. J. G., aged 21. Wounded on September 1, 1918 (bullet); bullet entered left parietal and passed out through left frontal region.

Operation in France on September 2, 1918: Entrance and exit wounds excised, and fragments of bone removed; extensive laceration of brain found.

Symptoms: Very severe frontal headaches.

Operation on November 22, 1918, by Major Shields: Plate of perforated celluloid placed over opening in skull in order to close gap (at this time the smooth celluloid plate was not being inserted between skull and dura). Headaches were much relieved for eight months, then again became severe.

Operation on October 28, 1919, by Colonel Sargent: Hole in skull enlarged and celluloid plate removed; dura freed from skull and thin sheet of smooth celluloid placed between skull and dura; perforated celluloid placed over opening in skull, and scalp sutured in place.

Gunshot Wound of Head; Fracture of Left Frontal and Parietal Bones.

By OSWALD SHIELDS, Major R.A.M.C.

PENSIONER S. H., aged 25. Wounded on April 27, 1915 (shell).

Operation (in France): Decompression; date uncertain.

Symptoms: Severe frontal headache, vertigo and fits, becoming more severe.

Operation on February 28, 1919, by Colonel Sargent: Dura found adherent to brain; fat graft inserted between dura and brain after freeing; double celluloid perforated plate placed over opening in skull; scalp sutured in place.

Operation on March 14, 1919, by Colonel Sargent: Celluloid plate removed and replaced by perforated zinc plate. Suppuration followed.

Operation on March 24, 1919, by Colonel Sargent: Zinc plate removed; fat graft seen to be necrosing in places; August 18, 1919: three fits; another on September 27, 1919.

Operation on November 18, 1919, by Colonel Sargent: Old fat graft found to be completely fibrous and adherent to pia, so that it could not be removed; it was freed from skull around opening; a thin smooth celluloid plate was inserted between skull and fat graft, and a large thick perforated celluloid plate made to the shape of the skull was placed outside skull to cover the opening; scalp sutured in place.

Facial Paralysis.

By E. FARQUHAR BUZZARD, M.D.

PENSIONER B., aged 23. Blown up by shell on April 1, 1918; face swelled up and paralysis was noticed two days later; weakness in left arm and leg was present at first, has since improved, but still shows on much exertion; there is now complete facial paralysis and loss to faradism, except for orbicularis oris. Probably there is a pontine lesion in region of right facial nucleus, orbicularis oris fibres escaping.

Case of Spastic Paraplegia illustrating certain Reflex Phenomena and the Impeding influence of Muscular Hypertonicity and Reflex Spasms on Voluntary Movements of the Lower Limbs.

By GEORGE RIDDOCH, M.D.

SECOND-LIEUTENANT F. C. J., late L.N. Lancs, was shot by a sniper in the back on July 31, 1917. He sank to the ground at once and was unable to move his legs. Free from pain at first, he suffered later from intense soreness of the inner aspects of the arms from the wrists to the axillæ and of the upper part of the back and chest while being carried to the aid-post on an improvised stretcher.

On admission to the Empire Hospital on August 3, 1917, his condition was found to be as follows: The bullet had entered in the left supraclavicular fossa, crossed the back and made its exit 10 cm. to the right of the second thoracic spine. The pupils were equal and reacted well to light and shade. Reflexes: Arm-jerks were present and equal on the two sides; abdominals absent in all segments; knee- and ankle-jerks not obtained. On scratching either sole with a pin a feeble flexion reflex with up-going toes was evoked. Motion: Complete flaccid paralysis of the lower limbs and trunk below the fourth rib. No involuntary movements of the lower limbs were present. Sensation: On the chest, back and inner aspects of arms and hands in the distribution of the eighth cervical, and first, second and third thoracic posterior roots, a dragged pin gave rise to a disagreeable feeling of soreness. Below the fourth rib, though there was complete loss of sensibility to prick, light touches were appreciated, and better on the right than on the left half of the body. Bladder and rectum: Retention of urine and fæces.

X-ray examination: No displacement nor fracture of bone could be seen.

Operation: Forty-one days after the injury the spines and laminæ of the seventh cervical, and first, second, and third thoracic vertebræ were removed. Unfortunately, a record of the condition of the membranes and cord found at operation does not seem to have been made.

A fortnight after the operation the patient could move the toes of his right foot and slightly bend the right knee. Some weeks later similar movements were possible in the left lower limb. At this time all involuntary movements were flexor in type, and it was not till March, 1918 (eight months after the injury), that strong extensor spasms appeared. About a month later he was able to initiate extensor movements of the legs by voluntary effort. It was now possible to begin stepping exercises. Though voluntary power was slight it was sufficient to change the direction of the reflex movement from extension to flexion or flexion to extension. So the sequence of events was as follows: When placed on his feet, the tension on the calf-muscles excited strong extension of both legs and he was able to stand upright, balancing himself with the help of the self-propelling chair. Then concentrating his attention on one leg he could inhibit the tonic limb extension and evoke a flexor spasm, this in turn being replaced by extension. The process was next repeated in the opposite limb. Because of the spasticity the effort expended was very great and the exercise could be gone on with for a short time only. Gradually, however, he regained more control over the limbs.

His present state, two years and four months after the injury, can be summarized as follows:—

Reflexes: Abdominals.—When the stimulus is applied in the epigastric region a local reflex response is obtained, but when the site of stimulation is near the groin a general contraction of the muscles of one half or of the whole abdominal wall usually results. Cremasteric and bulbo-cavernosus reflexes are present and erection of the penis is of frequent occurrence. Knee- and ankle-jerks are very brisk on both sides, and foot and patellar clonus is well sustained. The twitch of the knee-jerk is often followed by rhythmic extension of the limb at hip and knee due to contractions of the glutei and quadriceps extensor cruris muscles occurring at the rate of about four in five seconds. These movements, which are most often bilateral, move the pelvis forwards and backwards. The hamstring muscles take part in the backward movement. This reaction often follows a bilateral extensor spasm and is evoked by handling the penis or by rubbing the lower part of the abdomen or the inner surface of the thighs. It is inhibited by nocuous stimulation of the sole of the foot or by dorsiflexing the foot suddenly; the former mode of stimulation exciting a flexion reflex and the latter an extensor thrust.

Flexion reflex: Flexion of the lower limb with up-going toes is obtained by pinching, pricking or scratching the foot or the leg. Evoked by squeezing the upper part of the thigh it appears sometimes to be a primary reaction, but most often it is secondary to a brief downward movement of the toes. The lower limbs are so sensitive to stimulation and the reflex movements evoked are so vigorous that it is difficult to determine the exact nature of the crossed response with the flexion reflex. The obvious movement is flexion, but sometimes initial—very brief—extension is apparent. After a flexor spasm the lower limbs usually extend vigorously at hip, knee, and ankle, all the muscles of the thigh and leg being strongly contracted. The toes, however, are dorsiflexed. An extensor thrust of the limb, in which the toes move down, is obtained by pressing on the sole of the foot so that the tension on the calf muscles is increased. Abduction and eversion of the foot tend to be associated with extension of the limb and adduction and inversion with flexion.

Motion: He can perform all movements of the trunk and lower limbs, but in attempting to flex or extend the legs he has still great difficulty in controlling spasms. He can determine the direction of the movement, voluntary impulses exciting the appropriate motor paths. But once started the movement is carried on largely by reflex action outside his control, and he has great difficulty in regulating its amplitude and vigour. In addition the hypertonic condition of the muscles offers great resistance to voluntary effort. He moves his limbs more freely and in a more controlled fashion after they have been immersed for a time in a hot bath. Independent movements of the foot and toes are possible, but when asked to flex or extend the knee or hip all the flexors or extensors of the limb are brought into action. Involuntary flexor and extensor spasms of violent character are still frequent Both flexor and extensor muscle-groups are hypertonic, but when the limbs are moved passively the resistance encountered in the extensors is different from that in the flexors. The resistance of the extensors is maximal at the beginning and then suddenly gives way, while that of the flexors is more or less constant throughout, the muscles gradually lengthening. There is very little muscular wasting.

Sensation: The inner aspects of the arms and the upper part of the chest and back are much less tender to touch and pressure. Below the level of the fourth rib light touch is everywhere appreciated, though less clearly than in normal parts. The defect increases towards the feet. Pin-pricks are said to be "sharper" and "more disagreeable" than normal, and cold objects give rise to a sensation with a distinctly painful quality. The vibrations of a large tuning fork (C_{128}) are not appreciated in the lower limbs and trunk below the sixth rib, and the recognition of changes in posture and of passive movement is gravely disturbed.

Vaso-motor, secretory and trophic changes: Dependence of the limbs does not cause œdema, and the patient never sweats from the trunk and legs. The skin is dry and scaly. There are no bed-sores.

Bladder and rectum: He is able to control to some extent the automatic activities of the bladder and rectum. Micturition can be started off before the bladder is full, and provided the contents of the rectum are soft he can expel them by voluntary effort; he can prevent reflex micturition but for a short time only. He always knows when the bladder is full and when empty.

Case of Meningitis Circumscripta Serosa following a Bullet Wound of the Neck, with Injury of the Posterior Columns of the Spinal Cord.

By George Riddoch, M.D.

The clinical features of this case due to the medullary injury were described in a paper read before the Section on January 24, 1918. It will be sufficient here to summarize them briefly.

Lieutenant B., R.A.F., was struck by a bullet in the back of the neck on August 12, 1917, while flying. Both arms were immediately paralysed, but he could move his trunk and legs fairly well. He suffered from intense pain across the shoulders, which were very tender to touch. Injury to the third cervical laminal arch was noted at the casualty clearing station, and an unsuccessful attempt was made to remove pieces of metal which were lying in the soft tissues of the back of the neck.

On admission to the Empire Hospital on October 20, 1917, the wound had healed, but in the track of the scar there was an oval depression, 6 cm. long and 3 cm. broad, the floor of which appeared to rest on the laminal arch. X-ray examination showed that damage had been done to the spines and transverse processes of the third, fourth, fifth, and sixth cervical vertebræ. The left pupil was larger than the right and there was slight ptosis on the left side. Poorly sustained ankle clonus was obtained and on scratching the sole of either foot hamstring contraction accompanied downward movement of the hallux. Other reflexes were normal. Except for weakness of both arms, especially of the hands and fingers, and on the right side more than on the left, motor power was very little affected. There was slight wasting of the arms but more of the fingers, and the tone of the muscles was greatly diminished. The movements of the arms, especially of the hands and fingers, were grossly inco-ordinate. His gait was ataxic, and when he stood upright with his eyes closed he swayed from side to side. Tactile sensibility was gravely disturbed in the pre-axial side of the forearm and in the thumb and first three fingers. Over the same area sensibility to prick, heat and cold was defective. Tactile localization and the appreciation of deep pressure and pressure pain were everywhere unaffected except in the hands and forearms. The spacial elements were disturbed in the hands and arms out of all proportion to the other aspects of sensation. He was quite unable to find objects with his hands in the dark or when his eyes were shut, though he knew their position in space quite well. To turn on the bed-lamp in the night he had to grope for the switch with his upper arm and then press it down with his teeth. He was quite unable to dress himself and had to be fed.

Though the sensory condition of the hands has not altered much in the last two years, and the movements of his hands are clumsy, he can now do everything for himself, and drives a motor car with safety even in the dark. He rode on a bicycle for a time but had to give it up as accidents were too frequent. So long as he kept a watch on his hands everything went well, but as soon as his attention was diverted by obstacles on the road his hands left the handle-bars and he found himself in the ditch.

In March, 1919, he was readmitted to hospital and Mr. Trotter obliterated the hole in his neck by a plastic operation. His general health at this time was excellent, but for the last three months he has been gradually becoming less fit. His gait is again unsteady at times and he has greater difficulty in controlling the movements of his arms and hands. The physiological deterioration dates from a bathing accident in August. While swimming under water on his back his head struck a ridge of sand with such force that he turned a somersault: he felt something click in his neck, was giddy and felt nauseated for a few minutes, but was able to get out of the water without help. A few days after the accident he noticed that the depression in the neck had reappeared.

In September he went to bed one night feeling out of sorts, and about four o'clock in the morning woke up sick and giddy. He vomited and felt very faint and dazed. Severe pain radiated from the region of the depression in the neck, over the back of the head, and spasmodically down the spine. He was compelled by the pain to keep his head, neck and shoulders rigid. His mother, who was with him, noticed that the hole had disappeared. The attack, after lasting for about six hours, stopped suddenly and he felt quite well again. Since then he has had five similar attacks, four in bed in the early morning and one while he was lying in his bath.

On examination it was found that the skin corresponding to the distribution of all the cervical and the upper four or five dorsal nerves is very tender to pressure and to a dragged pin. The increased sensitiveness is most pronounced on the back of the head, the posterior part of the vertex, the sides of the head including the ears, the neck and over the shoulders and upper arms and the upper part of the chest and back.

In the attacks, which are precipitated by local circulatory changes connected with the supine posture, not only the cervical cord but also the medulla is implicated. Consequently decompression is a matter of urgency.

Subsequent report: On December 20 Mr. Trotter removed the laminal arches of the second, third, fourth and fifth cervical vertebræ. The exposed dura bulged through the opening and when pricked a stream of clear cerebro-spinal fluid spouted up to a height of 5 or 6 in. On slitting open the dura the arachnoid over the whole uncovered area was seen to be opaque and its strands thickened. The spinal cord, which pulsated fully, appeared to be normal. Having approximated the edges of the dura by two or three widely separated interrupted sutures, the muscles were firmly stitched together in layers and the wound closed. The patient stood the operation well and at the time of writing, seven days later, he is very fit, complains of no discomfort, and the tenderness in the cervical root distribution has gone.

Case of Lesion of the Suprascapular Nerve and First Dorsal Root, with Hemisection of the Cord produced by a Single Missile.

By C. P. SYMONDS, M.D.

E. R., AGED 28, while himself in the act of firing his rifle, was sniped at a distance of thirty yards: he fell back into the trench and at once noticed that his left arm was useless; he then found he could not sit up nor use his legs. On his way down to the clearing station he lost the use of his right arm and at the same time his power of speech, hearing and vision. He had retention of urine for a week, having to be catheterized. At the end of this time he recovered speech, sight and hearing, but remained quadriplegic. At the end of three months he recovered the use of his right arm somewhat suddenly. Power in the right leg returned slowly and it was eight months before he could walk. He continued to improve for another six months, and since then his condition has remained stationary. He now complains of weakness of the left arm and leg, and has noticed in his bath that he is unable to appreciate differences of temperature with the right leg.

On examination, there is the scar of a bullet wound on the outer aspect of the left arm 3 in. above the elbow: no exit wound. He shows slight narrowing of the left palpebral fissure, and the left pupil is constantly smaller than the right. There is complete atrophic palsy of supra- and infra-spinati on the left side; the intrinsic muscles of the left hand are completely wasted, resulting in *main en griffe*, and there is a band of cutaneous anæsthesia corresponding to the segmental distribution of the first dorsal root. Below this level, over the right half of the body and right lower limb there is impaired sensation to pin-prick and heat and cold, and some light touches are missed. The power and reflexes of the right lower limb are normal. The left lower limb shows spasticity in extension with exaggerated tendon reflexes, ankle clonus and extensor plantar response: the abdominal and cremasteric reflexes on this side are absent. Vibration sense is definitely impaired over the left tibia, and there is some loss of sense of position in the left foot.

Stereoscopic X-ray plates show the casing of a rifle bullet lying superficially, to the left of the fifth cervical spine: in addition very numerous small metallic fragments representing the burst core of the bullet; these fragments are all superficial except a tiny one which is apparently within the vertebral canal at the level of the sixth cervical vertebra, the arch of which is fractured. The upper border of the scapula in the neighbourhood of the supra-scapular notch shows evidence of injury. Thus a single missile has divided the supra-scapular nerve, cut the first dorsal root close enough to its origin from the cord to implicate the cervical sympathetic fibres, and finally produced a hemisection of the cord itself.

Case of Spastic Quadriplegia following Injury of the Spinal Cord in the Upper Cervical Region, showing Certain Unusual Reflex Phenomena.

By E. Farquhar Buzzard, M.D., and George Riddoch, M.D.

Sergeant V., aged 35, wounded by a spent bullet in the upper part of the neck on February 7, 1915. Entry wound: Small round scar 1½ in. above and 1 in. behind tip of left mastoid. No exit wound. Patient says the bullet was found in his coat collar. Immediate paralysis and loss of sensation in trunk and all four limbs. For some time after being hit he was unable to speak and his breathing was much embarrassed. He was compelled to take quick short breaths. There was retention of urine at first, but in a few hours the bladder began to empty its contents automatically.

In a little over a week he was able to move his right great toe voluntarily, and three or four weeks later his left great toe. Voluntary power in his lower limbs improved steadily. About two months after he was wounded he could flex and extend the right thumb at the metacarpo-phalangeal joint. Sensibility in the trunk and limbs also began to recover and he regained some control over the bladder and rectum.

Summary of Present State.

He has become, perhaps, too resigned to his disability.

Pupils: Right larger than left. Both are regular, central, and react well to light and on accommodation. Both dilate on shading, but the left sluggishly and with small excursion. Left palpebral fissure is narrower than right. Otherwise cranial nerves are normal.

Motion: He can perform all movements voluntarily. The legs are better than the arms and the right arm and leg better than the left arm and leg. Voluntary effort is handicapped by the muscular hypertonus. Of arm movements, adduction is performed better than abduction at shoulder and he finds it easier to move the arm backwards than to bring it forwards; at the elbow, extension is better than flexion, and pronation of the forearm than supination; at the wrist, extension is a little stronger than flexion. The fingers are used clumsily. On attempting to extend the fingers the hand remains somewhat flexed at the wrist, though less so than when the arm is lying at rest. When he makes a fist the hand is strongly extended at the wrist. He can oppose the thumb to the little finger but, like all other movements, opposition is performed slowly and deliberately. Muscular relaxation, on command, is delayed. On asking him to clench the hand and then to open it quickly, a quite appreciable interval elapses before the command is begun to be carried out and the movements are performed slowly and in a jerky fashion. When the patient flexes or extends either leg against resistance, the arms flex involuntarily at the elbow, extend at the wrist, and the fingers and thumb fan out; being extended at the metacarpo-phalangeal joints and slightly flexed at the interphalangeal joints (claw-hand). He can now walk for a short distance without support, though he has considerable difficulty in keeping his balance on account of the general extensor rigidity. He has to watch his feet carefully.

Posture: As he lies in bed the arms are adducted at the shoulders, almost completely extended at the elbows, slightly flexed at the wrists, the hands not being quite in line with the forearm, which is fully pronated; the fingers are extended, the thumb adducted and opposed to the palmar surface of the index finger, and the fifth finger a little abducted from the fourth. The lower limbs lie on the bed extended at hips and knees and rotated out; the feet point and the toes are somewhat adducted and in line with the feet. The right arm and leg are less rigid in their posture than are the left arm and leg. The patient, as a rule, sleeps lying almost supine, his shoulders flat on the bed, with his head rotated to one side, the arms extended along the trunk, the right hip slightly elevated, and the legs flexed at hip and knee, the left leg lying under and just behind the right. When he stands up, with his body bent forwards, his arms hanging straight and stiff and a little in front of his trunk, his neck flexed and rigid, he is the picture of helplessness and immobility.

Tone: All the muscles of the trunk and four limbs are hypertonic except those of the ulnar fingers. Tonus preponderates in the extensor muscle groups, hence the prevailing posture of the trunk and limbs. Resistance to passive movement is greatest when the arms are being abducted or rotated at the shoulder, flexed at the elbow, the forearms supinated, the wrists extended, and the thumb abducted. The ulnar fingers are comparatively mobile. In the lower limbs the adductors of the thighs are more tonic than the abductors, and the extensors of the thighs, legs and feet than the flexors. Hypertonicity is greatest in the muscles controlling the large joints, and is least in the small muscles of the fingers. If the arm is passively flexed at the elbow with quick jerks the resistance encountered is quick and sudden, as if the triceps had taken up a new posture for each varying length. This "lengthening reaction" can be evoked over a wide range of different lengths of the muscle. It is obtained with all the muscles of the arm and thumb, but is most evident in the adductors of the shoulder, the extensors (triceps) of the elbow, the pronators of the forearm, the flexors of the wrist, and the adductors of the thumb. The extensors of the trunk and lower limbs show the reaction less clearly.

Involuntary movements: An emotional disturbance such as a fright may cause the arms to jerk forwards, but the most interesting involuntary movement in the arms is the following: It is a sudden and violent spasm in which there is adduction and internal rotation at the shoulder, extension at the elbow, extreme pronation of the forearm, slight flexion at the wrist, and hyperextension and adduction of the fingers, the index passing below the middle finger and the little finger below the ring finger. The thumb is adducted and opposed, the terminal phalanx remaining hyperextended. The hand and fingers form a cone and the palm looks backwards and outwards. It may be brought on by a fright or by a tickling feeling in the nose. Flexor and extensor spasms of the lower limbs still occur, but they are less frequent than formerly.

Reflexes: All the tendon reflexes are much increased. Flexion of the lower limb with upgoing toes is evoked by scratching the sole of the foot. With it the opposite lower limb extends. The extension-rotation reaction in the arm can be easily excited by scratching the palm of the hand, the inner aspect of the arm or the front of the chest, between the second and sixth ribs. The threshold of stimulus is lowest in the palm and on the chest between the third and fourth ribs. The complete response is as follows: The stimulated arm is suddenly and vigorously thrust backwards. It is extended, adducted and rotated inwards at the shoulder, extended at the elbow, completely pronated, flexed at the wrist, the fingers being extended and in such a position that they form a cone with the middle finger in front and the thumb in the palm. The palm of the hand looks backwards and outwards. At the same time the trunk is rotated towards the stimulated arm, the opposite arm is adducted and rotated *outwards* at the shoulder, and slightly flexed at the elbow, while both lower limbs are extended, the ipsilateral more than the contralateral limb. The new posture is maintained for some seconds, though the exciting stimulus is brief, and then the body and limbs slowly assume their position of rest.

Sensation: All aspects of sensation are affected in the arms, trunk and lower limbs. Recognition of changes in posture, and of size, shape and form are grossly defective, especially in the hands and feet.

Bladder and rectum: He has almost complete control over the functions of the bladder. He still requires to take laxatives every day.

An Unusual Case of Complete Bilateral Spastic Paralysis of Face, Jaw, Tongue and Larynx, following an Acute Illness.

By JAMES COLLIER, M.D.

W. B., A GIRL, aged 10, was in every way healthy and normal until 6 years of age, when she is said to have contracted scarlet fever. Three weeks later, when convalescent from the scarlet fever, she is said to have acquired meningitis, the symptoms of which consisted in her developing a squint and becoming unconscious, and lying semiconscious for three weeks. Her limbs were not paralysed during this time. She has never been able to speak nor to move her face voluntarily, nor to eat nor swallow naturally since regaining consciousness. She is a very intelligent child and she writes well. With the exception that there is slight perversity of movement in the use of the fingers (she constantly uses the middle finger of both hands instead of the index finger for acts involving approximation with the thumb), the condition of the limbs and trunk are in every way normal. The ocular movements are normal, and are performed at command. With the exception of retraction of the angles of the mouth, there is no volitional movement of the face, jaw, tongue or larynx. The face is in spasm, as shown by the frowning expression and the retraction of the angles of the mouth.

The teeth are tightly clenched, with occasionally a little audible grinding, and the masseters are in spasm. There is much dribbling of saliva, which she circumvents with her handkerchief, so as to keep herself quite clean. She feeds by pressing soft food into her cheek with her finger, closing the oral aperture with the hand and squeezing it through the teeth by the pressure of her fingers upon the cheek. The reflex movement of the eyelids is normal. Reflex swallowing is normal. The emotional movements of the face in smiling and crying are normal, and during these movements only is the mouth opened, and it is then opened widely. The associated movements of the face on effort are normal. She makes no attempt at articulation. She has recently begun to use a slight laryngeal grunt as a query and as an affirmative.

Section of Neurology.

President—Dr. W. ALDREN TURNER, C.B.

Forms of Peripheral Neuritis among Troops serving with the Egyptian Expeditionary Force, 1915-19.[1]

By F. M. R. WALSHE, M.D.

DURING the recent campaign in Egypt and Palestine undoubtedly the commonest organic nervous disease among the troops was multiple neuritis. It was also the only one that could definitely be said to be associated with the prevailing conditions of active service. Post-diphtheritic paralysis formed the bulk of the cases, and it is to these that my last remark applies.

During the whole period of my service from 1915 to 1919, I saw no case of multiple neuritis unquestionably due either to dysentery or to malaria, nor did any instance of acute febrile polyneuritis as it has been described by Dr. Gordon Holmes or Sir John Rose Bradford come under my observation. Finally, there was no beri-beri in Egypt or Palestine, apart from imported cases from Mesopotamia and a few in 1915 from Gallipoli.

I propose to confine my remarks this evening to a certain group of cases of multiple neuritis, and, since many of the symptoms of this malady are common to all its ætiological varieties, it will save you much wearisome and familiar detail if I single out for remark such noteworthy features of my cases as may be more or less new to you, and that may throw some light upon the origin of post-diphtheritic paralysis.

Of this, I saw over 160 cases. They fell into two groups: the first consisting of the ordinary type following faucial diphtheria; the second, and in some ways the more interesting group, associated with extra-faucial infections of superficial skin lesions and of wounds by the diphtheria bacillus. The opportunity of seeing so large a series was unique, for over sixty came under observation.

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[1] At a meeting of the Section, held January 8, 1920.

However, before referring to the characteristic clinical picture presented by these I should like to make some general observations on the order of appearance of the signs and symptoms of multiple neuritis. Having access to a large Isolation Hospital, I was able to observe continuously a number of cases of diphtheria until their discharge to Convalescent and Command Depots. Many were examined almost daily from the date of admission, and others from the appearance of palatal paralysis in the second and third weeks of their illness. Multiple neuritis developed in several of these while under observation, and contrary to expectation, I found that diminution and abolition of the knee- and ankle-jerks were by no means the initial signs of the malady. Subjectively, aching pains in the legs on exertion, painful cramps in the calves during the night, numbness of the feet and, objectively, tenderness of the calf and plantar muscles on pressure, with a remarkable facility of the tendon-jerks were the earliest phenomena. In six cases I was satisfied that the knee- and ankle-jerks underwent a definite preliminary exaltation before they began to grow sluggish. In many cases the ankle-jerks became feeble while the knee-jerks were still brisk, and they always disappeared earlier than the knee-jerk.

The point is, perhaps, of minor clinical interest. Nevertheless, in the circumstances in which the observations were made there was a definite value attaching to them. For in dealing with convalescents from diphtheria, the eliciting of the knee-jerks was commonly regarded as giving a clean bill of health to the patient in this respect, and as being a sufficient reason for disregarding any subjective symptoms of which he might complain. But in my experience this test was by no means adequate to exclude an early polyneuritis, and thus many men were sent prematurely to duty only to be readmitted to hospital within a week or two with a fully developed polyneuritis.

To return to the question of post-diphtheritic paralysis: during the winter 1916-17 and onwards there was an epidemic of diphtheria in the field and throughout the lines of communication, which extended from Palestine to the Delta. Although of mild character, on the whole it was fairly heavy in incidence, and, associated with it, I saw numerous cases of the ordinary type of post-diphtheritic paralysis. I am not able to give even an approximate estimate of the total case incidence in this outbreak, nor of the proportion of cases of diphtheria in which nervous complications ensued, but I think that the latter was high, and I ascribe this to two causes: first, the small dosage of antitoxin, a single dose of from 4,000 to 6,000 units being the rule; and secondly, the premature return of the patients to conditions of physical activity. Often within a month of an apparently mild attack of diphtheria, a man resumed route marching at a Command Depot, only to fall out with pains and weakness of the legs on his first march. In such a patient, one could watch the development of a full-blown polyneuritis, which disabled him for at least six months. In many of these men the knee-jerks were still obtainable on readmission for a short period. Further, many mild cases were, I am sure, never recognized as such but passed under various diagnoses—for example, tabes dorsalis, neurasthenia, and "D.A.H.," and thus any estimate of the total number was impossible.

Of the cases of post-diphtheritic paralysis following the ordinary faucial infections, there is little of particular interest to record, except possibly the large proportion in which the usual palatal and accommodation pareses were followed by a fully developed polyneuritis, probably for the reasons I have already given.

Early in 1917 cases of a mild type of multiple neuritis began to appear. They were not associated with any history of sore throat, nor with any other evidence of faucial diphtheria. The common ætiological factors seemed absent also, and the only feature common to the cases was the recent occurrence of a crop of "septic sores," except in one case, in which there had been a chronic suppuration in a flesh wound of the arm of some months' duration. The significance of this history was not recognized at once. And although the possibility of a secondary diphtheritic infection of the skin lesions was thought of, it was dismissed because the multiple neuritis was neither preceded nor accompanied by palatal or accommodation paralysis. In fact, the first ten or twelve cases that I saw had no cranial nerve symptoms whatever. In every instance their general condition was good, though there was in all of them a mild degree of tachycardia ranging from 80 to 100 when at rest.

However, some light was thrown on the question in the April of that year by a case which I have already recorded elsewhere, which made it appear highly probable that diphtheria lay at the bottom of all these obscure cases of multiple neuritis. The case was this: a medical officer attached to the same unit as myself performed a tracheotomy upon a fatal case of laryngeal diphtheria. He was not aware of having scratched himself at the time, but next morning he noticed a painful bleb upon the dorsum of the first phalanx of the right thumb. This broke down leaving a sore raw surface. The ulcer rapidly spread over the surface of the thumb and ultimately reached the size of 1 in. by ½ in. It was covered by a greyish slough and exuded a scanty watery secretion. On the fourth day he began to feel ill and to shiver. There was a lymphangitis up the arm and the axillary glands were swollen and tender. On the sixth day the diphtheria bacillus was isolated in pure culture from the wound and he was admitted to hospital. During the second week of the illness, severe symptoms of toxæmia appeared: vomiting, cardiac irregularity and albuminuria, and these persisted for about ten days. Improvement then set in, the wound began to heal, and in the seventh week he was discharged to convalescence. The knee-jerks were still brisk at this time. During the following two weeks he improved considerably, was able to walk up hill and to swim several hundred yards in the sea without undue fatigue, but from the seventh week onwards he had begun to notice that the thumb over and around the healed scar was very numb to touch and even to pressure. This impairment of sensation spread over the thumb and radial half of the hand, then invaded the rest of the hand and the forearm. Simultaneously his hand became very clumsy in fine movements, there was a constant feeling of pins and needles in the fingers and his handwriting became very untidy and difficult to read. His grasp was weak. About a week after his return to duty, that is, in the tenth week, be began to find that walking upstairs was very exhausting and required effort; his knees felt as though they might give way under him. Writing became quite impossible not only on account of the ataxy but because of painful cramps in the small hand and forearm muscles. There was no cranial nerve involvement. Twelve weeks after the original infection he was readmitted to hospital. On examination I found a well marked polyneuritis, with absent tendon-jerks, moderate weakness, tenderness of muscles and the characteristic sensory loss. But in addition to this, the condition of the right arm at once attracted attention. It was considerably weaker than its fellow. Its muscles were more flabby and the distal half of the limb was distinctly thinner. All fine movements of the hand and the movements of the proximal segments of the limb were ataxic. There was complete loss to touch over the hand and the distal third of the forearm and gross loss of other forms of cutaneous sensibility.

Here, then, was a case undoubtedly diphtheritic in origin, in which with the absence of palatal and accommodation paralysis, there were an initial local onset of nervous symptoms related to the site of the infective focus, and a multiple neuritis.

A short time after, a second and similar case was observed, and this is the last case I shall quote: The patient had received a flesh wound in the left lumbar region at the battle before Gaza in April, 1917. Although freely laid open early in the course of treatment and subsequently treated with every new and old-fashioned antiseptic, it had resisted healing. During August he was allowed up more freely, but as soon as he began to walk about, he complained of an increasing weakness, of "pins and needles," and of cramps in his legs. A provisional diagnosis of syphilitic ulceration and early tabes dorsalis was made and the man was transferred to my care. From a small unhealed patch at the centre of the keloid scar, the diphtheria bacillus was isolated in pure culture, while clinical examination revealed a fully developed multiple neuritis. In this case there was neither local nor cranial nerve affection. I say there was no local palsy, but there was a large area of sensory loss surrounding the wound, which could not be accounted for by the local surgical condition.

During the summer of 1917 several pathologists were reporting the presence of "diphtheroid rods" in septic sores occurring in areas where faucial diphtheria was present, and at the same time numerous cases of polyneuritis following crops of septic sores came under my observation. I may say that by septic sore I refer to a condition in which small areas of superficial ulceration appeared upon the dorsum of the hand, the extensor surface of the forearm and on exposed parts of the leg round the knee. This condition was extremely prevalent throughout the campaign, especially in mounted troops when on trek in the desert. They began to concern me because from the time that diphtheria became prevalent, these skin lesions were undoubtedly associated with a considerable amount of multiple neuritis among the troops. Unfortunately, in the majority of cases the primary lesions were all healed by the time the cases came to me with nervous symptoms, but in addition to the two cases already described I ultimately saw nine cases in which the cautious pathologist was able to isolate "a diphtheroid organism morphologically indistinguishable from the Klebs-Loeffler bacillus."

In all, I examined over sixty such cases of polyneuritis, and in addition to their close association as to time and place with an outbreak of diphtheria and of diphtheritic paralysis, and with the finding of the organism in several of them, certain clinical signs that they showed convinced me that they were true post-diphtheritic paralysis following cutaneous infections. Some new features were seen later in the series that the first cases had not shown, and an analysis of the whole number, made when I had observed thirty and confirmed by a second thirty seen later revealed that the complete symptom complex consisted of three elements:—

(*a*) An initial local paresis related anatomically to the site of the infective focus.

(*b*) A paralysis of accommodation, and

(*c*) Polyneuritis.

The *local symptoms* usually preceded by two or three weeks the visual and general symptoms. They occurred in about a third only of the total number. The *accommodation paralysis* was also present in but a third of the cases, while the *polyneuritis* was present in all. In the majority of instances multiple septic sores on the limbs preceded the onset of nervous symptoms, but in several the infective focus was single—a gunshot wound or a boil—and it was in these that the local paralysis was most commonly seen. In the first thirty cases, eight had a single primary focus, and in six of these there was an initial local onset. In none of the number was palatal paralysis observed, and in none were diphtheria bacilli found in the throat.

A brief consideration will show that these three elements are also found in post-diphtheritic paralysis as it follows the ordinary faucial infections, for the palatal paralysis is a true local palsy; the musculature of the soft palate is clearly closely related both in the matter of proximity and in the more important respect of innervation to the local lesion on the tonsils and fauces. This formula of three elements in the clinical picture of a fully developed diphtheritic paralysis may, therefore, be applied to any case, no matter where the infective focus may be. The question at once arises as to the origin of the nervous complications of diphtheria, and the path by which the toxin reaches the nervous system. The view commonly held is that diphtheritic paralysis is the result of a blood-borne toxæmia of the nervous system in which certain centres in the brain-stem are picked out by the selective action of the toxin. And, since experimentally inoculated animals do not show cranial nerve involvement as a part of their nervous symptom-complex, the further opinion has been expressed that in animals, on the other hand, the spinal nerve centres are specially attacked.

It seems, then, reasonable to say that the cases I have briefly described do not accord with this view, and, indeed, cannot be explained by it. For if the nervous lesion in diphtheria be the result of a selective blood-borne toxæmia, its clinical manifestations should not vary according to the site of the infective focus, but should be constant and unvarying, except in intensity. It appears to me that the following view best accounts for the presence of the three clinical elements I have described; it is of course an hypothesis that as yet lacks both experimental and pathological confirmation; it is as follows: That the initial local palsy is the result of the passage of the toxin from the infective focus to the central nervous system, brain-stem or spinal cord, along the perineural lymph channels of the peripheral nerves innervating the site of the local lesion. This is the process so extensively investigated by Orr and Rows, and named by them "lymphogenous toxi-infection of the nervous system." The suggestion is therefore merely an extended application of a known pathological process. This is not to say that the symptoms are caused by an ascending neuritis, for we know from Orr and Rows that the nerve, as far as its nervous elements are concerned, is commonly intact and that the lesion is central and is situated in that segment of the central nervous system from which the nerve arises. We may thus explain equally well the initial palatal paralysis of a case of faucial diphtheria, or the initial sphincter disturbances of a case of diphtheritic infection of an ulcer in the perineum.

Accommodation paralysis and polyneuritis are common to all types. For the former, therefore, we may still invoke the hypothesis of selective action, while the latter may be regarded as the expression of the general toxæmia as it affects the nervous system, while in both instances the toxin probably reaches the nervous system by the blood-stream.

It is of interest to determine whether the initial local symptoms indicate a peripheral or a central lesion of the nervous system, and it does not appear to me that the palatal symptoms of the ordinary type throw much light on this point. In the extra-faucial cases it is equally difficult to decide because the presence of a multiple neuritis masks any distinctive characters the initial palsy may possess. However, I was so fortunate as to see one case in which this was in a region usually unaffected in multiple neuritis. The case was one of ulceration in the perineum, arising as a septic sore (and later, presumably, secondarily infected by the diphtheria bacillus), which was followed by a local, an accommodation and a generalized nervous symptomatology. The local symptoms were sensory loss over the territory of the lower four sacral posterior roots on both sides and definite loss of control over the bladder, which, I think, can only be explained by supposing that the lesion was central and not merely an ascending neuritic process in the afferent nerves of the region.

I also saw a case—and one has been recorded by Oppenheim—of a streptococcal infection of the tonsils and fauces in which palatal paralysis and a multiple neuritis supervened. In both repeated search failed to demonstrate the presence of the diphtheria bacillus. Oppenheim remarks of his case that this clinical picture cannot be regarded as absolute proof of a diphtheritic infection, and I would suggest that these cases tend to confirm the view of the origin of the initial local phenomena that I have put forward. Several recent case reports in German periodicals of extrafaucial diphtheria and a resulting nervous involvement also bear me out in this respect.

In considering post-diphtheritic paralysis from the point of view I have put forward this evening, one cannot escape the analogy it presents to tetanus. Meyer and Ransom have divided the symptoms of this into three groups:—

(*a*) *Local tetanus*, in which, after prophylactic inoculations of tetanus antitoxin, the first symptoms appear in the immediate vicinity of the infective focus.

(*b*) *Specific tetanus*, in which, irrespective of the site of the infective focus, there is spasm of certain muscles—namely, trismus; and

(*c*) *Generalized tetanus*.—If the amount of toxin be considerable a generalized tetanus is superadded to the two preceding forms.

After intravenous injections of tetanus toxin into animals, local tetanus is not seen; similarly, after intravenous injection of diphtheria toxin into experimental animals, the paralysis is generalized and presents no local phenomena. We may, therefore, compare the local, specific and generalized forms of tetanus with the palatal, ocular and generalized nervous symptoms of faucial diphtheria—the palatal palsy being the local element, the accommodation affection the specific element, while the polyneuritis corresponds to generalized tetanus.

In conclusion, I must ask your indulgence for the somewhat broad manner in which I have dealt with my subject. There are many gaps, I know, on the theoretical side of my paper. I have merely tried to describe a somewhat unusual type of peripheral neuritis, and to indicate briefly the theoretical considerations to which its study gave rise, and its bearing on the pathogenesis of post-diphtheritic paralysis.

I know also that this is by no means a comprehensive review of the various forms of peripheral nerve disease that one saw in Egypt. However, the rest of these were merely fortuitous in their occurrence and were in no way peculiar to that field of operations. I have thought it best, therefore, not to attempt completeness, but only to deal with what seemed characteristic of the campaign in which I served.

DISCUSSION.

Dr. PHILIP MANSON-BAHR: I feel I am here under false pretences; what I am going to say as a sort of confirmation of Dr. Walshe's paper is the result, not of my own work, but that of my friend and late colleague, Dr. Craig, of Kendal. It was during the advance through the Sinai desert, and especially on the line of the Wady Guzze before Gaza, that "desert sores," or "septic sores," as they were called, first began to claim serious attention. Now from what we know this desert sore is probably synonymous with the "veldt sore" of South Africa and the "Barcoo rot" of Australia; at any rate it has something to do with desert conditions, and is quite distinct from the Oriental sore due to *Leishmania tropica*. Now it was noted by Craig that in the Egyptian Expeditionary Force the majority of units afflicted with these sores were mounted troops, or those associated with camels, and he concluded that as the clinical appearances of the lesions were so characteristic they must have a *peculiar* and *specific* cause. The sores affected those parts of the body exposed to injury—for instance, the hands, knees, and face. In mounted troops who rode in "shorts," as they did at that period, the sores were apt to form over the points of friction at the knee. However, in many cases they supervened *de novo* after no obvious sign of any injury. The onset of the sore is sudden and very painful, quite out of proportion to the size of the lesion; within a few hours a vesicle forms filled with serous, or it may be hæmorrhagic, fluid, collecting generally round a hair follicle. The vesicle soon bursts and leaves behind a raw area generally covered with a pearly-grey membrane and moist with exuding serum. The edges of the skin soon become undermined and the ulcer spreads in a peripheral direction and soon becomes punched out, chronic, and most intractable. The surrounding skin is livid and blue. Apart from the pain and disability they caused, the amount of debility, cardiac irregularity, and often anæmia, which existed in association with the sores, were sufficient to attract general attention and speculation as to the specific cause. Early in 1917 Dr. Charles Martin had reported the isolation of a "diphtheroid" bacillus from one of these sores, a fact which he did not then regard as significant, but rather he looked upon the lesions as being either streptococcal or staphylococcal in origin. This line Craig followed up, and in a short time was able to isolate diphtheroid bacilli, indistinguishable morphologically from the Klebs-Löffler bacillus, from 129 out of 197 of the sores he examined, that is, in 67·5 per cent. Making due allowance for the fact that many of his cases had been extensively treated with all manner of antiseptics, he reckoned that the percentage of successful isolations should have been much higher. Almost pure cultures of Klebs-Löffler bacilli were obtained by Craig and myself from the serum which exuded from the vesicles on Löffler's glucose serum, but the longer the ulcer had persisted, the more difficult it was to isolate the organism and the more numerous were staphylococci and streptococci. There was no doubt that they were typical Klebs-Löffler bacilli; they conformed to all the well-known tests, produced death in thirty-six hours when injected into guinea-pigs or quails, with hæmorrhagic peritonitis and lesions of the suprarenals; on the other hand, these animals could be efficiently protected by a simultaneous injection of antidiphtheritic serum. At the same time as these sores were so prevalent a large number of cases of faucial diphtheria were occurring, and most of our energies, as pathologists, were directed to cultivating the Klebs-Löffler bacillus in throat swabs by the thousand. But I must confess that we were more fortunate in obtaining a higher percentage of positive isolations from desert sores than from throat cultures. When General Allenby came out, as one would suppose from such a great commander, he took, amongst other things, more than an active interest in these desert sores, as they were causing a great amount of disability; in some units as many as 40 per cent. of the men were unfit for hard labour and had their hands swathed in bandages. There was at this time, for all-sufficient reasons, a considerable amount of official reticence in acknowledging the importance of Craig's discovery. It was thought that the absence of fresh elements, and especially of beer, from the dietary had a mysterious influence upon these sores, so Allenby caused all the beer in Egypt to be sent up the line. This had the effect of raising the spirits of the men, but it did not heal their sores. At this time we began to collect together a considerable amount of evidence of the association of these sores with paresis or neuritis of various parts of the body, as Dr. Walshe has just described. For instance, I remember the case of a corporal of the H.A.C. who was covered with these sores, who had neuritis of both legs, with lack of knee-jerks, hyperæsthesia of his calves, and great ataxia. He came down the line with a provisional diagnosis of tabes. Another I remember with paralysis of the palate following sores on the eyebrows and cheeks, and paresis and numbness of the arms, with multiple sores on the fingers and hands. Probably paralysis was much more common than was supposed, and in many cases it had been overlooked or attributed to presumably untreated faucial diphtheria. The effect of injection of 4,000 units of antidiphtheritic serum in the neighbourhood of these sores was almost magical; raw patches and ulcers which had persisted for months, nay, even years, after treatment with every form of antiseptic lotion and paste, healed up perfectly with sound firm skin within a week or ten days. I myself am absolutely convinced that this was so, though no success was obtained in getting this mode of treatment recognized officially or universally adopted. The scars which remained after healing are sufficiently characteristic, and the stigmata of "desert sores" will be evident for years; there is probably not a single man who served in the Australian Light Horse or Imperial Camel

Corps who does not bear one or more. The hypothesis that these sores were a primary skin infection with the diphtheria bacillus led Craig to consider that they were in some way connected with desert transport, for they ceased to be a scourge directly we had moved forward into the more fertile and cultivated portions of Palestine. Suspicion fell on horse manure as a source of infection. At that time the desert roads were laid upon a foundation of horse dung, which in this manner afforded a harder surface than sand for travel. Finally Craig succeeded in isolating a diphtheroid bacillus from this substance, but lack of opportunity prevented him from carrying his researches any further. From the experience I have had of this subject I should not be at all surprised to learn that a considerable number of cases of tropical polyneuritis, formerly diagnosed as beri-beri, are explicable on a similar basis. Finally, I would add that the inability of some pathologists to isolate the diphtheria bacillus from these ulcers in base hospitals in no way militates against Craig's work. By the time they reached the base these sores had been contaminated and invaded by all sorts of organisms, such as streptococci, which therefore cannot be considered the actual ætiological agent or the cause of the debility and neuritis associated with them. As an illustration I show a photograph of a Mesopotamian case recently under my care in which a sore suspected of being cutaneous leishmaniasis had persisted for two and a half years, and which instantly healed up after injection of antidiphtheritic serum.

Dr. FEILING: During my three years' experience in Mesopotamia I saw many cases of polyneuritis of various kinds in both British and Indian troops. On the whole it was not a very common disease there, at least amongst the British. I wish first to say a few words on the subject of beri-beri. While having the greatest admiration for the work done by many observers in attacking the problem of its ætiology from the view point of a dietetic deficiency, I feel obliged to demur to this being the only cause or in all cases any essential factor at all. Beri-beri after all is at present only a clinical term and different infections or toxins can produce in the field of polyneuritis clinical pictures which are very difficult, if not impossible, to differentiate on clinical grounds alone. I would like to illustrate this by brief reference to two epidemics of polyneuritis with which I became acquainted in Mesopotamia. One affected a number of Chinese labourers. These men were all of a fairly superior class, skilled carpenters mostly, and were housed in two large huts. The cases were confined to men in *one* of these huts only. Their rations had been expressly designed to contain as many so-called "anti beri-beri vitamines" as possible. They received good pay and were able to, and did, supplement their rations by extensive purchases in the local bazaars of such fresh food as fish, eggs, chickens and vegetables: I myself often saw them returning from market loaded with such purchases. Clinically the cases were typical beri-beri; many of them of the so-called "wet" or œdematous type. Another example of an epidemic of polyneuritis, which illustrated the difficulties of diagnosis and its consequent evils, was that in which a large number of cases of a severe form of polyneuritis occurred in a detachment of British troops occupying an isolated post in the desert. These cases were diagnosed as beri-beri, and many of them ultimately found their way to Bombay. There fuller investigations were undertaken with the result that *arsenic* was found in large quantities in the hairs of many of these cases. Inquiries were instituted and it was eventually discovered that a barrel of white arsenic had by some means been included amongst the barrels of bleaching powder used for water disinfection, and that these unfortunate men had been steadily disinfecting their water supply for some time with white arsenic. With regard to *dysenteric* neuritis, I have seen a number of such cases in which a dysenteric infection, generally of the bacillary type, was followed by polyneuritis. Less frequently it occurred amongst amœbic cases. It has been suggested, though on no very clear grounds, that the emetine given therapeutically was the cause of the neuritis in the amœbic cases. I saw one rather remarkable example of this condition. The patient was a young officer, who was admitted to hospital for fever and shortly afterwards operated on and a large liver abscess evacuated; the *Entamœba histolytica* was found in the pus from the abscess. During convalescence he developed a very severe polyneuritis which in its clinical features was quite indistinguishable from beri-beri. *Typhus* fever has been the cause of a certain small number of cases. In one of such the signs suggested that the infection had fallen on the ganglion cells of the cervical enlargement of the cord rather than on the more distal part of the motor neurone, for the principal feature of the case was extreme atrophy of the muscles of the shoulder girdle, the whole picture closely resembling that produced by an acute anterior poliomyelitis of the upper extremities. The *influenza* epidemic of 1918 was responsible for many cases of polyneuritis, many of them of a very severe type. Some of these again bore a very close resemblance to the classical picture of beri-beri. I saw one case which showed the somewhat remarkable combination of extreme atrophy of the leg, with great œdema affecting the subcutaneous tissues of the trunk only; this case also showed diaphragmatic paralysis and a severe cardiac affection; in spite of the fact that malaria became a still further complication the patient eventually recovered completely. In conclusion I would point out that in only too many of the cases the ætiology has been a matter of grave doubt or even pure speculation.

Section of Neurology.

President—Dr. W. ALDREN TURNER, C.B.

Anxiety States.[1]

By R. G. ROWS, O.B.E., M.D.

OUR subject to-night is that of the "anxiety states." Amongst the mental disturbances which have occurred as a result of war strain the anxiety states have received little attention. Publications during the war dealt more with hysterical conditions, paralyses and contractures, deformities and loss of motion and sensation. But of the 4,000 cases admitted to the hospital at Maghull less than 20 per cent. exhibited a conversion hysteria, and even in many of them an anxiety state was frequently disclosed on examination.

In some the symptoms of the anxiety state—depression, irritability, disturbed attention, terrifying dreams and emotional outbursts—were complained of by the patient; in others the contracture, tremor or other hysterical condition was the more obvious, but after it had been got rid of the anxiety would be revealed; it had existed before, but the attention of the patient had been focused on the physical disability. In others, again, the physical condition proved to be obstinate, but it disappeared when treatment of the anxiety condition had been provided, and without any special attention being given to the hysterical symptoms.

One case to illustrate this may be mentioned. On admission the man complained of weakness and pain in the left leg, and he limped constantly. On his way to the hospital he had been told by the escort that he was going to an asylum. This frightened him, and he decided to complain only of this physical disability, hoping to be discharged in a short time. He asked for some work, and was given charge of my car. He took excessive care of the car so that he might "occupy his mind." At last he asked for his discharge, but, not being satisfied, I asked his medical officer to examine him again. At the interview he confessed that he had bluffed us all, and apologized for having done so. He admitted at the same time that he had had a great dread of becoming insane, and also that every night he saw a face at the foot of his bed. Here, therefore, there was anxiety and a definite hallucination. But the most interesting point was that the weakness and pain disappeared, and the limp was not seen again after this explanation. Many other cases might be cited to illustrate the importance of examining for anxiety states, and there can be no doubt that they would be found in a majority of the cases.

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[1] At a meeting of the Section, held February 12, 1920.

To-night, however, I do not intend to enumerate symptoms to you, but rather to inquire into the mechanisms concerned in the production of the anxiety states.

It is now recognized that with every stimulus which passes the threshold of consciousness there is to be recognized not only an intellectual but also an emotional factor. Försterling has suggested that a sensory stimulus which acquires a psychic quality at the moment of perception, brings with it not only an intellectual addition but also another quality, a feeling tone, and as a perception is laid down as a memory picture, so is its feeling tone always in close touch with the memory picture. When this is recalled, whether through a fresh stimulus or an antecedent memory picture, the feeling tone will be reawakened at the same time. The incident is therefore stored up in memory to be reawakened by an appropriate stimulus, and when the memory of the incident recurs the emotional state, which was originally associated with it, will be reawakened.

Under ordinary conditions we possess a more or less satisfactory control over these memories and emotions; this is developed as we grow up. But if because of fatigue, intoxications or intense shock this control is weakened, the memory will be reawakened by much slighter stimuli, and by repetition a habit of mind will be acquired so that in severe cases the patient may be dominated by the disturbing influence.

But not infrequently the emotional condition is produced without the memory of the original incident coming into consciousness. In one case the patient, after a severe illness following a septic wound, would experience an intense depression between 4 and 6 p.m. if alone and unoccupied. While in this state past, present and future appeared black, and he was acutely miserable. Exploration showed that during his illness he was worse, physically and mentally, from 4 to 6 in the afternoon. The emotional tone connected with this was stored in his memory, but no thought of his illness came into consciousness, and therefore the relation of cause and effect was not recognized. When this relation was understood by him and he reasoned out the whole matter, his attacks of depression disappeared, and he has not had them since, excepting when ill or fatigued. Even then the depression ceases immediately he remembers the cause and realizes the mechanisms concerned.

The day after this case was related to me I mentioned it to a patient, and at the end I asked her: "What does that suggest to you?" She said "I was trying to commit suicide from midnight to 4 a.m." Every night at midnight she left her bed, wandered about the dormitory, and refused to return to bed until about four o'clock. I asked her to send me a note as to how she slept the next night, and her message was: "Slept well, thank you." Here again the relation of cause and effect had not been realized, but the nightly disturbance ceased when she understood.

The reaction is possibly the result of a time stimulus similar to that obtained by Pavlov in his experiments on dogs. In these experiments reflex reactions, often involving some of the higher psychic mechanisms, were obtained by repetition of stimuli under certain definite conditions.

Now in these cases we find that past experiences with which an intense emotional tone was associated, whether recalled in consciousness or not, can give rise to emotional outbursts which disturb ordinary mental activities, and these outbursts may be expected to persist because they are not understood by the patient.

From our own experience we know that where the emotions are there the attention will be—and, as in the conditions we are considering, there is a feeling of anxiety because of the altered mental activities, the patient, instead of observing and estimating the value of phenomena affecting himself as he once did, lives with a narrowed field of consciousness, develops a dread as to possible consequences, and with keen anxiety watches for any sign which may confirm his fears. Trifling disturbances, which usually would pass almost unnoticed, acquired an undeserved importance; the disturbances of the mental processes of perceiving, thinking, feeling, judging and acting, become more frequent, and as the origin, and the mechanisms underlying such changes are not understood, it is difficult to see how an anxiety state can be avoided or overcome. This initial anxiety, due to the altered mental processes, becomes intensified by the repetition of the condition and by the dread of possible serious sequelæ. This dread is, as might be expected, present in the majority of cases, and frequently is an important factor in the development of the illness.

Such emotional conditions must be considered as normal reactions under the circumstances. Our emotions are always specific; an insult begets a feeling of anger; from success there springs a feeling of satisfaction; a shell explosion cannot but give rise to fear. And if the emotions constitute an important factor in our defence mechanisms they must be specific and unalterable except in degree.

The following case will illustrate some of the points I have laid before you. A soldier who had served for a considerable time in France became ill, and went to see the medical officer. The officer told him there was nothing the matter with him, and ordered him to return to the front line. He proceeded towards the line, but the illness developed and he was taken to a hospital with an acute pneumonia. As he recovered, the idea that the officer had practically told him he was a malingerer recurred frequently, and a feeling of revenge appeared. With the weakened control due to his illness this idea, with its associated emotion, repeated itself, until he became almost dominated by it. He was depressed, and unable to take an interest in his surroundings; he had disturbing dreams in which the medical officer often appeared, and his physical condition was unsatisfactory. It was a long time before he would allow this disturbing idea to be known, but improvement soon followed the exploration and explanation of the mechanisms concerned. This case showed also the way in which patients watch for signs which can confirm their fear of something serious existing as a cause for their illness. He complained one day of pains in his legs, and said he had not infrequently been troubled in a similar manner. Investigations proved that the pains in his legs were due to his having spent some time at the unusual exercise of kicking a football about. The pains were diminished by the time he mentioned them to me, but although he appreciated the relation of cause and effect when the explanation was offered, until then he saw in them only a confirmation of his fear.

There existed in this man therefore an unpleasant memory, with its specific and proper emotion, which by repetition had led to the development of a habit of mind that, with his lack of knowledge, he was quite unable to overcome, and this rendered him incapable of adapting himself to his surroundings. His condition was based on a bit of personal experience, as all mental illnesses are. There is no special mechanism for becoming mentally ill, and no gross organic lesion need be assumed.

I agree with Freud when, in discussing the defence neuro-psychoses, he says: "In the patients whom I have examined there existed psychic health until the moment when some incompatibility occurred in their ideation, that is, until there appeared an experience, idea or feeling, which evoked such a painful effect that the patient decided to forget it, because he did not trust his own ability to remove the conflicting resistance between the unbearable ideas and his ego." We might add: "if he could forget—and eliminate."

It is these unbearable ideas which are the cause of the primary disturbance of the mental processses which characterize mental illnesses, and which lead to the anxiety state with the varied symptoms that exist during this early phase of the disturbance.

All the other phenomena which provide the symptomatology of mental illnesses as seen in practice and as compiled and tabulated in our text-books are secondary products, that is, the symptoms by which we recognize hysteria, psychasthenia, phobias, melancholia, manic-depressive conditions, epilepsy in many instances, paranoia and the so-called dementia præcox, are secondary products superimposed on the primary disturbances which characterize the anxiety states. They indicate the attempts on the part of the patient to adapt himself to his altered mental condition, his rationalizations to explain his condition to himself or to escape from his torment.

The anxiety state is the primary illness, recovery from which is possible in a large number of instances if it be adequately treated, and therefore it is not to be viewed with the feelings of helplessness and hopelessness so prevalent at the present day.

Dr. ROSS: I agree with Dr. Rows' valuable paper: I would point out that not only is the complex forgotten, but when a symptom has suddenly appeared the complex stimulator which brought about its onset is very soon forgotten also. For example, a man has had a sudden return of symptoms while he was walking over a common. He has seen no one and there has been nothing to upset him. But on carefully thinking the matter over he has remembered seeing some bare trees, and then his attack comes on. On free association of the idea of these trees he is led back to a distressing incident in the war. I also agree with Dr. Rows on the question of men clinging to minor symptoms after their major symptoms have disappeared, and I suggest that the absence of stimulus which the indefinite pension causes is largely responsible for this.

Section of Neurology.

President—Dr. W. Aldren Turner, C.B.

The Results of Secondary Suture of Peripheral Nerves.[1] [2]

By John S. B. Stopford, M.D.

(I) Introduction.

Although further time must elapse before the end-results of secondary suture of peripheral nerves, injured in the war, can be judged in large numbers, it is now possible to communicate a preliminary report upon the progress to date of those patients in which this method of surgical treatment has been found necessary; and it seems necessary to do this for the benefit of the many pensioners who still await further treatment.

So many factors may influence the progress and extent of recovery that it is exceedingly difficult to interpret all the observations, or estimate the influence of any one individual factor. To attempt the elucidation of the numerous problems encountered, or to provide any guidance for the future, it is obviously necessary to consider large numbers of patients on whom regular systematic examinations have been made and careful records kept of all complications, methods of treatment, and progress. No reliable conclusions can be drawn from the publication of a few isolated cases, or even from a large series, unless attention has been directed to all the factors which may prevent or cause delay or acceleration of recovery. The closest co-operation between the surgeon and the investigator is essential; if the investigator is unable to be present at the operation, it is of fundamental importance that the most detailed notes should be provided of the anatomical and pathological conditions found on exposure of the nerve, together with an exact account of the surgical procedure adopted. It has been my good fortune to have the fullest possible support from my surgical colleagues at the Grangethorpe Hospital, Manchester, and I would like to express my deep appreciation of the careful and elaborate system of note-taking which they have instituted, as, without the information supplied by them, it would have been impossible to attempt an inquiry into the problems of secondary suture.

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[1] At a meeting of the Section, held March 11, 1920.

[2] This paper will be published *in extenso* in *Brain*, Summer Number, 1920.

In this report are included all the results of secondary suture, where it has been possible to continue observations for a period of not less than six months after the date of the operation. It was rarely possible to follow the patients even for so short a time before 1917, when the special hospitals were instituted; consequently almost all the patients quoted have been operated upon since the beginning of that year, and only a small number can be considered as end-results.

Altogether 271 examples of end-to-end suture are under review, and the majority of the patients have been examined at regular intervals of from four to six weeks for varying periods up to three years after the date of the performance of the suture.

As a preliminary it is convenient to consider the following general factors which may influence the progress and result.

(1) *The Interval which has elapsed between the Reception of the Injury and the Date of the Suture.*—It is necessary, in connexion with this, to take into account the cause of the delay. If the operation has been deferred because of the presence of sepsis, widespread intraneural changes are likely to be encountered which will interfere seriously with, or prevent, regeneration. Excluding such a condition, a delay of twelve to eighteen months appears to have no marked effect upon the date or extent of recovery. If the interval exceeds this time, in my experience the prognosis is not so good when the suture has been performed in the distal part of the limb; whereas, in the proximal part, a delay of two or three years does not seem to prejudice the chances of success. Suture of the median in the upper arm has been successful in two patients where there was an interval of two and a half years. A delay of three and a half years (the longest encountered) occurred in two patients, with injury to the median at the elbow. In one the only muscle to recover voluntary power was the flexor sublimis digitorum; in the other patient it is too early yet to give an opinion, but the signs are not hopeful.

(2) *The Surgical Technique adopted.*—In about 80 per cent. the

same procedure has been used; so that in this series this factor is almost constant, and, consequently, no opinion can be expressed upon the advantages of the various suture materials. After free exposure, the extremities have been excised until distinct nerve bundles could be seen, and then the ends brought into as perfect apposition as possible without distortion. Whenever practicable, only sheath sutures of catgut have been used, but in the larger proportion one tension suture through the substance of the nerve was found necessary. On comparing the progress and results, with and without tension sutures, there is no doubt that the effect of the through-and-through stitch is perceptible.

For some time my surgical colleagues have used no protective material to surround the line of suture; a new bed for the nerve has been constructed, or its anatomical course modified, so that the injured segment of the nerve lies only in relation with uninjured muscles. The clinical records clearly indicate the advantages of this over any protective material, either autogenous or heterogeneous. Several nerves have been re-exposed by my colleague, Mr. H. Platt, after various materials have been used, and it has been proved that neither fascia lata nor fat prevent adhesions, and in two patients, where Cargile membrane had been used, there was a thick collar of fibrous tissue around the nerve which in one was undoubtedly causing considerable constriction.

The prognosis is bad when a satisfactory bed cannot be procured, and the nerve has to be left in relation with scar tissue, a condition unfortunately encountered frequently when the external popliteal nerve has to be sutured in the region of the head of the fibula.

Displacement of the ulnar nerve in front of the internal condyle of the humerus has not, as far as I can ascertain to date, retarded the progress.

It is somewhat surprising that the extensive freeing of the nerve necessary in this manœuvre, or in other nerves when a large defect has to be bridged, has not had a more marked effect. The records I have are insufficient as yet to enable me to give an opinion upon the results after the two-stage operation.

The after-treatment has been the same—constant support of the paralysed muscles, interrupted galvanic stimulation of the muscles and massage, and later faradic stimulation and muscle re-education.

(3) *The Condition of the Nerve found on Exposure at Operation.*—Those patients in whom a complete solution of continuity was discovered

at operation have tended to make a more complete recovery than those in which incomplete division was found, but I am convinced that this difference is very clearly reduced, if not eliminated, when a liberal resection is performed. Interstitial changes are frequently very extensive after partial division, and from the histological examination of pieces of nerve excised at operation there appears to be a tendency not to resect sufficiently widely. Very free resection is particularly necessary after prolonged suppuration. Contractures and other mechanical disabilities are associated commonly with incomplete division and may be responsible for imperfect functional recoveries, although good regeneration may have succeeded the suture of the nerve.

(4) *The Effect of some of the more frequent Complications.*—Allusion has been made to the fact that extensive and prolonged sepsis in the neighbourhood of the nerve may seriously influence the prognosis. Microscopic examination has shown that, under such a condition, intraneural changes may be discovered as distant as 8 in. above the point of injury.

Ligature of the main artery in the proximal part of the limb, as the upper part of the brachial or the axillary, tends to retard the progress and, after suture of the ulnar or median nerves, appears sometimes to reduce the extent of the recovery of function, but it is difficult to exclude the possibility of this being due to secondary changes in the muscles as a result of the ischæmia. Involvement of the main blood-vessels in the distal part of the limb, as may be anticipated, does not usually affect the result.

Ununited fractures of the humerus present a serious complication. In four patients exhibiting this complication suture of the musculo-spiral was performed before the ends of the bones had been fixed. Two cases were complete failures, the third showed merely recovery in the supinator longus and extensor carpi radialis longior, whilst the fourth progressed favourably until a bone-graft operation was succeeded by a recrudescence of sepsis, which resulted in complete loss of conduction and failure of regeneration. These experiences seem to prove that where an ununited fracture of the humerus complicates a musculo-spiral injury it is advisable to treat the bone lesion first.

(II) ANALYSIS OF RESULTS.

(*a*) *General.*—In none of my cases of secondary suture can the result be described as perfect; in the best there has been an appreciable difference in power on comparing the two sides and at least an incomplete recovery of epicritic sensibility, even three and a half years after the date of the suture. The recovery of sensation has been particularly disappointing. More successful results have followed suture in the proximal part of the limb than the distal. Of sutures in the upper extremity, the failures in the upper arm amounted to 12 per cent., whilst in the forearm they amounted to 24 per cent. At both levels the recovery of voluntary power has been much less constant for muscles supplied by the nerve in the distal than in the proximal part of the limb. For obvious reasons muscles which have a large nerve of supply have a better chance than those in which the branch is smaller.

Exclusive of mechanical disabilities and the general factors already considered, four chief causes for imperfect recovery are readily recognized.

(1) Intraneural changes in the proximal end, which may occur irrespective of the presence of sepsis.

(2) Obstruction to the downgrowth of new nerve fibrils.

(3) Destruction of branches in addition to the injury to the nerve trunk. This is particularly prominent in wounds of the median in the upper third of the forearm.

(4) "Bad shunting" (efferent fibres passing down to afferent terminals or vice versa), which, with the most perfect surgical technique, must occur to some extent in almost all secondary sutures, since a considerable portion of the nerve trunk has to be excised and the intraneural anatomy disturbed. Such a factor will be of less consequence in the musculo-spiral than in the ulnar or median. It is possible that the persistence of Tinel's sign without any recovery of sensation may be due to afferent fibres descending efferent pathways.

(5) In addition to these there is an important but less obvious fifth cause of impaired function, which is particularly significant after suture of the median, and is probably one explanation of the poor functional result which usually follows suture of both the median and the ulnar. It is not uncommon to discover, after suture of the median, that all the muscles—when tested individually—have recovered voluntary power, and yet the hand is of slight practical service, when the patient attempts purposive movements. The patient will often volunteer the information that when he attempts to work he finds that he loses the grip of his tools, and on inquiry it will be found that he can use the hand fairly well as long as he concentrates upon the movements, but that the hand ceases to function satisfactorily as soon as he takes his eye off it. Such patients also frequently inform you that the hand is useless

in the dark, or when they cannot watch what they are trying to do with it. Such complaints may be heard even when cutaneous sensibility has made a fair recovery and all the muscles, tested individually, exhibit voluntary power. It seems clear that such a disability is due to the loss of afferent stimuli from joints, muscles, tendons and other deep structures. It is only practicable to investigate directly the recovery of fibres conveying conscious impressions from such deep structures as joints, but from an investigation of the sense of posture and the appreciation of passive movements in the finger and thumb articulations supplied by the nerve, it has been found that these were generally lost, or at the best very defective even when three years had elapsed since the time of the suture. It would seem that this is very important to remember at examinations for the assessment of pensions after nerve suture in the upper limb, since the routine investigations of voluntary power and cutaneous sensibility do not, in themselves, provide sufficient information to determine the real functional capacity of the hand. It is also an awkward problem to contend with during the later stages of recovery when muscle re-education becomes of greatest importance, since it provides one of the main obstacles to the satisfactory application of this form of treatment—an obstacle which is probably insufficiently appreciated. Previous to the war our chief experience of muscle training was derived from the treatment of infantile paralysis, and consequently many who are responsible for the supervision of muscle training are content merely to develop an increased range of movement of the individual muscles, and fail to realize the different problem which arises in peripheral nerve injury. After injuries to the peripheral nerves there is usually a serious loss of those afferent stimuli which are of such importance for the perfect performance and adjustment of the finer and more delicate movements, and consequently in the later part of the treatment every effort must be made to develop those purposive and more complex movements which the particular patient will require when he returns to a civil occupation.

(*b*) *Explanation of Charts.*—The vertical columns, 1 to 24, refer to the months after the performance of the suture; in the last column, "delay," reference is made to any case where an interval of eighteen months or more has occurred between the date of the reception of the wound and the time of the operation; the figures express this interval in months. In all the charts reference is made, unless otherwise stated, to the return of voluntary power, since in my experience this has been more frequently discovered at an earlier date than the recovery of faradic response. The muscles are recorded by means of the initial letters, viz. :—

SL = supinator longus.

As regards sensation information is conveyed by the following abbreviations and letters :—

P = commencement of recovery of protopathic sensibility.
P.rec. = complete recovery of protopathic sensibility.
E. = commencement of recovery of epicritic sensibility.

The interrupted lines in certain columns mark the date of the last examination, unless they occur in the last column, in which case several of the patients have been under observation for further periods, but the intervals between the later examinations have been longer than four to six weeks.

(III) CONCLUSIONS.

(I) The following appear to be the most important factors which may influence the prognosis:—

(1) Sepsis.

(2) Site of the suture, the prognosis being more favourable the nearer the suture is to the spinal cord.

(3) A delay of more than eighteen months before the suture is performed in the distal part of a limb prejudices the chances of recovery, but is not of so much consequence in the proximal part.

(4) "Bad-shunting" is probably a common cause of imperfect recovery in such nerves as the ulnar.

(5) Destruction of branches of the nerve, in addition to injury to the trunk, may be a cause of incomplete recovery.

(6) Ununited fractures, especially of the humerus in injuries to the musculo-spiral, are a serious complication. Evidence appears to be in favour of primary fixation of the bone.

(7) Ligature of the main artery in the proximal part of a limb may delay and limit recovery.

(8) Imperfect recovery of afferent fibres from such structures as joints, muscles and tendons, is probably an important cause of limited functional recovery of the hand for the performance of purposive actions, although the voluntary power of individual muscles is good.

(II) The intrinsic muscles of the hand and the muscles in the distal part of a limb recover less frequently than the larger muscles in the proximal part of a limb. Probably several factors contribute to the cause of this :—

(1) Distance which the young nerve fibrils have to travel away from their trophic centre in the spinal cord to reach the muscle.

(2) The size of the nerve of supply. The larger the nerve the greater the chance the young fibrils must have of reaching the muscle.

(3) Possibly the phylogenetic history of the muscles, since there does seem to be some support of the hypothesis that more recent acquisitions are the more vulnerable.

(III) The following points bearing on the surgical technique can be deduced :—

(1) It is advisable to avoid the through-and-through suture, whenever practicable, as proved also by the experimental work of Sargent and Greenfield.[1]

(2) The construction of a new bed for the nerve, after suture, is preferable to any autogenous or heterogeneous protective material. The unsatisfactory bed after suture of the external popliteal nerve, in the vicinity of the head of the fibula, is probably a cause of many failures.

(3) Displacement of the ulnar nerve in front of the internal condyle of the humerus or widespread freeing to procure apposition of the ends do not appear to retard the recovery.

(4) Liberal resection of the injured ends is essential and particularly necessary if sepsis is prominent.

(IV) No recovery after the performance of secondary suture could be regarded as perfect. The chief causes of the imperfect result after *secondary* suture appear to be :—

(1) The fact that an appreciable portion of the nerve trunk has to be resected causes inevitably a disturbance of the intraneural anatomy, however carefully the surgical procedure is carried out.

(2) After secondary suture the nerve is making a *second* effort to regenerate, the first attempt being represented by a neuroma or outgrowths along fascial planes.

(3) The almost constant occurrence of one or more of the foregoing factors which have been found to influence the prognosis.

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[1] Sargent, P., and Greenfield, J. G. "An Experimental Investigation of Certain Materials used for Nerve Suture." *Brit. Med. Journ.*, 1919, ii, 407.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 1				S.L.	E.C.R.		E.C.U.		E.C.D.				E.O.M.P.	E.L.P.		E.B.P.								-----	
,, 2				S.L.		E.C.R.	E.C.U.	E.C.D.	E.O.M.P.			E.L.P.												-----	
,, 3					S.L.		E.C.R.			E.C.U.				E.L.P.		E.O.M.P.	E.C.D.								
,, 4						S.L.	E.C.R.	E.C.U.				E.C.D.		-----											
,, 5				S.L.	E.C.R.	E.C.U.	E.C.D.	-----																	
,, 6				S.L.	E.C.R.	$\overset{3}{\text{E.C.D.}}$	E.C.U.				$\overset{1\,2\,3.4}{\text{E.C.D.}}$	E.O.M.P.	E.L.P.	E.B.P.		-----									
,, 7				S.L.	E.C.R.		E.C.U.	E.C.D.			E.O.M.P.		E.L.P.												
,, 8						S.L.		E.C.R.					E.C.U.		E.C.D.	E.O.M.P.			E.L.P.				E.B.P.	-----	
,, 9			S.L.		E.C.R.			E.C.D.			E.O.M.P.	E.L.P.													
,, 10				E.C.R.	E.C.U.	E.L.P.	E.O.M.P.	$\overset{1\,2}{\text{E.C.D.}}$																	
,, 11				S.L.	E.C.R.		E.C.U.	E.O.M.P.						E.C.D.					E.L.P.		E.B.P.			-----	
,, 12				S.L.	E.C.R.		E.C.U.									E.C.D.		E.O.M.P.		E.B.P.				-----	
,, 13			S.L.	E.C.R.				E.C.D.																	
,, 14		S.L.	E.C.R.																						
,, 15				S.L.	E.C.R.			-----																	
,, 16			S.L.			E.C.R.	E.C.U.	E.C.D.			-----														
,, 17											E.C.R.					E.O.M.P.	E.L.P.			-----					
,, 18			E.C.R.		E.C.U.																				
,, 19								E.C.R.	E.C.U.											E.C.D.		E.O.M.P.		E.L.P.	
,, 20			E.C.R.																						
,, 21			E.O.M.P.																						
,, 22				S.L.																					
,, 23													Failure							-----					
,, 24												Failure							-----						21
,, 25														Failure								-----			19

Chart I.—Musculo-spiral nerve (lower third of arm).

The numerals above E.C.D. refer to the first (index), second, third and fourth (little) fingers.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 26				S.L.	E.C.R. E.C.U.				E.C.D.	E.O.M.P.	E.L.P.			E.B.P.			-----								
,, 27				S.L.	E.C.R.	E.C.U.					E.C.D.		E.O.M.P.												
,, 28					S.L.		E.C.R. E.C.U.			E.O.M.P.		E.C.D.				E.L.P.								E.B.P.	
,, 29					S.L.	E.C.R.		E.C.U.	E.O.M.P.																
,, 30						S.L.		E.C.R.			E.C.U.			-----											
,, 31									E.C.R.		S.L.				E.O.M.P.						----				
,, 32					S.L.	E.C.R.		E.C.D.	E.C.U.		E.O.M.P.	E L P.		E.B P.			----								
,, 33					E.C.R.	S.L.	E.C.U.				----														
,, 34					S.L.	E.C.R.		E.C.U.																	
,, 35				S.L.	E.C.R.		E.C.U.		----																
,, 36			S.L.	E.C.R.		----																			
,, 37					S.L.	E.C.R.																			
,, 38				S.L.		E.C.R.																			
,, 39			S.L.			E.C.R.																			
,, 40													E.C.R.		E.C.U.	3.4. E.C.D.							----		
,, 41								S.L.	E.C.R.		E.C.U.				E.O.M.P.		E.C.D.		E.L.P.					----	
,, 42				S.L.																					
,, 43																					----	All	acting		
,, 44					E.C.R.		----																		
,, 45																				S.L.	2 ½ years E.C.R.	E.C.U.	E.C.D.	E.O.M.P.	
,, 46												Faradism S.L.	E.C.R.	E.C.U.	E.C.O.			Nil Vol:			----				
,, 47						S.L.	E.C.R.					Bone graft went septic —— Failure											----		
,, 48												E.C.R.	S.L.						E.C.U.		Ununited Fracture				
,, 49																	Failure				Ununited Fracture				
,, 50														----		Failure		–	Ununited			Fracture			
,, 51																		Failure			(2 yrs.)		Bulb.	----	24

CHART II.—Musculo-spiral nerve (middle third of arm).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Patient 52					S.L.	E.C.R.	E.C.U.	E.C.D.			E.O.M.P.	E.L.P.			E.B.P.									
,, 53							S.L.	E.C.R.	E.C.U.									E.O.M.P.	E.L.P.	E.C.D.				
,, 54							S.L.		E.C.R.		E.C.U.	E.O.M.P.					E.C.D.			-----				
,, 55							S.L. E.C.R.		E.C.U.	E.O.M.P. E.B.P.		E.C.D.		E.L.P.							-----			
,, 56			Triceps							S.L.			E.C.R.			E.C.U.	-----							
,, 57						S.L.	E.C.R.	-----																
,, 58						E.C.R.	-----																	

Chart III.—Musculo-spiral nerve (upper third of arm).

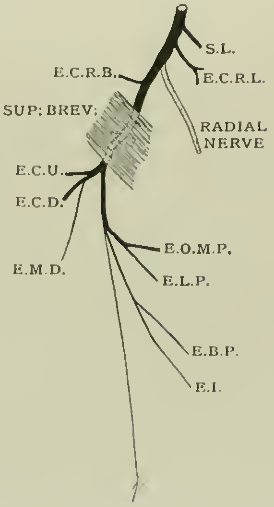

Fig 1.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.		1.	3.	9.	1.	2.																		
E.C.R.			3.	2.	8.	2.	2.	2.			1.													
E.C.U.					2.	1.	7.	1.	1.	1.			1.											
E.C.D.						1.	1.	6.	1.			1.		1.	1.	1.	1.			1.				
E.O.M.P.			1.				1.	1.	1.		2.	1.	1.			3.		1.				1.		
E.L.P.						1.						2.	2.	2.			1.		2.	1.				1.
E.B.P.														1.		1.					1.		1.	

Lower third (twenty-five cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.			2.	5.	5.	3.		1.			1.		1.											
E.C.R.				1.	5.	6.	2.	1.	2.			1.	1.											
E.C.U.					1.	1.	3.	2.	1.		2.				1.				1.					
E.C.D.								1.	1.		1.	1.				1.	1.							
E.O.M.P.									1.	2.	1.		1.		2.									
E.L.P.											1.	1.				1.			1.					
E.B.P.														2.										1.

Middle third (twenty-six cases).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
S.L.					1.	1.	3.			1.														
E.C.R.						2.	2.	1.	1.				1.											
E.C.U.							1.		2.		1.					1.								
E.C.D.								1.				1.				1.				1.				
E.O.M.P.										1.	1.	1.						1.						
E.L.P.												1.		1.					1.					
E.B.P.											1.				1.									

Upper third (seven cases).

Chart IV.—To show date of recovery of voluntary power of the various muscles at the three levels.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 59				P.							P.rec.							A.B.P.						-----	
,, 60					P.									P.rec.	A.B.P.	E.		-----							
,, 61					P.							P.rec.			E.			A.B.P.						-----	
,, 62					P.			P.rec.		-----															
,, 63					P.		A.B.P.				-----														
,, 64								P.	A.B.P.		P.rec.					E.				-----					
,, 65			P.					-----																	
,, 66				P.					-----																
,, 67				P.				P.rec.			-----														
,, 68				A.B.P.	P.							-----													
,, 69				P.											-----										
,, 70				P.		A.B.P.				P.rec.		-----													
,, 71			P.	A.B.P.						-----															
,, 72				P.						-----															30
,, 73				P.								-----													
,, 74			P.			-----																			
,, 75			P.			-----																			
,, 76			P.			-----																			
,, 77					P.												-----								
,, 78				P.								P.rec.													
,, 79					P.								-----												19
,, 80																	Failure								
,, 81																	Failure								18

Chart V.—Median nerve (wrist).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 82			$\overset{1.}{\text{F.S.D.}}$	P.			-----																		
,, 83				$\overset{1.}{\text{F.S.D.}}$	P.							A.B.P.													
,, 84				A.B.P.	$\overset{1.}{\text{F.S.D.}}$				P.				E.				-----								
,, 85					$\overset{1}{\text{F.S.D.}}$						P.	A.B.P.												-----	
,, 86				P.		$\overset{1.}{\text{F.S.D.}}$		-----																	
,, 87			P.	$\overset{1}{\text{F.S.D.}}$					-----																
,, 88						P.		$\overset{1.}{\text{F.S.D.}}$																	20
,, 89							$\overset{1.}{\text{F.S.D.}}$		P.					-----											
,, 90		P.			$\overset{1.}{\text{F.S.D.}}$																				
,, 91				F.S.D.	P.			F.P.D.		F.L.P.		E.		-----											
,, 92							P.	F.L.P.		F.S.D.	F.P.D.	P.rec.			A.P.B.			E.						-----	
,, 93				F.S.D.	P.R.T.			F.L.P.	P.						P.rec.										
,, 94				F.C.R.	$\overset{2.3.4.}{\text{F.S.D.}}$	P.			F.L.P.	F.P.D.								$\overset{1.}{\text{F.S.D.}}$						-----	
,, 95				F.L.P.														F.S.D.						-----	
,, 96			P.	F.S.D.														-----							41
,, 97										P.			F.C.R. F.L.P.				P.R.T.			-----					
,, 98			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 99					P.R.T.	F.C.R.					P.	-----													
,, 100						F.S.D.					F.L.P.		-----												20
,, 101										P.		-----													
,, 102				P.		-----																			42
,, 103																	All	acting	vol: –		3½ years Anaesth: -	only		index	
,, 104																	All	acting (except F.S.D.) -				2½ years Anaesth: distally			
,, 105																	All	muscles	acting –		2½	years Anaesth:		2 fingers	
,, 106															Nil vol:*		F.C.R. – F.S.D. – F.P.D. –				resp: to Farad:				
,, 107																		Failure							

CHART VI.—Median nerve (forearm).

Patients 82 to 90 below anterior interosseous branch; patients 91 and 92 above anterior interosseous, but below branches to flexor carpi radialis and pronator radii teres; patients 93 to 107 above all muscular branches. Numerals above flexors of fingers refer to first (index), second, third and fourth (little) fingers.

* Date when first examined after suture.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 108						P.R.T.	F.C.R.				P.	F.L.P.		F.S.D.				F.P.D.			A.B.P.	Prec.		----	
,, 109					P.R.T. F.C.R.	P.		$^{2.3.4.}$F.S.D.						$^{1.}$F.S.D.	Prec.					----					
,, 110					P.R.T.	F.S.D. P.	F.C.R.							F.L.P.	Prec.	A.B.P.					F.P.D.			----	
,, 111				P.	P.L.	F.C.R.	F.S.D.	F.P.D.		----															
,, 112				P.		$^{2.3.4.}$F.S.D.	$^{1.}$F.S.D.								F.C.R.		----								
,, 113					P.	P.R.T.	F.C.R.	$^{2.3.4.}$F.S.D.					----												
,, 114					P.R.T. F.C.R.	F.S.D.	F.L.P. P.	F.P.D.																	
,, 115						P.R.T. F.C.R.					P.	F.L.P. F.S.D.						F.P.D.						----	
,, 116							P.R.T.				F.S.D.	F.L.P.	F.C.R.												
,, 117					P.R.T.	F.S.D.		F.L.P.										F.P.D.		A.B.P.				----	
,, 118				P.		P.R.T.	F.C.R.																		
,, 119			P.R.T.	F.S.D. P.	F.C.R.	F.P.D. F.L.P.																			
,, 120			P.R.T.		P.		F.C.R.	F.S.D.	A.B.P.								Prec.								
,, 121							P.R.T.	F.C.R.	$^{2.3.4.}$F.S.D.			----													
,, 122				P.		P.R.T.	F.C.R.	F.S.D.		Prec.															
,, 123			P.R.T.	P.		$^{2.3.4.}$F.S.D.		$^{1.}$F.S.D.			F.L.P.														31
,, 124					P.																				
,, 125																			P.R.T.	- P.L.	F.C.R. -	F.S.D. -	* F.L.P. -	Prec.	
,, 126																P.R.T.	- F.C.R.	- F.S.D.		- F.P.D.*	- Prec.	- E.		----	19
,, 127														P.R.T.	- P.L.	- F.C.R.	- F.S.D.	* - F.L.P.		- A.B.P.	- Prec.				
,, 128												P.R.T.	- F.C.R.	* F.S.D.		- F.L.P.									
,, 129			P.R.T.	F.C.R.						F.S.D.	F.L.P.		----												
,, 130							F.C.R.	----																	

Chart VII.—Median nerve (arm).

* Date when first examined after suture.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 131							P.							A.M.D.		----									
,, 132						P.					P.rec.		A.M.D.									E.		----	
,, 133						P.				A.M.D.		E.			----										
,, 134			P.			A.M.D.		P.rec.	Inter.																
,, 135							P.						----												
,, 136							P.	A.M.D.																	
,, 137			P.			A.M.D.	P.rec.			Inter.										----					
,, 138															A.M.D.	P.	Inter.								
,, 139								P.																	
,, 140						P.												P.rec.			E.	----			
,, 141					P.		----																		
,, 142					P.	A.M.D.																			
,, 143			P.				----																		
,, 144						P.			----																
,, 145					P.	----																			
,, 146				P.				----																	
,, 147				P.				----																	21
,, 148					P.																				
,, 149								P.						----											
,, 150					P.					----															22
,, 151				P.		----																			18
,, 152						P.						P.rec.			----										
,, 153																				✳ A.M.D.	only.				
,, 154																				A.M.D.	- Inteross:		3 years No Sens: rec.		
	9 No evidence of regeneration 6 - 10 mths.																								
	7 Failures																								

CHART VIII.—Ulnar nerve (forearm).

Distal to branches to flexor carpi ulnaris and flexor profundus digitorum.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 171							P.		A.M.D.											P.rec.				-----	
,, 172			F.C.U.	F.P.D.	P.		A.M.U.		-----																
,, 173			F.C.U.	F.P.D.		P.								-----											
,, 174				P.						----															
,, 175				F.C.U.			F.P.D.				P.													-----	
,, 176						F.C.U.																			
,, 177						F.C.U.														P.			A.M.D.		
,, 178				F.C.U.									F.P.D.				A.M.D.							-----	
,, 179						F.C.U.																			
,, 180			F.C.U.						F.P.D.							-----									
,, 181			F.C.U.			-----																			
,, 182								P.																	
,, 183		F.C.U.			F.P.D.			A.M.D.																	
,, 184											F.C.U.	P.	F.P.D.									P.rec.			
,, 185				F.C.U.	P.	F.P.D.																			
,, 186			F.C.U.		F.P.D.			A.M.D.		-----															
,, 187						P.					-----														18
,, 188									F.C.U.																
,, 189				F.C.U.	F.P.D.																				
,, 190				F.C.U.	P. F.P.D.					A.M.D.		-----													
,, 191				F.P.D.								-----													
,, 192				F.P.D.	P.																				18
,, 193		F.C.U.	F.P.D.		P.								-----					-----							
,, 194			F.C.U.	F.P.D.		----																			
,, 195						F.C.U.		F.P.D.					----												
,, 196			F.C.U.										----												
,, 197				F.C.U.			----																		
,, 198			F.P.D.	P.																					
,, 199				F.C.U.				----																	
,, 200																						F.C.U. only *			
,, 201																			F.C.U. * & F.P.D. only						
,, 202																	F.C.U. * & F.P.D. only								
,, 203																		F. C. * U. only							
,, 204																		F. C. U.-* F. P. D. & Inteross: Prec							18
,, 205																				F.C.U. - F.P.D. - A.M.D. & P. *					
,, 206																			F.C.U. & F.P.D. only				3½ years		
								8 Failures & 2 nil at 6 months																	

CHART IX.—Ulnar nerve (arm).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Delay
Patient 217											Gastr:			E.L.D.	Peron:	T.A.		----							
,, 218								P.	Gastr:	T.A.		-----													
,, 219				P.										Gastr:											
,, 220													Gastr:		T.A.		E.L.D.			Peron:				-----	
,, 221												T.A.								Peron:	Gastr:		-----		
,, 222																P.		Gastr:						-----	
,, 223									Gastr:	T.A.												All	acting	2½ yrs.	
,, 224														Gastr:		-----									
,, 225					Gastr:												P.						27 mths. Same		
,, 226						Gastr:	T.A.					E.L.D.			P.										
,, 227					Gastr:	P.				T.A.							E.L.D.	-----							
,, 228							P.	Gastr:	T.A.	E.L.D.		-----													
,, 229					P.							Gastr:	T.A.	-----											
,, 230										Gastr:											P.			-----	
,, 231					P.			Gastr:	T.A.		----														
,, 232				P.		T.A. Gastr:	E.L.H.																		
,, 233								Gastr:			----														
,, 234					Gastr:	P.					Prec.														
,, 235					P.										Gastr:			T.A.			-----				
,, 236						P.		Gastr:																	
,, 237					Gastr:			T.A.	-----																
,, 238								Gastr:					----												
,, 239													----												
,, 240				P.		----																			
,, 241				P.	Gastr:						----														24
,, 242				P.			----																		30
,, 243																						Gastr:	2½ years only		
,, 244																				Failure					
,, 245																						Failure			
,, 246																						Failure			
,, 247											----														

Chart X.—Sciatic nerve.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	Delay
Patient 248						P.		T.A.				E.L.D.	Peron:													Prec.	
,, 249											P.		T.A.		Peron:						----						
,, 250					T.A.										P.	Peron:	E.L.D.	E.L.H.								----	
,, 251							T.A.	E.L.D.																----			
,, 252				P.		T.A.	Peron:					E.L.D.								Prec.						E.L.H.	
,, 253						P.							----														
,, 254				P.		T.A.							Prec.	----													
,, 255						T.A.	Peron:																				
,, 256										P.					Peron:		Prec.								T.A.	----	
,, 257			P.		T.A.	E.L.D.	Peron:		Prec.					E.L.H.				E.						----			
,, 258							----																				
,, 259							----																				
,, 260													----														27
,, 261																					Nil vol:		All respond to ✳ Farad:				
,, 262										----																	
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CHART XI.—External popliteal nerve.

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Patient 270							Gastr:											T.P.		F.L.D.				----
271					Gastr:			F.L.D.				T.P.										----		

CHART XII.—Internal popliteal nerve.

DISCUSSION.

Mr. HARRY PLATT, M.S.: Professor Stopford's careful investigations show that on the whole, the changes in the clinical syndrome indicating regeneration after end to end suture, are comparatively slight even after considerable periods of time. It is therefore easily understood that mistakes can be made by superficial observers; further, true motor signs of recovery have to be distinguished from the so-called trick movements with which we are now all familiar. The reported successful nerve-graft operations and other forms of nerve bridging, in most cases can be explained by the deceptive effect on the observer of such trick movements. Final end results after nerve-suture operations are not yet available. Therefore we are immediately concerned with a tangible problem—the complete failures. In these we have to decide whether re-exploration is worth while. In my own work I am acting on the principle that it is always well worth while. The second operation is often of great diagnostic value, as it has been my experience on several occasions to re-explore a nerve which after suture has remained "dead" for over a year, and to find that excellent conduction was present as tested by direct faradic stimulation. Such findings emphasize the great value of the routine use of the electrodes during all nerve operations. One has been interested in a few cases to find somewhat paradoxical electrical phenomena—viz., in complete anatomical lesions of long standing the retention of faradic response in muscle bellies supplied by the distal part of the nerve trunk. Perthes of Tübingen has recently reported a series of similar observations. The constant use of faradic excitation at operations has made one sceptical of the correctness of the classical laws of muscle response as laid down from tests carried out through the skin.

Mr. JOCELYN SWAN: The results I have obtained from end-to-end suture of peripheral nerves are mainly in accord with those detailed by Professor Stopford. There is no doubt that the best functional result is obtained with injuries of the musculo-spiral nerve or of the posterior cord of the brachial plexus, whilst those obtained in lesions of the external division of the sciatic are never very favourable. In cases of the median or of the ulnar nerve, suture does not hold out such good prospects, as voluntary movement of the intrinsic muscles of the hand is seldom complete. I am sure that end-to-end suture is the operation to be aimed at in all complete nerve divisions, or in those cases in which such a fibrous block results from the injury that no evidence of recovery is present after prolonged and efficient treatment, and in those cases in which, when the proximal part of the nerve is exposed at the operation, no reaction is obtained on direct stimulation of the nerve by faradic current. I have obtained approximation of the nerve where the gap is considerable by transplanting the nerve to a new position, as by bringing the ulna in front of the humeral condyle or by transposing the musculo-spiral to to the front of the humerus, and in some cases I have brought the ends together by considerable flexion of the limb, correcting the position subsequently. There remain cases, however, in which end-to-end suture is impossible; in the musculo-spiral or posterior interosseous nerve it is far better to abandon the operation on the nerve and to perform tendon transplantation. from which I have obtained excellent functional results. In the case of the median in the arm, I should not hesitate to resect a portion of the humerus, subsequently placing the latter to obtain end-to-end apposition, but I would not shorten any other bone for this purpose, especially in the lower limb—in fact, I have seen no necessity to do so. As to the technical details of the operation performed on the nerve, wherever possible I advocate the complete removal of fibrous tissue, so that the newly-joined nerve may lie in a bed of comparatively healthy tissue, even to the extent of removal of muscular planes so that freshly cut muscle surfaces are obtained. I have used very fine Cargile membrane very extensively and see no reason against it. In a large series of cases I have only seen it extruded twice, and for this I am inclined to blame myself rather than the membrane. In cases in which secondary operations are performed upon nerves previously exposed, I have noticed less infiltration and scar tissue where membrane has been used than in those in which no separate covering had been employed. In cases in which the nerve function is subcutaneous, as over the head of the fibula, and again in cases in which operation is necessary for causalgia, I substitute a fat graft for the membrane, and in cases in which the fornia nerve is in close proximity to a bone, as frequent in the musculo-spiral nerve in the middle of the arm, I have made use of a flap of muscle tissue, placed between the bone and the nerve. For the suture of the ends of the nerve very fine linen thread is used; one or two sutures are used directly through the nerve where any tension exists, supplemented by careful approximation of the nerve-sheath around the junction. In many cases the tension suture is removed when the suture of the sheath is completed. I prefer fine thread to catgut, which has been so freely advocated, and am glad to see that the experimental work done by Colonel Sargent at Tooting supports my contention. I desire to emphasize the necessity for free removal of the fibrous tissue at the proximal end of the nerve until healthy nerve bundles are exposed. I have seen no good result from the lateral anastomosis of the ends of a divided nerve into a neighbouring nerve, but I have seen improvement follow the direct transplantation of a nerve into the interval between the ends of a divided nerve, and do not look upon this operation as one to be entirely condemned. I regard the subsequent treatment after operation as of as much importance as the operation itself. The attention to the mobility of joints and tendons, the nutrition of the limb by massage and electrical stimulation, and above all the distraction of the mind of the patient from his injury and apparent slowness of his recovery by means of useful occupation, together with the encouragement of his attendant when recovering function becomes apparent. It is by no means uncommon to see functional paralysis associated with recovering organic disability.

Section of Neurology.

President—Dr. W. Aldren Turner, C.B.

On Decerebrate Rigidity in Man and the Occurrence of "Tonic Fits."[1]

By S. A. Kinnier Wilson, M.D.

(ABSTRACT.)

[This paper will shortly be published in *Brain*, in full with illustrations.]

For a number of years certain analogies between the results of experimental mesencephalic transection in apes and other mammals and occasional clinical phenomena in man in cases where cortical function is in abeyance have impressed themselves on my attention. These cases are marked by inhibition of cortical activity in the form, usually, of unconsciousness, and by the appearance of limb- and trunk-hypertonus, with frequent exacerbations of this tonus in the guise of "tonic" or postural fits. The cause of the cortical defect has been, as a rule, cerebral hæmorrhage—submeningeal, or intraventricular, or both—and the resulting rigidity closely resembles that produced experimentally by the method above mentioned.

Such characteristic attitudes and postural schemes as are to be seen in mescencephalic transection are reproduced with fidelity in man, and may also appear involuntarily in full consciousness, in certain types of organic and functional nervous disease, while partial or fragmentary types also occur. The hypothesis to be developed is that many of the involuntary postures of a limb, or even part of a limb, as well as many of the transient positions assumed in involuntary movements

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[1] At a meeting of the Section, held May 13, 1920.

such as chorea and athetosis, are in reality simply parts of the complete decerebrate attitude.

The grouping of the cases is as follows:—

(I) In association with loss of consciousness: (*a*) Decerebrate rigidity or attitude and tonic fits combined; (*b*) decerebrate attitude without fits; (*c*) tonic fits without persisting decerebrate attitude.

(II) Decerebrate attitude in conscious life: (*a*) More or less complete; (*b*) incomplete, limited, or partial; (*c*) occurring in the course of certain conscious involuntary movements.

The decerebrate posture, as I have observed it in man, consists of complete extensor rigidity of arms and legs, with extension of trunk and neck, amounting sometimes to actual opisthotonos; the arms are adducted, forearms pronated sometimes to a remarkable degree, hands clenched and wrists flexed, as a rule; legs adducted, feet inverted, toes pointing downwards.

Tonic or postural fits (so called cerebellar fits) are in reality the transient adoption of the decerebrate attitude, and there are changing phases of the tonic attitudes in the case of the fits which are less likely to be observed in the greater or less fixity of decerebrate posture produced by the release of bilaterally-acting and sometimes mutually antagonistic mechanisms. In the case of head retraction, for example, it has two components of rotation and extension, and the same is true of the trunk. Hence, in incomplete cases, there may not be that strict bilaterality of action essential for pure extension, and some head- or trunk-rotation may be noted.

There is an important difference between the unconscious and the conscious cases, seeing that in the latter, unless the corticospinal interruption is absolute, the cortex may and often does interfere in part with the activity of the lower mechanisms. In Sherrington's words "there is a struggle between two conflicting influences."

The phenomena of decerebrate rigidity are obviously release-phenomena. They make their appearance when the cortex is out of action, and they disappear when the lesion is below the neuronic level of the medulla; if we are to accept certain experimental observations, they disappear when the neuraxis is transected below the pontine nuclei.

Thus for their development we limit ourselves to the mesencephalon, cerebellum, and pons. It should, nevertheless, be made clear that there is evidence suggesting that similar attitudes can be obtained when the decerebration is higher, leaving intact the basal ganglia, in particular the optic thalamus.

The experimental work of Sherrington and others, in particular of Graham Brown, points definitely to the conclusion that the postural activities of the decerebrate animal are conditioned by mesencephalic centres and that these are the red nucleus and its immediate vicinity. Stimulation of the neighbourhood of the red nucleus in decerebrate preparations will evoke alternating movements of homolateral flexion and contralateral extension in varying combinations, precisely similar to those which we have seen to occur clinically.

In a personal communication from Professor Sherrington he says:—"The neighbourhood of the iter taken broadly so as to include the red nucleus, small-celled and large-celled parts, certainly has large postural (tonic) functions, and Graham Brown's experiments observed these in detail. Work on decerebrate rigidity has gradually come to suggest that the red nucleus region is of capital importance for the general reflex of 'station' ('standing' in its various forms and subpostures). Tonic (postural) fits would, to judge from laboratory observations, be likely to result from lesions of an acute kind in that region."

Integrity of the local cord proprioceptive arcs from muscle to cord and back again, is usually assumed if the phenomena of decerebrate rigidity are to appear, and section of these renders the limb-musculature flaccid at once. Graham Brown has shown, however, that though these arcs are sectioned the phenomena can, nevertheless, be obtained from the red nucleus region, as ordinarily, provided a sufficient time be allowed to elapse between the section of the dorsal roots and the decerebration.

The cerebellum is not entirely essential to the production of the attitudes. On this point there has been much controversy, but it seems to be an experimental fact that the cerebellum can be removed piecemeal down to the paracerebellar nuclei (Deiters) without disappearance of the decerebrate rigidity from a mesencephalic section. On the other hand, of course, it is known that the cerebellum must play upon these mesencephalic centres, though it be not the originator of the attitudes. After removal of the cerebellum decerebrate rigidity *gradually* disappears.

From the clinico-pathological standpoint, it is suggested that the evidence does not point to the cerebellum as being the seat of origin of the attitudes, though it may well maintain the proper activity of these other paths and centres. The suggestion is that flexor and extensor, pronator and supinator, &c., combinations of the ventral cell-groups of the cord are represented in the nucleus ruber, and that there is a physiological antagonism of the two nuclei in the mesencephalon, which, however, expresses itself at the level of the cord. Uncontrolled activity of the twin nuclei throws the individual into the decerebrate attitude, with predominance or changing activity of flexor or extensor attitude on one or other side, in one or other limb, as the case may be, owing to changing influences in the red nuclei or reaching them.

A hypothesis of this kind will explain, also, the fact of the association of tremor with the red nucleus, and its descending paths, since tremor consists of rhythmical alternating contractions of flexors and extensors, &c., and we have assumed a representation or re-assembling in the red nucleus of the flexor and extensor cord grouping.

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PROCEEDINGS

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VOLUME THE THIRTEENTH
SESSION 1919-20

No. 11

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1920

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Section of Obstetrics and Gynæcology.

President—Mr. J. D. Malcolm, F.R.C.S.Ed.

Case of Indirect Traumatic Rupture of the Uterus during Pregnancy.[1]

By Gordon Ley, F.R.C.S.

I consider the following case worthy of reporting, first, on account of the extreme rarity of indirect traumatic rupture of the uterus during pregnancy, of which condition I can only find nine previously recorded cases, and secondly on account of the peculiar clinical history of this particular case. For the early notes, I am indebted to Dr. R. Thorne Thorne, of Woking, to whom my best thanks are due.

Mrs. N., aged 22, a primigravida. The last day of her last monthly period was February 28, 1918, so that at the day of the accident, July 29, she was twenty-one weeks pregnant Her previous health, both before and during the pregnancy, had been excellent. On July 29, at 11 a.m., she was standing on Woking platform when she suddenly felt faint, and staggering forwards fell off the platform on to the metals. She fell doubled up in a crouching attitude. She was picked up, and, after regaining consciousness, was carried to a taxicab and taken home, where, having walked upstairs, she was put to bed and her doctor was sent for. He saw her at 12 noon. The temperature was 97·6° F., the pulse 76. She was pale and was suffering from nervous shock. She complained of pain in the hypogastrium; the lower lip was cut and the right shoulder bruised. An abdominal examination revealed an apparently normal twenty weeks' pregnancy. There was no rigidity. A catheter was passed, and clear urine was drawn off. She was reassured and became more composed.

At 6 p.m. Dr. Thorne Thorne again saw the patient. She had since

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[1] At a meeting of the Section, held October 9, 1919.

5 p.m. noticed a slight watery show; the hypogastric pain had increased, and was of a stretching character. Temperature 97·6° F., pulse 110. There was no alteration in the physical signs. She was given 5 minims of tinct. opii. From this time there was no further vaginal loss. At 9 p.m. she was again seen by her doctor. He then noticed that she had become extremely pale. The pulse was 120 and soft; the pain was still severe. The abdominal physical signs had not altered. A diagnosis of concealed accidental hæmorrhage was made, and he endeavoured to rupture the membranes by passing a sound. The sound appeared to come in contact with the fœtal head. The attempt to rupture the membranes was, however, unsuccessful. The patient found the left lateral position was more comfortable, and was accordingly left in this attitude. Dr. Thorne Thorne telephoned to me at 9.15 p.m. At 9.30 p.m. he found the patient much worse: pulse 140, feeble and soft.

I saw Mrs. N. about 1.30 a.m. on July 30. She was lying on her left side and complained of continual agonizing abdominal pain, made much worse by moving her. She was extremely anæmic. The pulse was feeble and uncountable; the respirations rapid. There was air-hunger and restlessness. The abdomen was distended, rigid and extremely tender all over. In the left lumbar region there was an ill-defined swelling about the size of an ostrich's egg. The uterus could not be palpated. On vaginal examination the cervix was closed, and Douglas's pouch bulged slightly. I came to the conclusion that there was hæmoperitoneum, probably from rupture of the uterus; the mass in the left lumbar region being the unruptured ovum; but I suggested as a second diagnosis a ruptured left kidney with perinephric hæmatoma perforated to the peritoneum.

I opened the abdomen fifteen minutes later. There was hæmoperitoneum to the amount of three pints or more. The ovum, unruptured, was lying free in the left kidney pouch. The uterus was contracted and showed on its anterior wall a laceration 3 in. long, running from the right cornu obliquely downwards and to the left; judging by the vascularity of the wall the rupture was through the placental site. The uterus was sutured with catgut in two layers and the abdomen closed. The placenta and membranes appeared normal. The urine contained no albumin. She was given a rectal saline, rallied well, and except for some pyrexia from the first to the tenth day, due largely, if not entirely, to the absorption of blood-clot, made an uninterrupted recovery.

I take it that in this case the sequence of events was as follows: (1) trauma; (2) incomplete rupture of the uterus involving the peritoneum and the outer muscular coats; (3) complete rupture of the uterus with gradually increasing hæmoperitoneum; (4) extrusion of the ovum into the peritoneal cavity at or about 9 p.m. on July 29.

Below I give the references of the nine other recorded cases of indirect traumatic rupture during pregnancy:—

DE LEE, J. B. *Amer. Journ. Obstet.*, 1904, l, p. 814.
LEOPOLD, G. *Arch. f. Gyn.*, 1896, lii, p. 376.
TISSIER and DAVID. *Bull. de la Soc. d'obstét. de Par.*, 1906, ix, p. 338.
NEUGEBAUER, Jun. *Centralbl. f. Gyn.*, 1890, xiv, Beilage, p. 88.
JELLINGHAUS. *Arch. f. Gyn.*, 1897, liv, p. 103.
HINTERSTOISSER, H. *Monatssch. f. Geb. u. Gyn.*, 1911, xxxiv, p. 652.
VON REUSING. *Centralbl. f. Gyn.*, 1895, xix, p. 41.
PLENIOR. *Centralbl. f. Gyn.*, 1885, ix, p. 47.
SLAWIANSKY. *Centralbl. f. Gyn.*, 1886, x, p. 22.

The Pre-cancerous Uterus.

By F. J. MCCANN, F.R.C.S.

IT is no exaggeration to state that a large proportion of the scientific intellects of the civilized world has been in recent years directed towards a solution of the cancer problem. Are we any nearer the truth? Do we know the cause? Can we produce a large percentage of cures? These and many other questions suggest themselves to those interested in the subject. From a study of recent researches, one arises unconvinced, wondering whether the trend of the modern scientific investigation of cancer is really along productive lines. Indeed there is a tendency to relegate the clinician to the background and to look towards the laboratory worker for a solution of the problem. This is unfortunate, for both are mutually helpful, mutually indispensable.

I have always thought that no real progress would be made until the clinician can seek out and identify the pre-cancerous changes met with in the different organs and tissues where cancer is prone to originate. Surgery has almost reached its limit regarding the extent of the operative procedures devised and practised for the cure of cancer. Yet after all the early examples of the disease, *ceteris paribus*, yield the best results, whilst operations for advanced disease are generally disappointing.

To operate when cancer is easily recognizable is not enough; we must, if we are to progress, anticipate its development and for this purpose, be able to recognize pre-cancerous changes as definite clinical entities.

The subject was brought forcibly to my mind by the occurrence of two examples in which the after-history of each has an important bearing on this subject.

The first example was that of a woman during menstrual life, aged 38, who underwent the operation of curetting for what is termed "fungous endometritis." The scrapings were examined by a high pathological authority and pronounced to be innocent. Here let me say that examination of scrapings is often most unsatisfactory, and indeed it is not quite fair to the pathologist to expect him always to give a definite opinion. It was my own opinion at the time that there was a suspicious multiplication of cells within certain gland tubules, but there was no evidence of penetration beyond the basement membrane. Five years later I was summoned to see this woman and found on examination that she had advanced uterine cancer, from which she ultimately died.

The second example, even more apposite, was that of a single woman, aged 54, from whom I removed a polypus about the size of a hazel nut which grew from the upper end of the cervical canal. This proved on microscopical examination to be innocent. Eight years later she died from uterine cancer with secondary hepatic deposits.

In both instances the cancer was too far advanced for any radical operation when these women were seen again by me. Had I not had the opportunity of seeing these women again I might have been under the impression that the curetting and the removal of the polypus had effected a complete cure.

This only serves to emphasize the great value of the study of after-results. Who know the ultimate fate of all the women they have curetted or from whom they have removed polypi? Indeed a department for the study of after-results will have to be established in connexion with every properly equipped hospital in this country. If this is not done we shall be left behind in the march of scientific progress.

What a wealth of information could be acquired by such a department in relation to this question of pre-cancerous conditions! The mamma and the uterus are the commonest sites for cancer in the human female. In both situations there must be pre-cancerous changes. It is the business of the clinician to aid in their identification, and to this end the study of after-results is of prime importance. Women are about twice as frequently afflicted with cancer as men, and about a fourth of all cancers are uterine. Parity appears to be a most important predisposing cause of cancer of the cervix uteri, for women so affected have usually borne more than the average number of children. From this it is reasonable to conclude that cervical injuries, the result of childbirth, are predisposing factors. I have satisfied myself that gonorrhœal endocervicitis if persistent is also a predisposing factor.

The age-incidence of uterine cancer is both interesting and important. From a study of statistics[1] 46 may be taken as that for cancer of the cervix, and 53 for cancer of the uterine body.

Several years elapse before a hot clay pipe produces cancer of the lip, or before the traumatism of childbirth produces cervical cancer. It is during those years that it is necessary to be on the look out for pre-cancerous changes. Let us see what these changes are in the cervix uteri and in the corpus uteri: (*a*) During menstrual life; (*b*) after the menopause.

(1) *Cervix Uteri.*

In the cervix uteri the changes which should give rise to suspicion of cancer may be grouped as follows: (*a*) Erosions; (*b*) fissures; (*c*) chronic endocervicitis; (*d*) chronic hypertrophy.

(*a*) *Cervical Erosions.*—Erosions have in the past been termed "ulcerations," and upon this faulty pathology faulty methods of treatment have followed. An erosion is a new growth composed of glandular tubules and interglandular tissue, which may under certain circumstances undergo true ulceration. Now in the absence of traumatism a definite ulcer in the cervix uteri is either tuberculous, syphilitic or malignant, and as the two former are relatively rare it should be assumed to be malignant until the contrary is proved by microscopical examination. An erosion being a new growth—adenoma—it should be treated as other new growths are treated—viz., by excision and not by repeated application of irritants in the shape of caustics and similar applications. Further it has been proved in many instances that cancer originates in an erosion, and if erosions are carefully examined under the microscope metaplasia of the epithelium is frequently observed, which is a pre-cancerous change. It should therefore be a rule of practice that erosions, if sufficiently marked to call for treatment, should be treated by excision.

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[1] "Cancer of the Womb," p. 13.

(*b*) *Fissures.*—The rôle of cervical lacerations — fissures — in the ætiology of cancer has been debated for many years. Some appear to hold the opinion that cancer commonly arises in a cervical laceration. The truth is that it may do so exceptionally, as is the case in scar tissue in other parts of the body. It is not however the laceration *per se* but the general condition of the cervical tissues in association with it, which predisposes to cancerous development. It is not only the single tear but the innumerable smaller ones causing dislocation of epithelium and associated with chronic inflammatory changes which predispose to cancer. Statistics have been produced favouring the view that stitching the lacerated cervix after labour has an influence in diminishing the incidence of cervical cancer. Whether one accepts such statistics or not it is undoubtedly good practice to stitch the cervix after labour if it is unduly torn. If this has not been done, it should certainly be done at a later date, as soon as it is discovered. No other treatment is so effectual in retarding the chronic inflammatory changes which develop and which predispose to cancerous formation.

(*c*) *Chronic Endocervicitis.*—A woman approaching the cancer age from whose cervical canal a chronic discharge continues to flow incurs the risk of subsequent development of cancer within the cervical canal. Moreover, the glandular involvement often extends so deeply into the tissues of the cervix that no local application or curetting reaches the full extent of the diseased area. To eradicate the disease and to prevent the development of cancer a supravaginal amputation of the cervix should be performed. If in addition to the chronic endocervicitis the uterine body is enlarged and the menstrual flow increased or, still more, if between the menstruation there is irregular blood loss, the uterus should be removed. Pan-hysterectomy, with conservation of the ovaries if otherwise healthy, is the operation which should be adopted.

(*d*) *Chronic Hypertrophy.*—Reference has already been made to chronic inflammatory thickening of the cervical tissues in association with lacerations or fissures. Should such hypertrophic thickening be marked, especially in women approaching the cancerous age, associated or not with cervical lacerations, the cervix should be removed by supravaginal amputation. If this operation is done in a skilful manner with due regard to asepsis, there should not be any subsequent contraction of the outlet to the uterine canal.

(2) *Corpus Uteri.*

(a) Fungous Endometritis.—"Fungous endometritis," which is in reality a new growth, adenomatous in character, bears a relationship to cancer of the corpus uteri similar to that existing between a cervical erosion and cancer of the cervix uteri. In other words, it is a pre-cancerous condition. Examples are on record in which, after repeated curettings, the scrapings at first innocent in character were later found to be malignant. If exploration of the uterine cavity shows that there is a considerable amount of new growth in the endometrium, and if the woman is nearing the menopause, the uterus should be removed. Curetting followed by the application of some irritant fluid which is so frequently advised and practised should be abandoned in favour of more thorough methods. Here again the line of demarcation between an adenoma and an adeno-carcinoma of the corpus uteri is exceedingly fine, and it is only by anticipating subsequent developments that real progress will be made.

(b) Chronic Metritis.—A woman over 40 years of age possessing a large painful uterus, bleeding profusely during menstruation and pouring out discharge in the interval, should have it removed. Such women are frequently treated by curetting, caustics and other irritants without benefit, and many of them subsequently become the victims of malignant disease.

(c) *Intra-uterine Polypi.*—Experience teaches that intra-uterine polypi are not only associated with important changes in the endometrium, but are danger signals giving a warning of future mischief. Reference is here made more particularly to glandular or adenomatous polypi. These polypi grow at the junction of the cervical and uterine mucosa and at the tubal orifices. They also grow from the cervical canal and the margin of the external os uteri. When examined under the microscope metaplasia of the epithelium, which is a pre-cancerous change, is frequently observed. It is easy during the operation of curetting to destroy such polypi, and then they are evacuated with the scrapings of the endometrium and thus escape recognition. If adenomatous polypi are found in the uterine cavity the uterus should be removed. "Fibroid polypi" should when removed be examined microscopically to make sure that the disease is not sarcomatous. Fibroid polypi (myomata) do not portend evil like glandular polypi and their removal usually gives satisfactory results. Glandular polypi growing from the cervical canal, if provided with a long thin pedicle, can be successfully treated by the tying or crushing of the pedicle. If, however, the polypi are sessile amputation of the cervix should be performed.

(*d*) *After the Menopause.*—The therapeutic problem after the cessation of menstruation does not present the same amount of difficulty. The normal course of events is for the uterus to diminish in size as age advances and to remain quiescent, not giving rise to any local disturbance. The normal post-climacteric uterus is a decadent organ showing no sign of pathological activity. Any departure from the normal is a danger signal calling for prompt treatment. If examples of uterine prolapse and acute infections be excluded it may be stated "that post-climacteric uteri showing pathological activity, evidenced clinically by bleeding, discharge or increased size should be removed." To delay by the employment of palliative methods is to court disaster, for valuable time is lost and the chances of successful radical treatment are jeopardized.

The disease termed "senile endometritis" is a pre-cancerous condition, and is indeed frequently associated with cancer of the corpus uteri. It has been my practice for many years to treat this disease by removal of the uterus, and I have been surprised to find that in nearly all the uteri removed there was new growth in the endometrium. In this relation let me recall the words of the late Dr. Matthews Duncan[1] when writing of this disease. He says, "I know of no way of diagnosing except retrospectively. If it is cured it is an endometritis. Retrospective diagnosis is not of much value to your patient. It is prognosis that you desiderate for her." Thus it must be the work of the future to anticipate the development of cancer by defining those conditions which experience teaches are followed at a later date by cancerous growth, and by treating them by radical methods. Real progress will result from work along these lines, much suffering will be mitigated and many lives saved.

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[1] "Clinical Lectures on the Diseases of Women," p. 61.

DISCUSSION.

Dr. LAPTHORN SMITH: I feel very strongly on the question of earlier operations for the removal of cancer, and Dr. McCann's paper is valuable and timely. He has tried to show that there are certain conditions of the uterus which render it more liable to cancer than others, and for want of a better term he calls these uteri pre-cancerous. If his paper will lead to the earlier removal of a part or the whole of the affected or infected organ, as I prefer to call it, at a much earlier stage than we are now doing, so that the time will come when there will be neither mortality nor morbidity after the operation, it will prove of great value. The Registrar-General's Report for 1916 says there were 40,630 deaths from cancer, of which 22,843 occurred in females. No doubt Dr. McCann had this great army of women marching to their death in his mind when he wrote his paper. Can most of them be saved? I believe they can. The numbers for 1916 were the largest ever recorded up to that year, and I have no doubt that the numbers have reached a much higher figure by now, probably 50,000 men and women. We have made great progress in stamping out many other diseases, but in this terrible one we seem to have made little if any. In deaths it is only surpassed by tuberculosis with its 54,000. Nearly everyone admits that it begins locally: in that case it must be due to a micro-organism which has found a suitable feeding ground and multiplies rapidly. That same cancer cell would not grow on the healthy uterus of a young woman. Emmett, one of the fathers of gynæcology, who had an enormous experience, once told me that he had never known a cancer of the cervix to have occurred in a woman who had not had a laceration. Only once had he thought he had seen one, but on inquiry he found that this woman had had her cervix dilated when she was a girl, for dysmenorrhœa, and it was accidentally torn. I am convinced that if it were the general practice to see that every lacerated cervix was repaired soon after the injury, which generally takes place at the first confinement, instead of leaving it to heal by granulation, which means scar tissue, there would be an immediate and very considerable saving of those 23,000 lives which are annually lost. Many of these lacerations are taken for erosions and treated with nitrate of silver, when all the time what appears to be an erosion is the delicate ciliated epithelium lining the cervical canal which is everted and exposed to friction. The result of this treatment is the formation of large masses of scar tissue and hypertrophy of the vaginal portion of the cervix. When the woman reaches the menopause we might call such a cervix a pre-cancerous one, not because there is any cancer in it at the time, but because it presents all the conditions favourable for the growth of cancer if it is infected. I have seen many such neglected lacerated cervices become cancerous, and I have performed the simple operation, amputation of the diseased cervix in order to save the patient from the dread disease. I do not say that the woman would surely have had cancer, but I do say that such a uterus is a candidate for cancer. I have seen a great many similar cases for the first time when the woman was having irregular hæmorrhages several years after the menopause, and when the pathologist reported on a section that it was clearly cancerous. Some of these women made good recoveries and remained well for many years after a high amputation of the cervix, while others were so far advanced that nothing short of total hysterectomy sufficed. Whether cancer develops in the scar tissue of a clay-pipe smoker's lip, or whether it develops in the scar tissue of an ulcerated stomach, or in the breast or in the uterus, it is a matter of life and death to the patient to have it removed at the earliest possible moment. By prompt action a large proportion of those 23,000 women would have been saved. It was largely, though not entirely, due to a policy of waiting and hesitation that such a large number died. By waiting until there is pain and a foul discharge we condemn the patient to a loathsome death.

Dr. HERBERT SPENCER: I am in complete disagreement with the author on two of the chief recommendations of his paper—viz., the excision of erosions on the plea that they give rise to cancer, and hysterectomy in cases of mucous polypi of the body. Erosions are exceedingly common in virgins and recover perfectly under simple local applications. Cancer of the cervix is exceedingly rare in virgins and even in nulliparæ; I do not think I have seen half-a-dozen cases. I have watched patients, from whom I have removed corporeal mucous polypi, for many years, without seeing the development of cancer in a single case. I regard the author's recommendations on these two points as quite unjustified by any evidence he gives.

Dr. FAIRBAIRN: Dr. McCann's paper might be described as suggestive but unconvincing, for he has brought forward no evidence as to what a pre-cancerous uterus might be. He attempted to make progress by jumping to conclusions. Polypi, adenomatous overgrowth of the endometrium and erosion of the cervix are all very common conditions, and must often precede cancer, but not an iota of proof has been brought forward this evening that they are a true pre-cancerous stage. They often accompany cancer, as they do fibroids, and polypi especially are frequently taken as the cause of the symptoms and early cancer overlooked. But that is an argument for more thorough examination, and not for the wholesale removal, of organs on the chance that cancer may develop later. Dr. Lapthorn Smith was even less convincing than Dr. McCann, and his remarks were not those which should be made in a scientific society. What knowledge has he to enable him to say that he has saved women from cancer by removing an unhealthy cervix or uterus if there was no microscopical evidence of cancer? His mere *ipse dixit* carries no weight unless he can give some better reason for his procedure. I would draw attention to one side of the cancer problem which has been entirely overlooked by those speakers who so readily swallow the pre-cancerous state, and that is the part which may be played by general bodily nutrition and resistance. Like many other invasions of healthy tissues by a morbid process, the cancer problem may not be entirely a question of local disease.

Dr. HERBERT WILLIAMSON: The recognition of pre-cancerous conditions of the uterus is a matter of the utmost importance, and when it can be established definitely that a lesion, trivial in itself, is a precursor of carcinoma it will be generally agreed that a radical operation should be performed without delay. Dr. McCann has included in his list of pre-cancerous conditions a variety of lesions which are very frequently met with, and it behoves us to examine carefully the evidence upon which his statements are based. When we look for the evidence we find none, which as a scientific society, we can accept. The drastic treatment he advises is based on a series of ex cathedra statements which he does not attempt to prove. The argument with which he introduces his paper is fallacious. Because he removed an adenomatous polypus from a patient, and eight years later she developed cancer, he argues, if I understand him rightly, that an adenomatous polypus is a pre-cancerous condition. If he wishes us to accept that deduction he must prove to us: (1) That women who grow adenomatous polypi are more prone to cancer than women who do not; and (2) that an appreciable proportion of the women from whom adenomatous polypi are removed subsequently develop cancer. He has produced no evidence to prove either of these things. Again he argues that because he curetted a woman for the condition commonly called fungous endometritis, and six years later she developed cancer, fungous endometritis is to be regarded as a pre-cancerous condition. I wonder how many patients those present to-night have curetted for this and similar conditions; what a holocaust would have resulted had we thought it necessary to remove all these uteri! Thousands of unnecessary and useless mutilating operations would have been performed and some lives would have been sacrificed, for no man in a long series of abdominal operations has yet shown a mortality of 0 per cent. Amongst pre-cancerous conditions Dr. McCann places cervical erosions: these are common in nulliparæ—possibly almost as common as in those who have borne children—but carcinoma of the cervix is seldom seen in nulliparæ. In my opinion it is quite unnecessary to excise these erosions, the majority of them can be destroyed by the application of a saturated solution of picric acid in alcohol Dr. McCann's list includes chronic metritis, metaplasia of epithelium, gonorrhœa and cervical lacerations, and without the production of evidence we are asked to accept these conditions as pre-cancerous. We shall all agree that child-bearing is the great pre-disposing cause of carcinoma of the cervix, because that statement rests upon established proof, but it is by no means certain that the lacerations are the exciting cause, indeed the evidence goes to show that chronic infection and continued irritation are probably more potent factors. If we are to excise every cervix which has been torn in child-birth, the possession of a cervix uteri will soon become an extreme rarity amongst matrons. A deeply lacerated cervix with ectropion and glandular overgrowth, the site of a chronic infection, should, in my opinion, be repaired in young women, and amputated in women approaching the menopause, but I know of no facts which justify us in regarding a simple cervical laceration as a cause of cancer, or in advising the drastic treatment Dr. McCann suggests.

Dr. R. A. GIBBONS: I gather that the gist of this paper is that if a uterus is not physiologically sound it is better to remove it lest carcinoma should develop. I consider that this is a very sweeping recommendation, and it cannot pass unchallenged. I should have thought that in bringing such a paper as this before a scientific society, Dr. McCann with his usual thoroughness would have brought forward carefully prepared statistics pointing out in how many cases of certain operations carcinoma followed, and what time elapsed between the date of operation and the time of the symptoms of malignancy. I have an impression that I have seen carcinoma following on lacerations of the cervix which have not been operated on, but I should certainly not make any dogmatic assertion concerning this without collecting statistics. We must have done hundreds of cases of curetting for chronic endometritis, polypus, &c., in hospital and private practice, but because the uterus for the time being is pathological, it seems an extraordinary statement that it should be removed lest carcinoma develop, for we know that in the great majority of cases treatment puts it into a healthy state again. Before making any statement concerning a pre-cancerous state, I am of opinion that we ought to have abundance of statistics showing in how many such cases carcinoma develops, and the time elapsing before signs of malignancy occur. I am sure that if those who have had much experience could say what are the signs of a pre-cancerous state, all would agree that the uterus should be removed as soon as those signs occur. I feel that I have formed the same impression concerning this paper as others who have spoken, and unless we are wrong in our interpretation of it, I consider that in such a society as this it certainly calls for criticism.

Dr. J. ELLISON: I have had the privilege of working with Dr. McCann at the Samaritan Hospital during the last year. I believe that his paper is a plea for the earlier removal of the uterus in cases in which, at or after the climacteric, a diagnosis of polypus, endometritis or chronic metritis is made, for he considers that such conditions appearing at the time are probably pre-cancerous. It is generally accepted that from the climacteric onwards the uterus should, under normal conditions, atrophy, and be both functionally and physiologically inactive. I have heard Dr. McCann state that any activity of the uterus after the climacteric as shown either by increasing size, discharge or hæmorrhage ought to be considered as a danger signal on the road to cancer, and should be treated not by palliative measures, but by removal of that organ. Any case presenting such symptoms, which may be submitted to a diagnostic curettage, may be the subject of a misleading pathological report. For in the case of an endometritis it may be that all the sections examined are quite innocent in character, whereas there might have been some small suspicious portion, which though removed, was not examined. Again the uterine angles are common sites at which corporeal carcinoma commences, and these are admittedly difficult to curette thoroughly. Thus the suspicious area may even have been left behind. In either case the symptoms may for a time be relieved while the carcinoma itself is progressing. In course of time the symptoms reappear, but reliance is placed on the previous pathological report, and the symptoms may be temporized with till perhaps too late. Advances in the surgery of carcinoma of the breast all tend towards an advocacy of an earlier and more complete removal of the organ, even in the supposed pre-cancerous stage, and results I believe have fully justified the procedure. Granted that from the climacteric onwards the uterus is a decadent organ, should we not be justified in removing it immediately these danger signals arise, when in the hands of competent operators the mortality rate is under 2 per cent., rather than run the risk of allowing the disease to become inoperable, either through the modesty of the patient or the misled judgement of her attendant?

THE PRESIDENT: I regret that Dr. McCann is not present to reply to the debate. The question he has raised is one of the greatest importance, and there can be no disagreement as to the necessity for removal of a cancer of the uterus when recognized, or for the prevention of the development of such a growth if possible. But the existence of a definite recognizable pre-cancerous stage is not so obvious. I understand that Dr. McCann advises the immediate treatment, by removal, if necessary, of all conditions which may constitute an irritation of uterine tissue. The debate has clearly shown that there are differences of opinion amongst the members of the Section concerning the importance as causes of cancer of some of the conditions which Dr. McCann regards as pre-cancerous. The contention seems to be that removal of all causes of irritation will prevent a development of cancer. Dr. McCann quoted Matthews Duncan to the effect that a retrospective diagnosis is the only one possible in cases of senile endometritis. In such cases when there is discharge and hæmorrhage after menstruation has ceased, without any apparent cause, hysterectomy seems to me the proper treatment, because the risks of waiting in such cases at this time are so great, but I am not persuaded that in younger women such conditions as fissure or chronic hypertrophy of the cervix require active treatment because these conditions may be followed by a development of cancer.

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Section of Obstetrics and Gynæcology.

President—Mr. J. D. MALCOLM, F.R.C.S.Ed.

The Rôle of the Cinematograph in the Teaching of Obstetrics.[1]

By G. DRUMMOND ROBINSON, M.D.

AT the present moment great interest is being taken in an effort to improve the teaching of obstetrics in London. Our Section has quite recently issued a Report on this most important branch of medical education which it is sincerely hoped may do much to improve a state of things which, to put it very mildly, is highly undesirable. Any method which may assist the student to get a better grasp of this subject is valuable.

We all know how helpful it is in any branch of study to be able actually to see what happens—if that be possible—as well as to hear and to read about it. By means of the cinematograph a process or an operation can actually be shown to as large an audience as may be desired and as often as may be necessary. By means of photographs of actual labours all that it is possible to show an onlooker of normal or abnormal labour may be demonstrated in the class-room, under the most convenient conditions and by the most experienced teachers, as vividly as at the bedside. This would to some extent remedy the defect in our present system of teaching, which has been pointed out in our recent Report: namely that instruction on the actual labour case is at present practically left entirely in the hands of junior teachers. By means of animated diagrams and drawings, the internal processes of development and of disease, as well as the manipulations and movements that take place unobserved during most obstetric operations, can be demonstrated to the eye.

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[1] At a meeting of the Section, held November 6, 1919.

I will now illustrate what I have said by means of the following actual films.

ANIMATED DRAWING.

First Stage of Labour.

Note 1.—Contractions of uterus (upper segment).
,, 2.—Dilatation of (*a*) internal os uteri; (*b*) cervical canal; (*c*) external os uteri.
,, 3.—Bulging of membranes.
,, 4.—Rupture of membranes and escape of liquor amnii.

ANIMATED DRAWING.

Second Stage of Labour—Vertex Presentation.

Note 1.—Descent of the head.
,, 2.—Rotation of the head.
,, 3.—Extension of the head.
,, 4.—Restitution of the head.
,, 5.—Moulding of the head.
,, 6.—Distension of perineum.

ANIMATED DRAWING.

Second Stage of Labour—Birth of Body of Fœtus.

Note 1.—Contraction and retraction of upper uterine segment.
,, 2.—Rotation forwards of the anterior shoulder.
,, 3.—Lateral flexion of the body.

NATURAL SUBJECT.

Second Stage of Labour—Vertex Presentation:

Note 1.—Bulging of the perineum and dilatation of the anus.
,, 2.—Birth of fœtal head.
,, 3.—Wiping eyes of fœtus.
,, 4.—Birth of body of fœtus.
,, 5.—Ligature and division of the umbilical cord (after all pulsation has ceased).

ANIMATED DRAWING.

Second Stage of Labour—Breech Presentation.

Note 1.—Descent of breech.
,, 2.—Rotation forwards of anterior buttock.
,, 3.—Lateral flexion of body of fœtus.
,, 4.—Birth of after-coming head assisted by (*a*) traction from below by jaw and shoulder grip; (*b*) pressure from above.

NATURAL SUBJECT.

Second Stage of Labour—Breech Presentation.

Note 1.—Distension of perineum and dilatation of anus.
,, 2.—Birth of breech.
,, 3.—Delivery of after-coming head by means of (*a*) traction by jaw and shoulder grip; (*b*) pressure from above.
,, 4.—Asphyxia neonatorum.

ANIMATED DRAWING.

Third Stage of Labour—Birth of the Placenta.

Note 1.—Retro-placental hæmorrhage.
,, 2.—Contraction of the uterus.
,, 3.—Expulsion of the placenta.

NATURAL SUBJECT.

Third Stage of Labour—Birth of the Placenta.

Note 1.—Expression of the placenta from the vagina.
,, 2.—Twisting the membranes into a cord.
,, 3.—Examination of placenta and membranes.

NATURAL SUBJECT.

Artificial Respiration.

Note 1.—Silvester's method.
,, 2.—Schultze's method.

For the present occasion it seemed desirable that the process of labour should be shown completely by the photographs as it actually occurs. For teaching purposes no doubt the photographs would be arranged according to the stages of labour; for instance, all the photographs dealing with the first stage of labour would probably be shown together. Any arrangement of photographs could be made to meet the views of the teacher without the slightest difficulty.

I have only concerned myself with normal labour, but it is obvious that such subjects as the development of the Graafian follicle and corpus luteum, the passage of the ovum along the Fallopian tube, the meeting of the ovum with the spermatozoon, the development (microscopic and macroscopic) of the decidua, the development of the placenta, the changes in size and shape in the gravid uterus, the involution of the uterus, the action of the uterine segments, ante-partum hæmorrhage, obstetrical operations such as version, forceps, craniotomy, &c., could all be admirably shown. Such operations as forceps delivery, Cæsarean section and others could also be demonstrated by means of photographs of the actual subject.

The drawback to this method of teaching is the initial expense of the production of the film. The photographing of the actual subject is not unduly expensive, but in the production of animated drawings the result depends largely upon the number and character of the drawings, and as a large number of drawings is necessary this can never be cheap. However, if this method of teaching were largely adopted and a considerable number of copies were disposed of the expense would be relatively insignificant.

In conclusion I feel convinced that some persons of means might confer a great benefit on medical education and on humanity in general by investing a sum of money in the production of films that would illustrate the whole subject of obstetrics completely.

DISCUSSION.

Sir FRANCIS CHAMPNEYS: Dr. Robinson's films, which must have cost him much thought and expense, are admirable. I believe that they will prove a valuable aid to teaching. There are few details in which they may be improved, I think, quite easily.

Dr. AMAND ROUTH: Dr. Drummond Robinson's film is likely to be of great use in the effort to improve the teaching of obstetrics. This is more particularly the case with the films illustrating actual labour, and if the "jerks," which rather spoil the normally smooth sequence of events in the animated diagrams can be obviated, these representations also will be of great value.

Dr. M. CORNER: The lecturer deserves our congratulations on his subject, but I think the film is too precipitate—almost indicating a "precipitate labour." In my work at the East End Lying-in Home, in East London, I have to teach a number of midwifery pupils, and it is in this stage of labour, the third, that I particularly emphasize certain signs, so that each pupil should wait (unless an emergency arises) before expressing the afterbirth. These signs are: (*a*) Rising of fundus uteri in the abdomen, (*b*) "wobbling" or feeling of loosening, (*c*) extension of umbilical cord from the vulva. I do not offer further criticism, for Dr. Robinson has stated that it is the first film of the kind. I think it would be dangerous for such a film to go broadcast without more detail from a physical and aseptic point of view, but further experience will, I have no doubt, clear up these dubious points.

As it is at present I do not consider the film an ideal method of teaching midwifery.

Mr. McADAM ECCLES: Dr. Drummond Robinson's "natural films" are excellent and the right type of film for practical teaching in obstetrics. But the number of such films now existing is small. The authorities of medical schools hesitate to instal an expensive cinematograph apparatus unless there is a definite possibility that it will be of real use. In only one of the English medical schools does such an apparatus exist, and there it is the private property of the lecturer. What is needed is a full catalogue of medical films now obtainable, opportunities for making a much larger number of such films, and a central bureau from which they can be hired. I believe that there is a great future for education by means of films, provided that really good ones be produced and that efficient apparatus be established, and operators forthcoming in the leading medical schools.

Dr. EDEN: It is rather unfair to criticize the details of Dr. Drummond Robinson's pictures. We all know how difficult it is to get an artist to produce exactly correct diagrams, and it must be recollected also that this is pioneer work, and that the author has had no previous efforts to guide him. There is a great difference in value between the "animated drawings" and the cinema pictures of the natural subject. The latter are most interesting and will prove of enormous value, especially in tutorial instruction. The process can be applied very widely to operative procedures, both gynæcological and obstetrical, but it can never take the place in the smallest degree of practical instruction at the bedside and in the labour ward. The "animated drawings" do not appear to me to be of much educational value; they have an air of unreality which robs them of any real superiority over the ordinary diagram.

Dr. HERBERT SPENCER: Dr. Robinson's demonstration has been excellent. The drawings, however, are not above criticism, and I suggest that a series of photographs of an actual fœtus placed in successive positions in a model will give a more life-like film.

Nine Cases of Inversion of the Uterus.[1]

By Herbert R. Spencer, M.D.

SUMMARY OF CASES.

Case I.—Inversion at unassisted labour; putrid placenta and membranes, adherent, removed on admission (nineteenth day). Under continuous irrigation with boric acid solution spontaneous reduction on forty-first day. Good recovery. Highest temperature 102·6° F., fell to 100° F. fourth day; normal nearly every day afterwards. Discharged thirty-first day,

Case II.—Inversion at unassisted labour; extreme anæmia. Seen on fifth day, uterine tissue too soft to permit pressure safely. Continuous irrigation. Aveling's repositor on eleventh day; reduction within eight and three-quarter hours. Good recovery. Temperature on admission 101° F., second day 101·4° F., third day 100·8° F., afterwards below 100° F. except on one occasion (100·6° F.). Discharged twenty-ninth day.

Case III.--Inversion at labour, attended by medical assistant; condition unrecognized. Aveling's repositor on twenty-second day; reduction within five and half hours. Normal afebrile recovery. Discharged fourteenth day.

Case IV.—Inversion at labour, attended by medical assistant; condition unrecognized; extreme anæmia. Twenty-two months later advised at a hospital to have uterus removed: refused. Subsequently another doctor tried cup and straight-stem pessary for thirty-six hours, and again for fifteen hours, without benefit. Aveling's repositor applied two years and eight months after labour; reduction within forty-four hours. Good recovery. Highest temperature 102·4° F., gradually fell to normal. Discharged eighteenth day.

Case V.—Inversion at labour, attended by doctor; condition unrecognized. Aveling's repositor nine months later; reduction in forty-seven and half hours. Good recovery. Discharged eighteenth day.

Case VI.—Inversion by gangrenous sessile submucous myoma. Temperature 99·6° F. Excision of myoma; immediate reduction with fingers; iodoform gauze tampon applied for twelve hours. Good recovery. Temperature only twice reached 99° F. Discharged twenty-first day.

Case VII.—Inversion by gangrenous sessile submucous myoma. Bisection and enucleation of myoma; immediate reduction with fingers; iodoform gauze tampon for a few hours. Good recovery. Temperature only once rose to 100° F. after the operation. Discharged thirteenth day.

Case VIII.—Inversion by fundal intramural cystic myoma of size of pigeon's egg. Inverted organ resembled ordinary inversion except that it was twice as big as normal uterus; no line of demarcation between and no hardness where tumour was. Incision into fundus; enucleation of tumour; immediate reduction with fingers; iodoform gauze tampon for twelve hours. Good recovery. Highest temperature 99·6° F. Discharged fourteenth day.

Case IX.—Inversion by spindle-cell sarcoma of body. Uterus bisected and removed *per vaginam*. Recovery. Recurrence after four months. Local removal with cautery. Recurrence.

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[1] At a meeting of the Section, held November 6, 1919.

Case I.—Mrs. L. F., aged 39 (twelve children, no abortion), was admitted to University College Hospital on September 20, 1890. She had been married twenty-one years and had had twelve children, the last being born on September 1, 1890, nineteen days before admission. All her labours were easy and rapid. The last child was born before the arrival of the midwife who attended her. The labour only lasted three-quarters of an hour. The patient was very ill from post-partum hæmorrhage immediately after the child was born and had had abdominal pains ever since. The discharge became exceedingly offensive at the end of the first week. A lump was noticed to be hanging from the passage, and it was necessary to use the catheter for retention of urine. On admission the patient was pale and sallow, but had a good pulse, beating 100, and a temperature of 101° F. She complained of no pain. The abdomen was a little full, slightly tender: deep down beneath the promontory of the sacrum a swelling could be felt up to a height of 3½ in. above the pubes. The upper border seemed somewhat flatter than the normal fundus uteri. The glands in the groins were slightly enlarged. The vulva was normal, except that the perineum had been torn. A tumour as big as a fist was found in the vagina. It was rough on the surface and somewhat mushroom-like in shape, having a perfectly smooth stalk about one and a quarter in. thick. The surface of the tumour was irregular, hardish, and had adherent to it large pieces of placenta and membranes, the latter being green from putrefaction. The rim of the cervix could be felt all round the stalk. The uterus was inverted: the fingers can be inserted for ¼ in. in front of the stalk, and nearly ¾ in. posteriorly (*see* fig. 1). The body was absent from its normal position. There was a horribly offensive discharge. The speculum showed the placental site to be in a sloughy condition. The membranes and small pieces of placenta were removed with the fingers: but some adherent portions were left behind. Continuous irrigation with warm boric acid solution was given and this lessened the discharge and offensiveness.

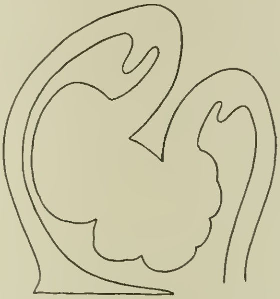

FIG. 1 (Case I).

Showing the irregular surface produced by inflammation of the inverted placental site and the cleft at the fundus, probably traumatic.

On September 27, the continuous irrigation being still employed, the inverted uterus was rather smaller than before, and in its lower part a cleft could be felt nearly $\frac{3}{4}$ in. deep into which the finger could be passed. This was probably a wound made in removing fragments of placenta. On September 30 the discharge was sweet. The mass was much higher up and smaller and was now mainly within the cervical canal. The cleft felt a little shallower. The lower end of the tumour could now be received into the mouth of a tubular speculum $1\frac{1}{2}$ in. in diameter. It was uneven on the surface, pink in parts, in others covered with a yellowish exudate. There was no bleeding on examination. The cervical canal was now $1\frac{1}{2}$ in. in length. The irrigation was continued up till October 6, when the patient began to menstruate. On October 11 the cervix was found in normal position, large and patulous, and the os admitted the fingers to the root of the nail, the body had become spontaneously replaced in its normal position, was large, round and fairly movable. There was slightly increased resistance in the left posterior quarter of the pelvis, otherwise nothing abnormal was found. The patient left the hospital on October 21. A small ulcerated surface was seen on the left posterior lip of the cervix; this healed in about a fortnight. Some thickening was still to be felt in each posterior quarter of the pelvis when examined on November 8. The temperature reached 101° F. on the day of admission, 102·2° F. next day, 100·6° F. on the third day; afterwards it did not reach 100° F. and was normal nearly every day during her stay in the hospital.

Case II.—A. H., aged 27, was admitted to University College Hospital on March 17, 1901. She was an unmarried cook in the family of a doctor who was unaware of her pregnancy. She had been delivered, unattended, on March 12 at 3 a.m. while straining to pass water, the child (apparently of seven and a half months' development) falling into the chamber pot. A great deal of blood gushed out at the same time. The patient got up and went to the wash-hand stand to get a drink of water, but fainted away. She had a feeling as if something had given way. When she recovered from the fainting attack she crept into bed, still bleeding; there was no pain and no vomiting. A doctor was sent for and found the child dead. Apparently he did not make a vaginal examination. On March 17, five days after the delivery, I saw the case in consultation. The patient was very pale, the uterus was completely inverted and occupied the vagina. The abdomen was quite flat; the perineum was slightly torn. The lining of the uterus was fairly smooth, pinkish grey in colour, the orifices of the Fallopian tubes could not be seen. The uterus was fleshy in consistence. On making pressure the fingers were found to sink into it and it was felt that the uterus might easily be perforated; it was decided not to attempt to replace the organ until it was clean and more involuted. Boric acid douches were ordered and a catheter was passed every eight hours to relieve retention of urine. On the 20th the patient felt well; but the temperature varied between 98·8° F. and 101·4° F. On the 21st continuous irrigation with boric acid solution was given: the patient found this very uncomfortable. On March 28 nothing could be felt above the pubes. The uterus, completely inverted, lay with its fundus about 3 in. in diameter, on the perineum. A slight rim could be felt at the cervical end. At 2.30 p.m. (on the sixteenth day of the puerperium) Aveling's repositor was applied. In the course of five minutes the inverted fundus was pushed out of reach and a tumour could be felt above the pubes. Temperature 98·6° F. Three times during the day the repositor was found to have slipped into the anterior fornix, the last occasion being at 10 p.m. This displacement of the repositor caused pain, and $\frac{1}{4}$ gr. of morphia was given, hypodermically. At 11.15 p.m. eight and three quarter hours after the first insertion of the repositor and one and a quarter hour after its final application the uterus was found replaced and the repositor was withdrawn by traction without much difficulty. A douche was given and next day continuous irrigation was employed. The patient gradually improved in colour and left the hospital well on April 15. I am indebted to Dr. Teale for the following observations on the blood:—

	March 21	March 28	April 10
Hæmoglobin ...	24 per cent. ...	25 per cent. ...	36 per cent.
Red corpuscles ...	600,000 (12 per cent.) ...	1,800,000 ...	3,000,000
Colour index ...	2 ...	0·7 ...	0·6
White corpuscles ...	15,000 ...	9,700 ...	9,000
Lymphocytes ...	25 per cent. ...	23 per cent. ...	38 per cent.
Neutrophil (polynuclears) ...	73 per cent. ...	75·6 per cent. ...	59·4 per cent.
Eosinophils ...	1 per cent. ...	2·2 per cent. ...	1·2 per cent.

The highest temperatures were 101° F. on the day the repositor was applied, next day 101·4° F., on the third day 100·8° F.; afterwards below 100° F., except on one occasion (100·6°).

Case III.—Mrs. W., aged 24, was placed under my care at St. Saviour's Hospital, by my friend Mr. John Murray, on June 13, 1903, twenty-two days after her first confinement (May 22, 1903). She had had a long and painful labour lasting thirty-eight hours, but there had been no difficulty until the third stage. It was said that the cord was pulled upon and the patient lost consciousness and bled profusely. The accident was not recognized by the doctor, who was not the patient's ordinary attendant but a temporary assistant, and the patient continued to bleed somewhat freely for three weeks, ceasing two days before admission. The patient was then very pale, but in fair general condition. The uterus was completely inverted and was well involuted for the time. I introduced Aveling's repositor at 9 a.m. It caused very little pain, and in the side rather than in the abdomen; the pain was apparently due to lying on the side. At 2.30 p.m. I examined her and found the displacement completely reduced and the cup in the uterus; it was withdrawn without much difficulty by pulling. The pulse had gone up to 140, respiration 28. After removal of the instrument the patient had no pain at all. Next day (14th) the pulse was 96, temperature 99° F., and the patient felt quite well. There was very little (coloured) discharge; the uterus remained in normal position; the external os easily admitted the finger, the internal os was closed. On June 17 there was no pain and very little discharge; the patient was still pale, but felt quite well. She was taking tr. ferri perchlor. 20 minims. There was no tenderness over the uterus. On June 20 the patient felt a little stiffness in the left thigh, but no tenderness, swelling or exudate was felt in the pelvis and the uterus was freely movable. On June 22 there was still a little stiffness in the left leg; it was diminished by movement and was probably rheumatic. The cervix was closed. The colour had improved. June 25: Patient got up. June 29: Patient was quite well except for a little pallor; she had no pain or trouble of any kind. The uterus was quite normal, freely movable. The cervix was notched slightly on the right side and behind. The sound passed $2\frac{1}{2}$ in.

In 1914 the patient became pregnant and was delivered of a living seven and a half months child in the spring of 1915 by Dr. Montague Smith, after an easy labour lasting four hours. There was no difficulty with the placenta. The child throve and the mother made a good recovery.

Case IV.—A. T., aged 33, a widow (four children), was admitted to University College Hospital on February 20, 1904, complaining of loss of blood from the front passage. The blood was clotted and very black in colour. The loss used to occur between, but recently only at the menstrual periods. The discharge was often jelly-like. The patient had no pain, but suffered a little from palpitation. She had no trouble with micturition or defæcation. Menstruation occurred first at 15 years of age, was always regular every twenty-eight days, lasting five days and requiring fourteen diapers. Lately she had used thirty diapers and the period lasted nine days and occurred every twenty-eight days. Her trouble dated from her last confinement, two years and eight months ago, June 7, 1901: the labour lasted twenty-seven hours and was attended by a doctor's assistant. The child was born head first and lived. No instruments nor anæsthetics were used. The patient had a great loss of blood after the child was born and became unconscious. In fourteen days she was so weak that she could scarcely speak. For three days large clots were passed: on the third day she wanted to pass a motion and something came out of the front passage. She sent for the doctor, who washed the lump with "gallons of water" and pushed it back into the vagina. Another doctor now attended her and told her the womb was in the wrong position, and advised her to go into the hospital: she said she was too weak to move and lay bleeding for five weeks, when she went to the country and could walk about a little. She now stopped bleeding and had no hæmorrhage for thirteen months, suckling her child throughout. In April, 1903, she was admitted to a provincial hospital, where she was told that her womb would always be a trouble, and that nothing could be done for her except hysterectomy; this she refused. She then saw Dr. Stork, who said he would try an instrument. A cup with a straight stem was put in for a day and a night; but it caused a good deal of bleeding and had to be removed. Another attempt was made with the same instrument for fifteen hours a week later, but without effect. After admission to University College Hospital, Aveling's repositor was applied at 4.30 p.m. on February 25 (two years and eight months after the occurrence of the inversion). The uterus was found to be reduced on the 27th at 12.30 p.m., forty-four hours after the application of the repositor. The cup was withdrawn by a 6 lb. weight extension in two and a half hours. The strain on each rubber band attached to the repositor was about 4 lb. The temperature was over 100° F. on admission; it remained up to 101° F. for the most part, on one occasion reaching 102·4° F., but gradually fell to normal on March 10 when the patient left the hospital. She was in good health ten years later.

Case V.—R. B., aged 27, married eight years (one child, no abortion), was admitted to University College Hospital on January 22, 1916, complaining of "prolapse of the womb" since her confinement nine months ago (April 25, 1915). The labour was at term, and was terminated by instruments, but no particulars could be obtained from the medical attendant as to pressure on the fundus or traction on the cord. The patient lost so much blood afterwards that she fainted away several times on the third day. She remained in bed for nearly three weeks, during which time the lochial discharge and bleeding were profuse; after the month it got less, though it continued, the bleeding being at times rather free, but for the most part scanty; there had also been a thin yellowish inoffensive discharge. The patient had suckled the child till her admission. The urine had to be drawn off for two days after the child was born, but there had been no difficulty with micturition since. The patient had had peritonitis eight years ago, a fortnight after marriage, and, three years later, had an attack of rheumatic fever. Menstruation began at the age of 14, was always irregular and rather scanty, lasting only three days, and being painful for the first two days. On admission the patient was very pale, the lips, hands and lobes of ears nearly white, the tongue clean and moist, the temperature slightly raised. Nothing abnormal could be detected in the chest or abdomen. On vaginal examination the uterus was found to be completely inverted and well involuted, the mucous membrane smooth and red, the orifices of the Fallopian tubes could not be seen. The cervix passed smoothly on to the vaginal fornix on the left side and in front, apparently on account of a tear. Elsewhere there was a slight shallow groove around the inverted portion. Warm boric acid douches were given for a week, and on January 29, 1916, Aveling's repositor was applied at 11.30 a.m. The pressure exerted by the elastic bands was about 4 lb. Next day the repositor was removed and the fundus was found to have been repressed, but still projected about 1 in. beyond the os. The repositor was replaced. Morphia was given for the pain, which was mainly due to stiffness in the hips from lying in one position. Temperature 99·8° F. On January 31, 1916, at 11 a.m., examination showed that the inversion was completely reduced forty-seven and a half hours after the application of the repositor. The temperature was 100·5° F., pulse 150. The instrument was removed without any difficulty in a few seconds by drawing it down and then tilting the cup on one side while still attached to the stem. On February 1, 1916, the uterus was found to be in good position, ante-flexed and freely movable. The os admitted the finger easily: an old tear was felt on the left side and in front. The patient felt a little stiff, but had no pain. She had no trouble afterwards, gradually gained strength and colour and the temperature gradually fell to normal. She left the hospital on February 9 quite well, though still anæmic. Dr. Teale's report on the blood is as follows:—

	January 27, 1916	February 4, 1916
Red cells ...	2,020,000	2,120,000
White cells	10,000	16,000
Small lymphocytes	17 per cent.	13 per cent.
Large ,,	5 ,,	4 ,,
Hyaline ...	1 ,,	2 ,,
Transitional	2 ,,	—
Polymorph. neutrophils	74 ,,	81 per cent.
Hæmoglobin	40 ,,	—
Colour index	0·9	—

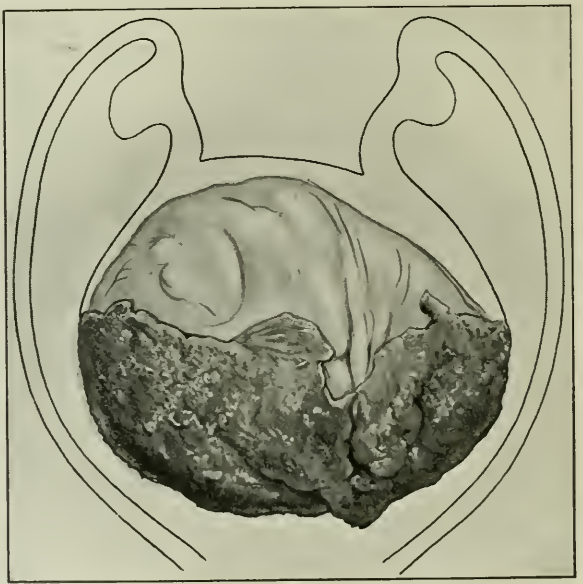

Fig. 2 (Case VI).

Showing by diagram the condition found on examination, with an accurate drawing of the sloughing tumour removed (three-fourths natural size).

Case VI.—M. B., aged 43 (four children, three abortions), was admitted to University College Hospital on February 29, 1904, complaining of a thick dirty white discharge from the vagina and pain in the back. She dated her illness from March, 1897, when she had a miscarriage; since then she had never been regular. In December, 1901, she was three weeks in bed with hæmorrhage, and again in December, 1903, she lost a large amount of blood for five weeks; it was at this time that she noticed the pain in the back. Menstruation began at 12 to 13 years of age, was regular every twenty-eight days, and at first seldom lasted more than two days. She had miscarried within three months of her marriage at the age of 26. After her first child the placenta was removed under chloroform. Some months later she had another miscarriage, and subsequently two more children. In 1895 she had another child, and in 1897 another miscarriage. Since that date she had not been regular, losing excessively, especially since 1899. On examination there was a mass in the vagina as big as a small orange. It appeared at first to be malignant. It was so large that without an anæsthetic the finger could not be passed round it to determine its attachments and mobility. There was an exceedingly foul-smelling discharge. Under ether an irregular tumour as large as an orange was found in the vagina. It was green in colour and œdematous and excessively foul: it was too friable to hold the volsella. Its surface was rough: around its neck, which was smooth, a continuous shallow groove could be felt. The body was absent from its normal position and was completely inverted by a tumour attached to its fundus. The condition is roughly represented by the diagram taken at the time, with an accurate drawing of the tumour by Mr. Shiells (fig. 2). It was found difficult to reach the healthy mucous membrane

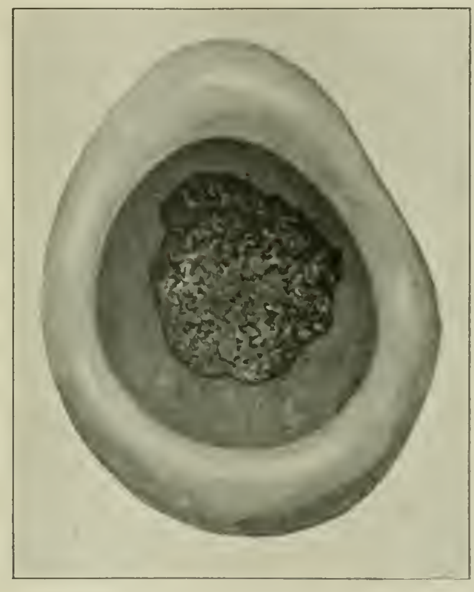

FIG. 3 (Case VI).

Showing the gaping cervix and the rough site of attachment of the tumour immediately after the tumour had been removed and the fundus restored to its normal position (three-fourths natural size).

owing to the size of the tumour: an incision was therefore made through its gangrenous surface bisecting the tumour, which was then easily enucleated. The collar of mucous membrane then retracted and was carefully trimmed with scissors. The inverted fundus was swabbed with 1 per cent. formalin solution and pushed up; this was easily accomplished owing to the wide dilatation of the cervix. The parts then had the size and appearance shown in the drawing made from a sketch taken at the time (fig. 3). An iodoform gauze tampon was tightly packed in the uterus and was removed next day. Perchloride of mercury douches were then given. The patient made a good recovery. The temperature, 99·6° F. on admission, only twice reached 99° F. after the operation. She left the hospital on March 20.

Case VII.—M. J., aged 41 (two children), was admitted to University College Hospital on June 23, 1904, complaining of severe pain for fifteen days, offensive discharge for thirteen days, and pain at the periods, especially on the last occasion, when it was very severe. Menstruation began at the age of 14, recurred every thirty days, lasting ten days, and requiring thirty diapers. She had always had a good deal of pain for the first two days of the flow. She had never had any intermenstrual bleeding. The patient had had two children, the last twelve years ago. On examination there was a greenish-yellow discharge, which had been offensive in odour. Projecting into the vagina was an irregular body as big as a Tangerine orange, of firm consistence, not breaking down on pressure. It had a thick smooth stalk, which could be felt to be surrounded by the rim of the dilated cervix. On passing a speculum the lower end of the tumour was found to be of a greenish-yellow colour, owing to a superficial slough. Under an anæsthetic it was found that the tumour had inverted the uterus, not completely, but so that the reflexion of the cervical canal was half an inch up in front of the lip and nearly an inch behind. The tumour, which was sessile on the anterior wall of the fundus, was seized with a volsella and pulled upon; a circular incision was then made behind the gangrenous portion, and the tumour was easily removed by enucleation, the inversion meanwhile being rendered almost complete. The mucous membrane was then trimmed with scissors and the fundus easily replaced by pressing upon it with a tampon. The uterine cavity then measured 4 in. in length. An iodoform gauze tampon was applied and removed after a few hours, during which a little blood had been lost. A little hæmorrhagic discharge continued for five days. The patient left the hospital on July 6, quite well, except that she was anæmic. The temperature only once rose to 100° F. after the operation.

Case VIII.—Mrs. B., aged 50 (two children, no abortion), was seen in consultation with Dr. T. Sayer, on November 11, 1909, on account of severe hæmorrhage. She had had two children, no miscarriage; the last child was born twenty-five years ago. Menstruation began first at the age of 14, was always regular and not excessive in amount until three years ago. For the last two years she had had a continuous loss, only having a day or two free.

She never had much pain at the periods till the last two years and latterly it had been less severe. There was no family history of tumour. When I saw her there was a tumour in the vagina resembling a polypus, at least twice as big as the normal uterus, of uniform consistence and of piriform shape, with the smaller end upwards. It was too big for an old, inverted uterus. As the patient was stout and difficult to examine, she was admitted to a nursing home and anæsthetized and then it was found that the uterus was completely inverted. There was a slight groove at the edge of the cervix, but the body was absent from its normal position and on drawing down and inspecting the neck there was no cervical canal (fig. 4). It was quite certain that the uterus was inverted; the organ was regular, smooth, and of uniform consistence: the difficulty was to account for its size, which was quite twice the normal bulk of an old, inverted uterus. I came to the conclusion that there was probably a soft intramural fibroid although there was no line of demarcation, groove or hardness pointing to a fibroid, and as I had never seen nor read of such a case, I confess I incised the fundus with some anxiety. The diagnosis proved to be correct: a degenerated myoma of the size of a pigeon's egg, with cystic cavities in it was easily enucleated. The inverted organ was then easily replaced with the fingers and an iodoform gauze tampon put in for twelve hours. There was a slight rise of temperature (up to 99·6° F.) and pulse (up to 108) during the convalescence; but the patient made a good recovery, left the home at the end of a fortnight, and was well when last seen by her medical attendant three or four years after the operation.

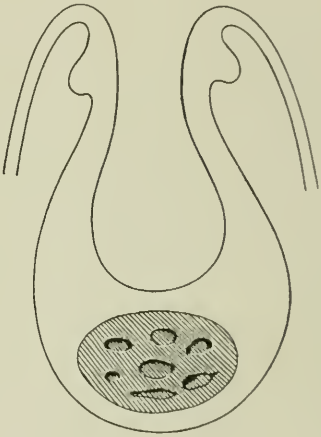

FIG. 4 (Case VIII).

Diagram showing enlargement of the inverted uterus produced by a cystic myoma embedded in the wall of the fundus and no distinguishable, either by a line of demarcation or consistence, from the rest of the body (half natural size).

Case IX.—A. J., aged 45 (ten children, no abortion), was admitted to University College Hospital on October 7, 1912, complaining of irregular menstruation for the last three years, the intervals varying from two weeks to six months. There was great loss of blood and some pain at the periods, and a thin, watery discharge in the intervals. The patient had lost weight lately. Her last child was born thirteen years ago; only six of her children were living. The patient on admission was well nourished and looked healthy. There was some tenderness and resistance above the pubes, but no mass could be felt. The cervix had been torn; the edges of the tear were smooth and not ulcerated. The body of the uterus appeared to be enlarged. There was no thickening of the broad ligaments. The cervix bled on examination and the supravaginal cervix appeared to be thickened. The uterus was ante-flexed and fairly movable. The finger could be introduced into the os and did not feel any growth. Examined under anæsthesia the uterus was 3 in. long, the appendages appeared normal, the cervix patulous. A small piece of soft white growth was removed by the curette from the body of the uterus. The growth looked like cancer. Unfortunately the cervix was not dilated in order to pass the finger into the uterine cavity. The microscopic examination showed to my surprise what appeared to be benign mucosa containing glands with a single layer of epithelium or in one or two places with two or more layers evidently due to obliquity of the section: the glands lay in a stroma which did not attract attention as differing very much from the ordinary. The case was regarded as one of "endometritis." The patient did not lose weight during her stay in hospital. She left on October 16. During the next three or four weeks the patient had no loss at all. Then considerable hæmorrhage occurred and a watery discharge and difficulty and pain with micturition and defæcation. After examination the doctor wrote that there was a large "cancerous" mass in the vagina and that the patient had recently had retention of urine for twenty-four hours. She was readmitted on January 5, 1915, and was found to be very ill, sallow and anæmic. A large tumour could be felt by the abdomen reaching nearly up to the umbilicus. The tumour felt tense and elastic, but did not give a fluid thrill to percussion and was not altered by passing the catheter. The vagina was filled by a large irregular mass which pressed on the perineum and broke away and bled freely on examination. There was a very offensive discharge. The cervix could not be felt; but the case appeared to be one of a large sarcoma inverting the uterus and stretching the vagina far up into the abdomen. On January 17, 1913, under anæsthesia, after removal of part of the growth by the hand the uterus was found to be completely inverted by a lobulated and papillary tumour growing from nearly the whole of the body (fig. 5). By pulling on the tumour the cervix and vagina were found to be smooth. The mass was now reduced in size to that of a double-fist and broke down and bled readily. Vaginal hysterectomy was performed by bisecting the tumour and ligating the broad ligaments with thick silk. A small portion of the cervix on the right side was left for fear of damaging the bladder, which had been located by the sound. The patient recovered fairly well, but a minute vesico-vaginal fistula occurred and was at once treated effectively by touching it with a cautery. The temperature during convalescence was slightly raised, the highest point being 101·8° F. The patient left the hospital on March 4, 1913. Microscopic examination showed the tumour to be a spindle-cell sarcoma. The patient was admitted again on May 30, 1913. She remained well for sixteen weeks after the hysterectomy; but bleeding began again on May 11. A red lobulated growth was found depending from the top of the vagina and a large mass could be felt extending out to the right pelvic wall. An attempt was made to do an extensive abdominal operation; but the intestines were found to be so extremely adherent at the site of growth that the abdominal wound was closed and the vaginal growths were removed with the cautery. On July 8 the vaginal growths had recurred: the patient looked very ill. It was intended to try the effect of radium; but the patient left the hospital on July 18 and died on August 29, after suffering great agony from the recurrent growth.

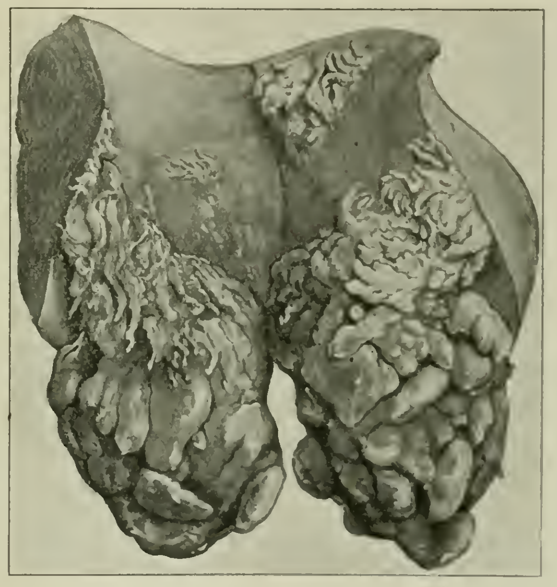

FIG. 5 (Case IX).

Showing the uterus (which was bisected during the operation) inverted by sarcoma. Above is the cut edge of the cervix with a patch of shaggy growth just below it. The greater part of the lining of the body is covered with a lobulated and shaggy growth. The growths are spindle cell sarcoma. Large masses were removed from the lower end of the tumour before the hysterectomy was performed (three-fifths natural size).

REMARKS.

These nine cases of inversion of the uterus are the only cases (apart from inversion produced during operation[1]) which I have had under my own care. I have also seen five cases under the care of Sir John Williams, three of which followed labour and were reduced by Aveling's repositor, and two were caused by sarcoma and were reduced after removal of the growth; all these five cases recovered. I am induced by this experience of a rare accident to make some remarks upon the subject of the treatment of chronic inversion of the uterus.

Puerperal Inversion.

The younger generation of British gynæcologists, following in the wake of the Germans, show a tendency to neglect the simple, safe, and effective treatment of chronic puerperal inversion originated in 1878 by Aveling, who, in inventing his "repositor," conferred honour on British gynæcology and benefit upon humanity. Some of them have recognized the disadvantage of operating through the vagina and have substituted for it operation by the abdomen with its somewhat greater risk; but I think the cases here described and many others (such as the ten cases published by Aveling, three of which were under his own care) show that cutting operations for chronic puerperal inversion are rarely, if ever, required, and they have the great disadvantage over the use of the repositor that they weaken the uterus and render it liable to rupture in a subsequent delivery, to say nothing of the necessity of administering an anæsthetic to patients enfeebled by hæmorrhage, the time taken by convalescence, and the dangers and disabilities, slight though they be, which follow vaginal and abdominal section.

The drawbacks to the use of Aveling's repositor arise from using an unsuitable instrument, or a proper instrument in unsuitable conditions, or in an improper way. Difficulty may also occur in extracting the instrument if it is left too long in the uterus.

The instrument should be made after Aveling's own model, the essential feature of which is a cup carried on a curved German-silver stem, which is soft enough to permit it to be bent so as to transmit the pressure in the axis of the pelvic brim, and yet so rigid as not to bend under the strain of the elastic bands which keep up a constant pressure. In applying the instrument a cup of suitable size should be employed, and the elastic bands (letter bands) fixed at the end of the stem should be stretched and then fixed by two tapes, one in front and one behind, hanging over or through a 3 in. band of strapping fixed round the patient's waist, the tapes being tied with a bow-knot. Thus a gentle continuous elastic pressure is maintained, the resultant force acting in the axis of the pelvic brim. The tapes may need re-tying as the reduction proceeds, and it is important, by frequent examination, to ascertain the progress made, to see that the cup has not slipped off the fundus, and to withdraw it as soon as possible after reduction has occurred. When the cup slips into the fornix it gives rise to pain, and by prolonged pressure might cause ulceration of the fornix. The narrow stem of Aveling's instrument permits examination while the cup is in position, which is not possible with the cylindrical and conical modifications of the cup which have been suggested by Galabin and Blacker with the object of rendering its extraction easier.

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[1] I have several times produced inversion during the enucleation through the os of large submucous or intramural myomata. If the tumour has been completely removed and the peritoneum has not been opened, I have always replaced the organ. If the peritoneum has been opened I have either stitched up the hole and replaced the organ (one case) or removed the uterus by the vagina (several cases). In one case of Cæsarean section I inverted the organ through the incision in order to permit the complete removal of adherent membranes. All these cases recovered.

As to the cases suitable for Aveling's repositor any case of chronic puerperal inversion is suitable, provided the proper time be selected for its use.

An important question arises: How soon after labour should the instrument be used? In this communication I say nothing about *acute* puerperal inversion which should be reduced manually at once. But if twenty-four or forty-eight hours have elapsed before the case is seen, and a cautious attempt at manual replacement fails, I believe it is wiser to resort to vaginal irrigation with boric acid or saline fluid, and to postpone treatment with Aveling's repositor until the parts have become clean and firm. In support of this opinion I would allude to Case I, in which the irrigation was sufficient of itself to reduce the inverted organ; to Case II, in which the uterus, continuously irrigated, was successfully reduced by the repositor eleven days later; and, on the other hand, to the late Dr. Maxwell's[1] case, in which immediate reduction on the third day terminated fatally by sepsis.

These patients, when they survive the shock and hæmorrhage at the time of the accident, suffer from severe anæmia, aggravated by bleeding and discharge, until involution takes place. Yet in spite of offensive discharges and fever they do not get peritoneal symptoms, owing probably to the complete drainage and to the compression to which the veins and lymphatics are subjected in the inverted organ. Involution is usually complete and menstruation ceases during suckling, so that the local and general condition may be greatly improved by waiting. I think the best time for the reduction will usually be towards the end of the puerperium or as soon as the parts are in good condition. There seems no limit to the time after labour at which reduction is possible; thus Tyler Smith[1] reduced an inverted uterus of eleven years' standing, and the patient bore a child two years later when the uterus again became inverted and was readily reduced, and subsequently had other children,[2] and Audigé (whose publication I have not been able to obtain) is stated by Pozzi[3] to mention a case successfully treated which dated back thirty years.

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[1] *Proc. Roy. Soc. Med.*, 1915, viii (Sect. Obstet. and Gyn.), p. 53.

With regard to extracting the cup after reduction by Aveling's repositor no great difficulty is experienced if the cup has not remained long in the uterus, which is best insured by repeated examinations, and it may be easily overcome by Dr. Handfield-Jones's suggestion of continuous traction. This will be rendered more speedy if the cup be unscrewed and seized by the edge with strong forceps.

As to the time taken to effect reduction with Aveling's repositor, that will vary with the duration of the inversion and with the care taken in carrying out the details of treatment mentioned: in any case it should take place within forty-eight hours (Dr. Lewers says two weeks! I should have thought this was a misprint for two *days* had not other gynæcologists given a long time for reduction; the explanation no doubt being, as Dr. Herman pointed out, that the elastic bands employed were too weak). If frequent examinations be made in order to make sure that the cup is in position and that the elastic bands remain stretched, I think a few hours will usually suffice for a fairly recent case (*see* Case II, in which the cup slipped several times, but finally reduced the inversion in one and a quarter hours, having been employed in all for eight and three quarter hours).

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[1] Tyler Smith, *Med. Chir. Trans.*, 1858, xli, p. 185.

[2] J. Marion Sims, "Uterine Surgery," 1886, p. 137.

[3] S. Pozzi, "Traité de Gynécologie," 1905, p. 746.

Inversion due to Myoma.

It is an axiom amongst gynæcologists never to remove a uterine polypus before making sure that the "polypus" is neither the inverted uterus nor the sessile myoma inverting the organ. There is a specimen in University College Hospital Museum illustrating the grave error committed by an experienced surgeon of amputating the uterus inverted by a myoma, with a fatal result. A fibroid polypus may be easily distinguished from an inverted uterus by the fact that the uterus is in its normal position and its cavity present or even lengthened. The only polypi which give rise to any difficulty are those with their stalks adherent to the cervical canal (in this case the adhesions are easily separated and the uterine canal revealed) and cases of myoma of the portio vaginalis in which it is sometimes difficult to find the minute external os, displaced to one side.

The orifices of the Fallopian tubes can sometimes (but not usually) be seen on the inverted organ: in one case the orifice was dilated, and this led the assistant to diagnose a hypertrophied cervix, as the tube admitted a sound for 4 in. Careful and bimanual examination in these cases will clear up the difficulty. The existence of a myoma inverting the organ is usually made out with ease by the harder consistence of the tumour, which is usually separated by a slight groove from the mucous membrane of the uterus proper. Case VIII, in which the inversion was due to a small degenerated intramural myoma which did not alter the shape or consistence of the organ is, as far as I know, unique.

The treatment consists in removing the myoma either by circumcision or bisection with enucleation of the two halves. The organ is then easily replaced by pressure, and a packing of iodoform gauze is applied for twelve hours.

Inversion due to Sarcoma.

The endeavour should be made to diagnose the sarcoma before it has inverted the organ. This is most easily done by examination of the organ with the finger after dilatation with laminaria tents and by removing portions of growth for microscopic examination. Case IX shows how necessary it is to pay attention to the stroma as well as to the epithelial structures in curettings. If inversion has occurred, the growth sometimes assumes a mushroom shape, and may be removed with the écraseur or scissors, hysterectomy being subsequently performed. Or, if the growth is large, sessile or infected, vaginal hysterectomy followed by the application of radium or X-rays may be the best treatment.

Conclusions.

(1) For inversion of the puerperal uterus, if not seen till twenty-four or forty-eight hours after its occurrence, a cautious endeavour may be made to replace the inverted organ by hand. If the uterus is soft or septic, irrigation with boric acid or salt solution should be employed, and no attempt be made to replace the organ until the latter part of the puerperium.

(2) *Aveling's repositor is the best means of replacing the chronically inverted uterus, and it is unjustifiable to perform any cutting operation until the repositor has been tried.*

(3) It is the writer's belief that the original Aveling's repositor will replace any chronically inverted uterus after labour. Should it fail he would operate by the abdomen, incising the neck and replacing the organ.

(4) Before removing a fibroid polypus it should be ascertained that the uterus is not inverted. In cases of myoma inverting the uterus, it is not usually necessary to remove the uterus, but only the tumour: the uterus being then easily replaced.

(5) For sarcoma inverting the uterus, if polypoid the tumour may be removed *per vaginam* and the uterus subsequently *per abdomen*; if the tumour is large and infected, vaginal hysterectomy with the subsequent application of radium or X-rays may be the best treatment.

DISCUSSION.

Sir FRANCIS CHAMPNEYS: The subject of Dr. Spencer's paper is welcome. It is one on which there is a surprising amount of ignorance, though it is by no means obscure. The proportion of cases in which the inversion is complicated with other conditions such as tumours strikes me as high: this may possibly be due to a diminution in uncomplicated puerperal inversions. I have little to say on the subject of the complicated cases, except to express general approval of the principles on which Dr. Spencer has treated them. As regards uncomplicated chronic inversion, the subject is generally very poorly treated in books. The great principles are: (1) That the uterus when inverted undergoes normal involution; (2) that involution is complete at eight weeks or thereabouts; (3) that at this date the case becomes technically "chronic"; (4) that it becomes no more chronic in twenty years; (5) that it is as easy to reinvert an inverted uterus twenty years after its inversion as after eight weeks; (6) that reposition with Aveling's repositor properly used practically never fails. I have seen a good number of these cases, and that is my experience. It was also the experience of Dr. Matthews Duncan. Adhesions in the inverted uterine cup are extremely rare. In adjusting the repositor, the best elastic is indiarubber tubing; it has to be adjusted like the shrouds of a bowsprit, and the direction of the axis of the repositor, which should be more or less in the pelvic axis, can be easily regulated by the relative length and tension of the elastic tubes. Care must be taken that the cup of the repositor clasps the inverted fundus, the largest cup first, gradually diminishing as the fundus is pushed within the cervix. Two days should see it over. To remove the cup the best way is to "reverse engines," and tie the elastics to the foot of the bed. Cutting operations are unnecessary and unjustifiable.

Dr. AMAND ROUTH: Dr. Spencer is right in his statement that Aveling's repositor practically never fails to reduce an uncomplicated chronic inversion, provided that patient supervision be exercised. The spontaneous and unrecognized cases are those in which inversion has only been partial at first and then gradually becomes complete. In such cases shock is less marked than when the inversion is acute and complete, for the sudden constriction of important structures drawn down into and imprisoned in the inverted body is the main cause of the shock. The anæmia which is associated with chronic cases of inversion is due to the concurrent septic toxæmia which makes convalescence very slow.

Dr. HANDFIELD-JONES: I strongly advocate the use of Aveling's repositor, for I have not met with any case in which this instrument has failed to accomplish its object. I would emphasize the fact that shock is by no means a constant symptom in acute inversion of the womb; it seems that errors in diagnosis sometimes arise from too great stress being laid on the occurrence of this symptom. I have had several cases in which the womb has become completely inverted after delivery at full-term, and yet no evidence of shock has been shown.

Dr. HERBERT SPENCER (in reply): I am glad to find myself in agreement with Sir Francis Champneys, Dr. Routh and Dr. Handfield-Jones, as to the uniform success of Aveling's repositor in reducing a chronic inversion of the puerperal uterus.

Section of Obstetrics and Gynæcology.

President—Mr. J. D. Malcolm, F.R.C.S.Ed.

DISCUSSION ON THE REPORT ON THE TEACHING OF OBSTETRICS AND GYNÆCOLOGY.[1]

Dr. T. W. Eden.

The Teaching Committee was appointed to inquire into and report upon the teaching of midwifery and gynæcology to medical students and graduates in London. The heart of the matter lies in the teaching of midwifery to students and I shall therefore confine my remarks to that subject.

The training of students in midwifery is largely determined by the requirements of the examining bodies. In London we are concerned with the Society of Apothecaries, the Conjoint Board, and the Universities of London, Oxford and Cambridge. Generally speaking the view of the examining bodies is that the essential part of midwifery training is attendance upon, or conduct of, twenty cases of labour. The Society of Apothecaries allows the student to be signed up for his twenty cases by any general practitioner. This is in direct contravention of the regulations of the General Medical Council. The Conjoint Board and the University of London require the certificate of attendance to be signed by a member of the hospital staff. The Universities of Oxford and Cambridge require a certain amount of hospital training in addition. These latter regulations are comparatively new and came about as follows :—

In 1906, Sir John Williams, then a member of the General Medical Council, reported to the Council that its regulations were not at that time being complied with by the Medical Schools, and that students were not gaining the requisite knowledge for the efficient practice of midwifery. A Committee, of which he was Chairman, advised that every student should be compelled to do his practical midwifery in hospital, where he should give it undivided attention for a period of at least one month. Unfortunately this very sound provision was not adopted by the Council, but eventually its regulations were altered so that every student was to attend for three months the indoor practice of a lying-in hospital, or the midwifery ward of a general hospital, and during that time to conduct twenty cases under medical supervision—or, alternatively, he should attend for one month, and being adjudged competent, should proceed to the maternity district and there complete his twenty cases. The Universities of Oxford and Cambridge are the only examining bodies which have attempted to comply with these regulations.

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[1] At a meeting of the Section, held December 4, 1919.

Under present arrangements our students, therefore, actually attend their cases in three different ways:—

(*a*) On a maternity district of their own hospital.

(*b*) Partly in the midwifery ward and partly on the district.

(*c*) In a lying-in hospital alone.

The great majority come under the first heading.

Practically all these students begin their cases on the district without ever having seen a woman delivered either in lying-in hospital, maternity ward, or elsewhere, and without having received any *clinical* instruction in the conduct of labour. The hospital regulations provide that the Obstetric Resident should accompany them to their first one or two cases. Usually this is done, but the Obstetric Resident may have other things to do and cannot always accompany the student, who may then have to attend his first case alone. He is also directed to report all abnormal conditions during labour or the puerperium to the Obstetric Resident. Owing to his lack of training it is often difficult for him to know whether the conditions are normal or not. During his attendance on his cases there is no supervision, and he has no practical assistance of any kind, even in the management of the infant, a subject of which he is profoundly ignorant. Students trained in this way therefore gain their first ideas of the conduct of labour under the muddled, makeshift, insanitary conditions which are usually to be found in the homes of the poor. They have no hospital standard by which to correct it, for they have never seen labour conducted under hospital conditions. What wonder if the student concludes that strict surgical cleanliness and careful precautionary measures are unnecessary in midwifery!

The case of the student who goes first to the midwifery ward is much better. Here he sees labour properly conducted under hospital conditions before he begins his work on the district, and he receives a certain amount of clinical instruction from his teachers. Unfortunately, very few students have this training, as only four teaching hospitals in London at the present time have a midwifery ward for training students.

The third case, that of the student who goes only to the lying-in hospital, arises from the fact that certain hospitals, owing to the nature of the locality in which they are placed, have only a small maternity district, and are unable to provide all their students with twenty cases to attend. These students go to a lying-in hospital where they can get "signed up" in fourteen to twenty-one days, as having attended twenty labours. The greater number of the labours which they attend, they attend as spectators only, standing round to watch someone else deliver the patient. They do actually deliver a certain number themselves. In these hospitals practically all normal labours are conducted by the resident midwives, therefore the certificate of attendance is in direct contravention of the regulations of the General Medical Council which require that the student should attend the cases under the supervision of the medical officer. In addition the student sees a certain number of abnormal cases and gains a certain amount of clinical instruction from the visiting physicians on their rounds. He does not attend cases on the district at all.

There is no doubt that on the whole the training of our students is really bad; nothing more unsuitable than the district conditions could possibly be devised for training students; and it must be admitted that the unsatisfactory results of midwifery practice in general are largely the result of bad training. These bad results of midwifery were brought before the Society in May last by Mr. Bonney,[1] and I may remind the meeting that Mr. Bonney pointed out that during the last twenty-five years, a period in which wound infection has been practically eliminated from surgical operations, there has been almost no improvement in the national death-rate from puerperal infection, the rate having fallen actually from 5·8 per 1,000 to 5 per 1,000 only. In addition, the mortality rate from accidents and diseases in pregnancy and in labour continues very high. It has fallen in about the same proportion only. To give a concrete example, I may point out that during the years 1912-15 from 1,200 to 1,300 women died per annum from puerperal infection in the United Kingdom, and further, from 550 to 600 women died per annum from hæmorrhage in connexion with pregnancy or labour.

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[1] *Proceedings*, 1919, xii, pp. 77-107.

The actual mortality is not the only point. We all know how much chronic ill-health and disability for work results from the minor infections of labour which are not attended by any mortality. The out-patients' departments of our hospitals are thronged with such cases. The economic value to the State of female labour is very great, and the loss which occurs from these illnesses is of national importance. A further frequent late result of minor infections is sterility, which has also its bearing on the national birth-rate. In addition, the bearing of this matter on infant mortality is important. The doctor who attends the confinement is in charge of the infant for the first month of its life, and it is during the first month that infant mortality is highest. It is therefore of great importance that doctors should be well trained in the management of the newborn child.

The conclusion drawn from these facts is that the general level of midwifery practice is low and should be raised without delay. Medical practitioners are not entirely to blame, because 40 per cent. of the confinements in the United Kingdom are attended by midwives, but much of the responsibility rests with the medical profession. I do not suggest that practitioners are solely to blame. I hold that we as teachers must bear a great part of the responsibility ourselves because we have been working on a bad system, and if our students are badly trained we cannot expect their practice to be good.

Up to this point there will probably be general agreement among us. When we come to consider what has to be done our difficulties begin. It is necessary to look at the question broadly having regard to :—

(1) The educational needs of the student.

(2) The public health needs of the community, and the necessity for establishing a proper midwifery service for London.

(3) The interests of all London students, which must be regarded as a whole and not from the point of view of individual hospitals.

The present inequality of teaching as between one hospital and another is one of the weakest points in the medical education of London. The members of the Teaching Committee had no preconceived opinions on the subject, and I should like to explain, if I can, how we arrived at the recommendations we have made.

In the first place, we were satisfied that present conditions are so bad that only drastic and far-reaching changes would suffice. It was no use tinkering at the present system.

In the next place, it was obvious to us that the amount of time allotted in the curriculum to midwifery and gynæcology is quite inadequate, and would have to be largely increased. We suggest that the students should devote four months to these subjects alone.

The next step was that proper training in practical midwifery could only be given in hospital. The management of normal labour is perhaps the most important single clinical subject of the curriculum, for it is of this work the young practitioner gets most as soon as he begins practice. The management of normal labour must be taught as a surgical procedure, under proper conditions, and this can only be done in hospital. In addition, students should be taught in the hospital and under the eye of their teacher how to deal with all the common obstetric difficulties they will meet with in practice.

If these principles are to be carried out it is clear that a very large increase in the hospital accommodation for midwifery cases is required. Fortunately this is required not only from the educational point of view, but also in the public interests. There is no doubt that midwifery accommodation in London is scandalously deficient. It is very difficult to get the obstetric emergency case into any hospital. This is particularly the case with puerperal fever; the lying-in hospitals cannot take such cases at all; very few general hospitals can do so, therefore they drift—some to the Poor Law infirmaries, and others to the fever hospitals, where they come under the care of specialists in diphtheria and scarlet fever. It ought to be possible for all obstetric emergencies to receive hospital assistance as readily as all surgical emergencies receive such assistance now.

Further, we thought that midwifery training should be extended in both directions—i.e., so as to include a fuller view of pregnancy, by means of the ante-natal clinics, and a fuller training on the management of the infant, through the infant-welfare clinics.

Lastly, we formed the conclusion that the senior obstetric teachers ought to take a very much larger share in the practical teaching than they do at present. The share they now take is very small and compares unfavourably with what is done by teachers in medicine and surgery. Practical midwifery is irregular work, and unless the senior teachers are always available they cannot take their proper share in teaching. We say that all serious obstetric difficulties should be dealt with, not as now by residents and registrars, but by the senior men, who should even take their share in teaching students the conduct of normal labour. The moral effect, as an object lesson, upon the students of seeing their senior teachers conducting a simple normal case under the same minute and exact precautions as they have been taught are necessary, will be of the greatest possible value, and will be quite worthy of the senior teacher's attention.

The next question we had to face was, could these principles be put in practice with our present institutions?

First of all we turned to the lying-in hospitals and after consideration we came to the conclusion that they would not do. In the first place they are needed for the training of midwives, the supply of which is quite inadequate. If their patients are utilized for the training of medical students they will be lost for the training of midwives. Again, these institutions are so organized that they practically exist upon the revenue received from midwives' fees. If the midwives are withdrawn, or their numbers diminished, these hospitals will be in serious financial difficulties. And, further, the practical instruction in normal labour in these institutions is all given by midwives. It is against the regulations of the General Medical Council that students should be so taught, and we are quite in agreement with these regulations.

The next alternative was the midwifery ward of the general hospital. Could this system be so far developed as to meet the requirements of the situation? We think that it could to a great extent, and we have sketched on pages 15 and 17 of our report a scheme for enlarged midwifery wards (departments) which we think would be very useful. It should have a minimum of seventy-five beds, fifty for midwifery cases and twenty-five for gynæcology. The students should be in residence, at any rate for a part of their training, for if not in residence they will never see the night-time cases and will lose a great part of their opportunities of learning. They ought to be attached to the department for a period of three or four months. Such a department could be run by two or three visiting physicians, a resident "chef de clinique" of the status of a resident assistant surgeon, with a certain number of resident assistants under him.

The objection to this scheme is, in the first place, that only the largest hospitals could give up the required number of beds—probably not more than four of them. If only four of such departments were formed it would simply exaggerate the present inequality existing between one hospital and another, and we therefore say that arrangements should be made by which students from other hospitals should be allowed to attend these departments for instruction in practical midwifery.

In order to meet the needs of all the London students at least six such departments would be necessary. Such a large increase in midwifery accommodation in the central parts of London, where these hospitals are situated, is not required by the public needs, and we cannot count upon Government assistance in establishing them. Whatever may have been done in the past it is quite certain that in the future hospital accommodation will be provided where the public need it and not where it is convenient to the schools to have it. An increasing proportion of the cost of medical education is being borne by the Government, and their views must be considered.

While the provision of such departments will be an important step in the right direction it will not provide a final solution of the difficulty, for the reason just mentioned. There must therefore be an alternative and the alternative we have suggested is the formation of institutions which to give them a name we have called "centres." They would be hospitals for women of a type new to London but similar to the Rotunda Hospital in Dublin. They would be for the reception of pregnant women, women in labour, lying-in women, nursing women with their infants, and the ordinary run of gynæcological cases, and they would be organized to provide ample training for medical students and graduates, and to provide also facilities for research.

As we see it, such an institution would be a large one, containing, say, 200 beds, 140 for midwifery and sixty for gynæcology. Its staff would be entirely a resident staff, and they would have to be highly paid, as the best men are required. There would be a resident director, who would be in charge of the clinical work, the laboratory work, and the teaching. Under him would be two assistant directors, and under them a sufficient staff of resident assistants. The students would be in residence for a part of their training, and they would be attached to the "centre" in all for four months.

The advantages of such a scheme are as follows:—

In the first place these centres could be arranged in parts of London where urgent public need exists for increased midwifery service. On this point I would like to mention one important fact. In 1915 the Local Government Board issued a Report on the maternal mortality of childbirth in the United Kingdom. A study of this Report shows that in comparing one district with another the death-rate is highest in districts where there is least hospital accommodation, and it is lowest in districts where hospital accommodation is greatest. There is therefore every reason to believe that the increased provision of maternity hospitals would have a very beneficial effect upon the mortality in connexion with childbirth.

Another advantage would be that ample clinical material would be provided for training students and junior practitioners.

In the next place the system, by which all the teachers would be resident, would enable the senior teachers to take their proper share in practical teaching, which they cannot do at present, and it would offer them scope for clinical work and for research which would be worthy of the very best men.

There are certain obvious objections, of course, to the scheme, the most important perhaps being that which our critics have pointed out when they say that the "centre" would be isolated from general hospital services such as surgery, medicine and pathology. I should like to say at once that I do not admit that these centres need be isolated, but even if they were I say that it is better to teach the students properly in separate institutions than to train them imperfectly within the four walls of their own school. But it must be recollected that the outlying districts which require midwifery accommodation require general hospital services too, and in all probability general hospitals will be built simultaneously with the provision of such centres.

In addition, it is our view that schools which are unable to provide a large midwifery department of their own will be affiliated to the new centres. These teaching hospitals might in the first place provide the medical, surgical and radiological services required by the centre, which would form a close bond between them. In addition, we expect that the medical staff of the centre would be recruited from the affiliated school, thus forming another link between them, so that the students when they go to the centre would not be altogether out of touch with their own teachers.

It must be borne in mind that there is in progress a movement of concentration in clinical teaching. The waste of time and effort which results from the multiplication of schools and of teachers in London is a very serious matter. The formation of units of medicine and surgery is the first expression of the new movement. If we are going to demand a higher standard of efficiency from general practitioners there will have to be a higher standard of efficiency of teachers too. The position of the present teacher is a very difficult one. Most of us belong to two, or perhaps more, hospitals. This involves an enormous amount of unpaid work, and takes up a great deal of time in routine; we are engaged more or less in writing or in research, we have to earn a livelihood, and, in addition, we are teachers. To men so occupied teaching can be nothing more than an incident in their daily work, whereas teaching ought to be the principal concern of a teacher.

We say, then, that much more will be required of teachers than has been required in the past; and as regards the teaching of midwifery, we believe that it can only be taught properly if senior teachers will give their whole, or practically their whole, time to it for a series of, say, five years, during which they will be in residence in the teaching centre.

In conclusion, I want to say that we recognize that if we are to get on quickly with the job there must be something like substantial agreement among us. I do not think the differences between us and our critics are really fundamental. We agree that their proposals are good proposals, but we do not think they are adequate to the serious situation which has arisen. We think they are timid proposals, lacking in courage, and lacking in thoroughness, and we hope that as the result of this discussion they will be inclined to take a broader view of the matter. In any case there is no time to spare. The General Medical Council is again stirring in the matter; the Government departments are alive to the importance of midwifery from the point of view of public health, and it is much better that we, as teachers, should be in a position to take our share in directing the changes that are coming, than that we should have to submit unwillingly to changes imposed upon us by others.

A Criticism of the Report of the Committee of the Council of the Section of Obstetrics and Gynæcology of the Royal Society of Medicine upon the Teaching of Obstetrics and Gynæcology in London.

By Lady BARRETT, C.B.E., M.D., HERBERT SPENCER, M.D., and HERBERT WILLIAMSON, M.D.

(Read by Dr. HERBERT SPENCER).

WE endorse the sections of the report dealing with the deficiencies of the present system of education in obstetrics and gynæcology and wish to express our indebtedness to the Committee for their clear and outspoken review. We agree fully that reforms are urgently needed but we do not think the system proposed is the best that can be devised.

Although an intermediate phase is suggested the ultimate goal of the Committee is a system of education based upon two essential principles:—

(1) The establishment of large centres devoted solely to the study of obstetrics and gynæcology and not forming part of a general hospital and medical school.

(2) That the student can only be educated efficiently when large masses of clinical material are available.

We think these two principles are wrong. We believe that the obstetric and gynæcological clinic should be part and parcel of a general hospital and medical school. These subjects are only one part of general medicine and are intimately connected with every other branch. From the point of view both of the student's education and the advancement of the science they should be kept in the closest possible touch with other branches of medicine; the student should not be taught to regard them as something separate and distinct. There is no essential difference between puerperal infections and other wound infections; the fact that cardiac disease complicates pregnancy does not put it in a new category, nor is pyelitis of pregnancy a disease *sui generis*.

Close co-operation should exist between the gynæcologist, the pathologist, the surgeon and the physician; such co-operation is only possible when the obstetric clinic forms part of a general hospital both geographically and constitutionally. It is essential that the help of specialists in every branch of medicine should be available—a radiologist, a neurologist, a chemical pathologist, a physiologist, a bacteriologist, a morbid anatomist and an ophthalmologist, to mention only a few. Consultations should be frequent and should take place at the bedside of the patient in the presence of the student. By such consultations the student is taught the essential unity of medicine and the application of general principles to special problems. The consultants learn much from one another and the patient is benefited. This close co-operation can only be obtained within the walls of a general hospital.

We do not think very large quantities of clinical material are essential for the education of the student.

The clinic need not be a large one. The minimum requirements are:—

(1) An out-patient pre-maternity clinic.

(2) A few (say six) pre-maternity beds.

(3) A lying-in ward of not less than twenty beds.

(4) A few (say four) beds for septic cases.

(5) An out-patient maternity district adequately supervised.

(6) An infants' welfare centre—preferably a branch of the children's department.

(7) A gynæcological ward of twenty-five to thirty beds with operating theatre.

(8) A well-equipped clinical laboratory.

(9) A room for storage and investigation of morbid anatomical material.

The Staff of such a clinic should consist of:—

(1) Three visiting obstetricians, all of whom should teach both obstetrics and gynæcology.

(2) A resident officer of the standing of a resident assistant surgeon or physician. He should hold office for a period of not less than three years, and should be responsible, under the direction of the visiting staff, for the practical instruction of the students in the lying-in ward, and should also be responsible for seeing that the pathological work of the clinic is carried out. This officer should receive a salary of not less than £500 per annum.

(3) A registrar and tutor.

(4) Two resident officers of the standing of house physicians or house surgeons.

We believe this system to offer many advantages to the student, the school, and the public over that of the whole-time officer proposed by the Committee.

The system of teaching large classes of students by means of lecture-demonstrations produces a practitioner inferior to the one produced by a system which aims at the instruction of the student individually by the bedside of the patient. A few cases adequately investigated and well demonstrated teach the student more than many cases hurried over.

We think it desirable that obstetrics and gynæcology should be taught as one subject, should be taught by the same teachers, and that the student's studies in the two branches should be carried on simultaneously.

We think that the clinic should remain essentially a part of the general hospital, and the whole course in obstetrics and gynæcology should be taken in one school.

We think that every medical school should have its own lying-in ward. At the present time this appears to be impossible in the case of some of the smaller schools, and in our opinion the deficiency would best be met by amalgamation. One or two new hospital centres in outlying parts of London might be formed in the near future by co-operation amongst themselves of the smaller teaching hospitals, and by association of their plans for expansions with State schemes of hospital provision. This would involve the sacrifice of their present central positions and the fusing of two or more hospitals and their staffs in the creation of the new hospital. The medical school would still be retained, and its students would have the advantages of modern and efficient hospital equipment, of adequate buildings, of working in a district where the need was great, and of a consultation service with local practitioners. The funds obtained by disposal of existing hospital sites supplemented by State grants would be used for building, equipment, and endowment of the new hospital.

It is likely that under the Ministry of Health obstetric and gynæcological institutions will be established in outlying districts of London, probably as parts of new general hospitals. If these institutions are staffed on the principle suggested by the Committee they will form centres for research and post-graduate work. Such institutions will be of the greatest value and are urgently needed.

We think that the Director of Clinic of these new institutions should hold office in most cases for a limited term of years, and on his resignation of the post be eligible for the staff of the hospitals with medical schools. The experience and skill he gained as director would then become available for the general public and for the education of medical students.

Dr. Herbert Spencer.

I should like to add a few remarks to the conjoint report by Lady Barrett, Dr. Williamson and myself. First I will emphasize the points in which the majority and the minority reports agree—viz., the need for more beds for gynæcology and midwifery, the need for the devotion of more time by the students to the study of these subjects and the teaching of the two subjects at the same time and by the same teachers.

The essential difference in the two reports is that the minority uphold, and give reasons for upholding, the present system of teaching the subjects in the general hospitals; the majority propose that they should be taught in special institutions. Yet the majority evidently foresee difficulties in the future in keeping these institutions *special*, the needs of the public will require *general* hospitals, and the system will then be essentially the same as at present. But the general "centres," with 10 per cent. of the beds devoted to obstetrics and gynæcology, will need to have 2,000 beds and, with the directors' and the students' residences, will involve an enormous expense. And I do not think, if the existing general hospitals keep their out-door maternities, that the "centres" will find it easy to fill their beds with patients suitable for teaching purposes.

I should like to point out that the recommendation of the majority report that the directors should be allowed private practice is opposed to the rules under which the few directors of units have been already appointed and will give rise to a good deal of opposition.

Principally I object to the *direction* of studies by one individual as being opposed to the liberty which has always characterized British medicine. It is a German method and entirely contrary to the British spirit of individual freedom.

Dr. Russell Andrews.

There is very much that I should like to say, but I must confine my remarks to only a small part of our subject.

The old system of midwifery training has turned out a certain number of legally qualified medical practitioners who are nothing less than a danger to the community and to the State. In the East End of London obstetric tragedies are still terribly common—preventable tragedies which are due mainly to hurry and ill-applied force. This is a matter on which I feel very strongly. These cases in which skilled help is not called in until brute force has done its worst are some of the saddest cases which one sees in practice. Some of my obstetrical friends think that I exaggerate and that I still talk about what I used to see twenty-five years ago, but I assure you that I do not exaggerate and that I am talking about what happens at the present day. My late colleague, Drummond Maxwell, was just as strongly impressed as I am, and I am sure that my colleagues, Dr. Holland and Dr. Luker, and Mr. Gordon Ley—from his experience during the war—will back me up. In the limited time at my disposal I can mention only one case, that of a normally built primipara who was admitted into my wards this year after the child had been killed during delivery with the forceps. She had a complete rupture of the perineum and a vesico-vaginal fistula. The labour had lasted, from start to finish, twelve and a half hours! Cases occur in which patients are sent up to the hospital because of obstructed labour, the forceps having failed. The child is often dead on admission. The only obstruction that can be found is the incompletely dilated cervix. The treatment adopted is to leave the patient to deliver herself, which she does with little difficulty after full dilatation of the os, the second stage having often been rendered easy by means of a deep laceration of the posterior vaginal wall and perineal body caused by the slipping of the forceps during the unsuccessful attempts at delivery. When I see a patient whose cervix or perineum has been badly torn in an instrumental first labour I always ask how long the labour lasted, and the answer often is, less than twelve hours. In some cases irreparable damage has been done to the mother before she is sent to the hospital. A few years ago I showed before this Section a uterus the removal of which I had completed, the incomplete removal having been effected by a doctor who said that he had done his best—he had pulled for four hours as hard as he could. He had not noticed that one blade, if not both blades of the forceps, had been placed outside the cervix. Examination before the onset of labour is seldom carried out in some poor-class practices. It is impossible to believe that these obstetric tragedies, resulting in the death of the child and injury or death of the mother, could occur except extremely rarely in the practice of a man who had as a student spent three or four months in an obstetrical atmosphere, attending the ante-natal department, the lying-in wards, and the gynæcological wards and out-patient department. These cases could only occur under the bad old system under which students seldom had the advantage of seeing labour conducted properly. The difficult cases which they saw during their hospital training were probably managed by a resident who had not had much experience. Surgical operations, except minor ones, are not left to newly qualified residents. Most obstetrical operations, on the other hand, are carried out only nominally by members of the visiting staff, and are really performed by the residents. Even with the best intentions it is impossible for an obstetric physician who has to manage the out-patient department, to give certain lectures and classes, to perform a large number of gynæcological operations, besides earning his own living; it is impossible for him to go down to the hospital at night and spend a long time over a midwifery case. Consequently men when they get into practice feel that they can confidently tackle midwifery cases although they would not dare to attempt simple surgical operations which do not offer nearly so much chance of doing harm. I do not wish to be misunderstood. I am not blaming, in all cases, the overworked and inadequately paid doctor; it is his misfortune, not his fault, that his midwifery training was even more inadequate than his fee. Many people will agree with me that a woman in labour would often be safer under the care of a midwife than under that of the first doctor whom she met haphazard, as he may have been imperfectly trained, whereas the midwife has always seen labours conducted in hospital and has learnt the virtue of patience and of giving the natural forces a fair chance. If students are to be taught properly and to be duly impressed with the importance of ante-natal investigation and of patience and skill in midwifery there must be, on the spot, a man senior to the ordinary hospital resident, a man who has had a thorough training in midwifery, to deal with a certain proportion of the normal cases, and, in the absence of one of his seniors, with all the abnormal cases. He must deal with them as his job, not one of his nominal jobs to be sandwiched in, if possible and convenient, between half a dozen other jobs. You cannot get such a man, and pay him an adequate salary, or persuade a Government Department to do so, if his energies are all to be expended on fifteen to twenty beds. You must have a larger number of beds even if in some cases you have to lose the advantage, an advantage which I appreciate as highly as anyone, of their being in close touch with specialists of every variety.

Professor HENRY BRIGGS (Liverpool).

In response to the President's call I may remark upon the two reports; their confessions are frank and complete. In my judgment this mass of credible evidence is not inconsistent with future action on both reports: on the one, the preservation and extension—not the destruction—of the adequate teaching facilities attached by old roots or attachable by new roots to general hospitals, and on the other the establishment of new "centres." All should be encouraged and from none ought pupil-midwives to be excluded. In general hospitals and universities the widest and best of training and association can be promoted.

Dr. Fairbairn.

As in the previous discussion on this question and in serving on the Sub-Committee which drew up the report I have made my contribution to the constructive side, I propose to devote my remarks this evening to destructive criticism of the alternative scheme as outlined by Dr. Spencer.

The gist of that report could have been expressed in a very few words and most of what Dr. Spencer has just read to us might have been comprised in the following: "The scheme we recommend is one similar to what has been carried on for some years past in those schools with a maternity ward in which students are trained." Dr. Eden has drawn attention to the great differences in the standard of the training, and this report merely states that the training must be brought up to the level of the best. It marks an enormous stride beyond the teaching that was given before the Williams Report of 1906, and I agree that such a clinic as outlined is the best that is going at the present moment. Having for the past ten years had a share in the working of a scheme which, so far as the signatories to the alternative report can conceive, is the ideal to be aimed at, I think I can offer some criticisms from actual experience, but, to begin with, I would draw the attention of this meeting to the unnecessary character of this report. All it can say is that with a little polish here and a lick of paint there, the best of the present system cannot be bettered. It is indeed a barren effort and the Council of this Section would have stultified itself had it adopted it. Except for the normal developments to keep up to the times, most of what is in this report was described in the *British Medical Journal* for 1912,[1] in an account of the working of the first complete year of the clinic at St. Thomas's Hospital. That in itself shows that this alternative report was a "back number" before ever it saw the light of day.

It would be a waste of time to go through its requirements from (1) to (9) and its staff from (1) to (4) (pp. 48, 49), except to say that from experience of a department only five or six beds short of the minimum of the alternative report and with a Staff of three visiting physicians, a registrar and two house officers, I do not see how even in these days of restricted output and forty-four hour weeks, the work could be found to occupy an extra official at £500 a year, "a resident officer of the standing of a resident assistant, surgeon or physician."

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[1] *Brit. Med. Journ.*, 1912, ii, pp. 1005-9.

The alternative report starts from the wrong base line. Its primary object is to keep the medical school, as we now know it, as the unit, and to maintain it as such, and its whole purpose is to confine the student's view to what he can see within the four walls of his school. This brings it up against the basal principles of the report adopted by the Council, which were: (1) That the training of the student should be conducted by an experienced teacher resident in the place, so that he can be responsible for the normal cases and see the abnormal ones through from start to finish and not delegate his supervision to house officers or other juniors. (2) That a large service of beds is needed—at least four or five times the minimal requirements of the alternative report, to provide the student with the necessary training in complicated as well as normal midwifery, and also to attract and keep a teacher of the requisite standing. As it is clearly impossible to provide this service of beds in a general hospital, Dr. Spencer and his two co-signatories are therefore trying to persuade the meeting that large clinical material is positively dangerous to efficient training; and the bogies of lecture demonstrations, large classes of students and such like were produced to frighten you, but I hope you will refuse to be scared by them. Every one concerned with the working of a maternity hospital or ward knows that the vast majority of the cases will be normal—about 80 per cent. of them. It cannot be otherwise if admissions are booked ahead—the place must either be half empty for emergencies or kept going with all and sundry. So I do not think this meeting will be bluffed with the idea that the student can gain experience in the management of difficult as well as normal midwifery better on twenty beds than 200.[1] I feel sure that Dr. Spencer is juggling with figures and his fantastical ideas as to the interference by the Director with the freedom of his juniors in research, and even their teaching will fail to carry conviction. I have worked on the staff of a maternity hospital and on that of a general hospital with a maternity ward and I fully appreciate the advantages of the latter in regard to access to the resources which a general hospital affords, and I would not disagree with that part of the report read by Dr. Spencer. It is the contra side of the account, what has to be paid for the concentration of teaching material, and I think the penalty exacted will diminish as these special centres develop and, one hopes, become linked up with general hospitals as they are sure to be. Hospitals and centres must follow the population they serve, and we must look forward to a time when all the material scattered over London will be brought into the teaching scheme and strive to make it available to all students. The present day concentration round certain hospitals with medical schools is not a permanent state, even though it has existed for centuries.

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[1] During 1918 in twenty beds of the Maternity Ward at St. Thomas's Hospital out of 551 deliveries 453 were normal vertex, two face, seven breech, and two cases of twins. There were only two cases of eclampsia and two of ante-partum hæmorrhage. There were eleven cases of Cæsarean section and two of induction of labour because of contracted pelvis. Forceps deliveries were twenty-four, impacted breech four, brow presentations two, and prolapse of cord one. The number of cases of eclampsia and ante-partum hæmorrhage was below the average—usually there are about six of the former and fifteen of the latter—but the year's record shows the chance the student had in six weeks' residence of seeing abnormal labour.

Lady Barrett, C.B.E.

I should like to endorse what Dr. Eden has said so admirably in reference to the smallness of the difference between those who drew up the report and the few who criticized it. The subsidiary report was not intended to be in any way complete in itself, because all those who signed it agreed so cordially with the rest of their colleagues on most of the issues concerned. The one point upon which they felt very strongly was that undergraduate training in obstetrics and gynæcology ought not to be given in an isolated institution, but in a department organized in close relationship with all other branches of medical studies. The majority report had somewhat recognized this principle in the suggestions made for the development of such departments of obstetrics and gynæcology in teaching hospitals, and Dr. Eden in his closing remarks again expressed agreement with the principle when he hoped that round the associated units of Obstetrics and Gynæcology complete hospitals might ultimately grow up. We desired that the whole needs of the public with regard to medical service should be kept in view from the beginning, as it is probable that there would be demand for services in medicine and surgery in the same districts in which accommodation for midwifery and gynæcology were deficient, and the smaller teaching schools might be combined together to fill this need with advantage both to the public and to their own students.

Dr. W. S. A. Griffith.

Dr. Eden has presented this subject to the meeting in an admirable way; he has given a perfectly clear résumé of the Report of the Committee which has been adopted by the Council. I have waited to hear some effective reply by the opponents of the scheme in support of their views, and some practicable constructive scheme as an alternative to that put forward by the majority, but there has been none.

It appears that the chief and only essential point of difference between them is clear enough. We are all agreed that the ideal course is for each medical school to have its maternity unit in its own premises, but we understand that with the exception of two this is not possible, and in neither of these cases is the accommodation adequate. We say therefore that the only satisfactory alternative is to provide a complete unit outside, which students from all schools could attend. This our critics decline to accept, on grounds the importance of which we all admit—namely, the necessary separation of the units from the other departments of the schools and hospitals, the closer union of which is so desirable. But if we are convinced of the urgent necessity for reform, are we wise to reject a scheme of such great value though not ideal? Ought we not rather to press it forward with all the means we can secure?

I appeal to both sides to avoid trivial but irritating matters in the discussion, and to unite in the production of a scheme worthy of British obstetrics and of the centre of the Empire.

Dr. Herbert Williamson.

I wish to add a few words upon the disadvantage of removing the study of obstetrics and gynæcology from a general hospital to a special institution. We are in a phase of transition as regards our treatment of many conditions. Take for instance the treatment of malignant disease. We are all of us, I suppose, profoundly dissatisfied with the results of operative treatment. We recognize that surgical operations so far have failed to cure in a large percentage of cases, and we look for other methods. The properties of radium are now in process of exploration, but at present we know little of dosage, of methods of application or of mode of action, but no one can doubt that radio-active substances are destined to play a very important rôle in the gynæcological clinic of the future. The best results will not be obtained by the gynæcologist working alone, a band of workers is essential—the surgeon, the radiologist, the physicist, the chemist and the physiologist must all play their part. Such a combination is easy in a general hospital but it will be difficult, well nigh impossible, in a special institution. What I have said of malignant disease is equally true of many other conditions. The toxæmias of pregnancy, the treatment of septic conditions, the treatment of gonorrhœa in women and the prevention of infant mortality are all problems which must be attacked simultaneously by many men working upon different lines but in close touch with one another.

The enlightened policy of the Board of Education, which we devoutly hope and believe will be extended by the Ministry of Health, has made combined research possible, but it will be to a great extent frustrated if the different branches of medicine are cut off from inter-communication with one another. I believe, therefore, that in the cause of science and through that, of the art we practise, we should preserve the study of obstetrics and gynæcology in the general hospital.

The gifts of obstetricians to surgery are immense: it was Semmelweis, an obstetrician, who first taught the true principles of wound infection; it was Simpson, an obstetrician, who gave to surgeons the gift of chloroform, and in return the surgeon, physician and pathologist have given many things to the obstetrician, but results will accrue more rapidly when the workers in the different fields combine in attacking one problem.

We have referred to the importance of teaching the students to take a broad view of medicine, to realize how closely the different parts hang together and how the advance of one branch is dependent on every other branch. The same general principles govern all and must determine the solution of whatever problem may arise in clinical work. If we isolate obstetrics and gynæcology we shall find inevitably that we destroy this sense of unity. By all means encourage your post graduates to work in these special institutions, but do not send your undergraduates there for part of their general education.

Can obstetrics be taught adequately on a comparatively small amount of clinical material? I believe the answer is "Yes." It must be remembered that only the minimal requirements are given in the criticism on the report. We all agree that more is desirable, but we believe that the students will get a better education in such a clinic as we have outlined than in the special institution described in the report. To transplant him out of the environment to which he has become accustomed, to divorce him for the time being from his general teachers, to focus his attention for the time upon one branch of medicine to the exclusion of all others is, we believe, undesirable at this stage of his career. He will learn less from watching many abnormal labours than in conducting one normal labour himself under the immediate supervision of a good and competent teacher. It is not the function of the senior teacher to guide the hands of the student in a series of labours, but to lay down the routine and to found the traditions of his labour ward, whilst one of his junior officers, who has held the post of intern, who is engaged in obstetrical research, who is a whole time man and holds office for a period of not less than three years, watches the student conduct the labour and instils into him by example and by precept the teaching of his chief.

We are laying, and rightly so, more and more stress upon the preventive aspect of obstetrics—what is needed for teaching this is an out-patient pre-maternity clinic, and adequate material for such a clinic is available in every hospital with an extern midwifery district. Physical examination of the pregnant woman, detection of abnormalities, the hygiene of pregnancy and details of pelvimetry can be taught to a large number of students provided that an adequate staff of teachers is available, and that they know how and what to teach. If each case be allotted to a student, if he take the clinical history and examine the urine before the case is brought in to the teacher, if he be made to examine the patient, take the pelvic measurements, determine the lie and position of the child, and finally make a pelvic examination in front of the teacher and of his fellow students, the value to him of such a clinic will be incalculable. Such a clinic is just as easy to work in a general hospital as in a special institution.

Our scheme presupposes that a larger number of hours will be devoted to the work by the senior teachers and that their remuneration will be increased. Further, that the junior teachers will be given a living wage and not compelled to take work outside the hospital to supplement their income. A good laboratory is essential, and the services of a specialist in bio-chemistry, in bacteriology, and in morbid anatomy must be available for consultation. This is our conception of the clinic which is most likely to improve the quality of the teaching and to stimulate further research.

Sir ROBERT MORANT, K.C.B.

In the survey which the main Committee made on this question in framing their report no mention was made of the possibilities of the Poor Law institutions. This appears to have been overlooked or perhaps deliberately set aside when the Committee started to look round to see what material was available. A good deal of the discussion this evening has been devoted to the kind of hospitals that should be developed and on what lines. Much thought is being given in the Ministry of Health to this very serious and urgent problem. The actual provision of hospitals of whatever kind—maternity or anything else—is at present entirely haphazard. It has been nobody's business to survey the ground. Systematic provision presupposes a systematic survey, taking account of all that was on the ground. If the Poor Law is going to be reformed, the existing institutions, losing all their stigma, and the infectious diseases hospitals maintained by the Metropolitan Asylums Board, will all be brought into survey. No one bit of hospital provision can wisely be considered by itself. Those present are looking at the matter from the teaching point of view; but presumably the whole hospital question is to be analysed. It is complicated by the new development of clinics which are, so to speak, out-patient departments dotted about in different places, seldom under the same roof as the hospital; and also by the numerous maternity centres which are springing up. I have been amazed at the statement that it would be difficult to get material—i.e., persons willing to come to such places. At the Ministry of Health we are getting applications from all sorts of people to get sanction for places with, say, twenty maternity beds, just for normal cases. The question rather is: How is all this material best to be dealt with? Those present will have to get their minds on to that. Furthermore, the advice to be given by them to a central department which has to disburse considerable sums of public money must be advice which is agreed on. Some main lines ought to be unanimously accepted. It would be waste of money which would not be tolerated, to spend public funds on pressing forward schemes which could not be shown to be in direct relation to other necessities of bed provision for the metropolis. A careful and scientific hospital survey seems therefore to be most disastrously overdue. May I suggest that the obstetricians should urge representatives of other branches of medicine to collaborate with them in order to see how all should fit together? If the profession is not going to be alive and awake to see the problem which is upon them, the Ministry of Health will be prevented from doing the right thing for the profession. The Ministry ought to be provided with a survey map of a given area to show the different kinds of things that should be included, those that already exist, and those that are needed; how many of each kind; how large; how inter-related; where best situated; with what catchment areas in each case; how related to leading requirements. London offers the most complex problem of any city in the kingdom because of the multiplicity of existing authorities. The Ministry is only there to dispense public money; in this country the determining voice is mainly with the local authority. The survey must be as inclusive as possible. I urge you not only to try for an agreed scheme on this question of midwifery and gynæcology, but also to get it related with hospital provision as a whole.

Dr. Amand Routh.

I am glad that Sir Robert Morant has drawn attention to the maternity cases attended in the Poor Law infirmaries which may be utilized in any scheme for the improvement of teaching in and around London. Infirmary patients are frequently unmarried women, who often have serious complications and a greater mortality. These infirmaries, which are practically general hospitals with maternity beds, may possibly prove to be suitable nuclei of centres, capable of easy local extension into completely organized general hospitals such as the majority report contemplates. Such centres, however, would take years to develop, and if the Ministry of Health and private benevolence fail to find means to build maternity wards for thirty beds on the top floors of the smaller hospitals, and if the General Medical Council insists upon students being taught midwifery in maternity wards by their senior teachers, the smaller hospitals will be forced to send their students to learn their practical obstetrics at one of the large hospitals in the wards of which any such teaching could be provided. This plan could be carried out without delay, and time would be given for other plans to develop.

Mr. Gordon Ley.

With regard to the objection to the Committee's scheme that the provision of the teaching centre will involve large classes, I do not think trouble on this score is likely to arise if, for example, there are four centres, one for each quarter of London. With regard to plenitude of material, I am only acquainted with two lying-in centres. One of them is almost reduced to the floor, and the other is always full. It was never suggested that the director of the centre would be able to do ordinary midwifery practice; he was only to be available for consulting work. The enormous population of London is not really appreciated; it has a population greater than that of Ireland, and while the population of Ireland is receding, that of London is increasing. It seems to me that we require much more space for beds than is actually available, and that we shall go on requiring it.

Dr. G. F. Blacker.

This Section is concerned with two main problems. The first is how to increase the number of lying-in beds to meet the public need, and the second is how to increase the facilities for teaching. As regards the first problem, the present provision is so inadequate as to form an utter scandal. Four institutions such as proposed in the report could easily be filled—there is ample material. A bed should be available for any woman who desires to be confined in a lying-in hospital. In some districts no such hospitals exist. However, this is a matter for the local authority and the Government. The second problem, how to improve the teaching, is the one which primarily concerns the doctors. The teaching of obstetrics as compared with that of surgery is admittedly bad; all are agreed that the student does not get the same quality of teaching of operative procedures and, in fact, does not receive as good value for the fees he pays. Under the alternative report presented in the criticism, the resident in charge of the lying-in hospital would get £500 a year instead of £150, and the teaching would be no better, as it would still be given by men of the standing of registrars and by other residents. The contrast with other subjects in which the staff themselves teach the students would still be maintained. If large centres are started, whole-time directors should, in my opinion, be appointed, with an adequate salary and proper facilities, comparable to those of other clinical units. There is really little difference between the report and the alternative in the criticism, but the report is much the better of the two schemes.

Section of Obstetrics and Gynæcology.

President—Mr. J. D. Malcolm, F.R.C.S.Ed.

The Origin of Epithelial Tumours of the Ovary.[1]

By J. R. Goodall, O.B.E., B.A., M.D., C.M., D.Sc.,

Assistant Professor of Gynæcology, McGill University, Montreal.

This paper is but a résumé of a larger work which will appear shortly in monograph form. A review of the literature involves a colossal work and can be referred to only in a very cursory manner in a work of this nature. Indeed the more one delves into the subject the more one is confused with the multiplicity of views and the number of structures from which tumours within the ovary may arise. Some authors have dealt with embryological studies of the ovary, and others have given their attention to determining the origin of tumours, but seldom has any one author taken up the second portion after having acquired a good knowledge of the first by his own research. It is quite impossible to determine the origin of epithelial tumours, unless one knows the structures from which these tumours may arise.

It is my purpose first to describe the epithelial structures found in the normal ovary, and having determined the origin and development of these, then to pass on to the consideration of the origin of tumours. The work therefore divides itself under two headings:—

(I) The origin of epithelial structures in the ovary.
(II) The origin of epithelial tumours of the ovary.

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[1] At a meeting of the Section, held February 5, 1920.

PART I.

THE ORIGIN OF EPITHELIAL STRUCTURES IN THE OVARY.

It is impossible to understand the reason of many epithelial structures found in the adult human ovary except by a careful and painstaking study of the human embryo at various stages of its development. Human embryos, well preserved and fit for serial sections, are difficult to obtain, and after most careful study of those at my disposal I had to have recourse to comparative embryology to find the origin and nature of many epithelial structures. Comparative embryology has not only the advantage of material easily obtained in a fresh state, but also the advantage of presenting the phenomena of ovarian development in a much clearer sequence of changes. That these phenomena are the same in the higher vertebrates and in the human species cannot be doubted.

This work is based upon sections both serial and non-serial from ovaries of 127 individuals of all ages from a few hours to eighty years, as well as upon serial sections of five human embryos. The comparative study covers roughly 15,000 sections, mostly serial, from the cow, pig, sheep, cat, mouse, rabbit, guinea-pig and the dog, and from embryos of the cow, dog, pig and cat.

It would be impossible to over-emphasize the importance of serial sections. In many instances my most obvious conclusions, drawn from the study of a single slide, suffered a complete contradiction by a mere glance at the neighbouring slides in the series, and it is possible readily to realize that the conclusions of many authors would never have been put on paper, had serial sections, and not single slides, been at their disposal.

For purposes of comparative study leading to a better knowledge of human histology and embryology there are no two domestic animals so useful as the cow and the dog, and since it was the generative organs of these two which led to this research it will best serve my purpose to confine descriptions to these species, describing others only when corroboration or elucidation are necessary.

A careful study of the mammalian ovary leads at once to the important fact that, from a developmental point of view, the resemblance between the ovary and the testis is a very striking one. There is nothing in the testicle that has not had at some time in embryonic life its homologue in the ovary. It therefore follows that of the structures that are permanent in the testicle some are transitory in the ovary and disappear completely, others degenerate but do not disappear; still others remain but are modified to suit a modified function. The female embryo having developed more or less perfect testicles, progresses still further by destruction, by modification and by addition. It is the development of all the testicular elements and their subsequent incomplete atrophy which has made of the ovary such a fertile field for the development of epithelial tumours of the most varied types.

What makes the ovary of one mammal differ most from the same organ of another mammalian lies in the amount of atrophy which ensues after the ovary has reached its full structural development. Of all the species examined, the cow and the bitch and the cat suffer the least amount of atrophy. Hence, for this very reason they are exceptionally convenient for developmental study.

Until recent years it was almost universally thought, chiefly due to the teaching of Waldeyer, that the germinal epithelium of the ovary never penetrated farther than the outer cortical third of the organ. Whatever glandular structures were found within the hilus or medulla of the ovary were thought to be due to the invasion of the ovarian stroma by the tubules of the Wolffian body. The Wolffian body or mesonephros was consequently divided into two portions: that which invaded the ovary was called the genital portion, whereas that outside the boundaries of the ovary was called the urinary portion of the Wolffian body.

Comparative Study of Adult Ovary.—If we examine almost any section from the ovary of the bitch we find an unexpected wealth of epithelial structures. These at first seem to be without order and of several types. In the heart of the ovary, frequently in the midst of the great blood-channels, we find the rete ovarii.

The Rete Ovarii.—This structure corresponds to the rete of the testicle. It is an irregular tortuous gland-like structure, with numerous folds of its mucous membrane, which is lined for the most part by a columnar epithelium. The structure in some animals suggests a low grade of malignant carcinoma. The rete ovarii varies much in size in different species. In my series the sheep presents a very small rete ovarii, ofttimes difficult to find in the centre of the ovary. In the dog and cow it is a large structure, often extending almost from pole to pole of the ovary as it passes on its tortuous course between the main blood-vessels. In the human subject the rete ovarii varies considerably in size in different individuals and even in the two ovaries of the same individual. In one of my cases one ovary is a mass of fœtal remnants without ova, whereas the other contains thousands of ova without any trace of fœtal remnants except the diminutive rete. We must look upon the rete as a fœtal remnant, and its atrophy up to complete disappearance is quite the exception. In my numerous specimens of human ovaries a careful search has always resulted in finding a rete though at times it is quite diminutive and might readily be overlooked. But in several of the ovaries from the sheep I have not been able to find a trace of it.

The Medullary Cords or Rays.—In the ovary of the bitch there are numerous solid cords or tubules which course in a very irregular manner from the cortex to the centre. Their arrangement generally speaking is radial throughout, very irregular and convoluted. In the multiplicity of cells which line them or compose them one may distinguish three dominant types. The lining which conforms with the first type, consists of tall shaggy cells with a wide base and shrunken body prolonged into a thin protoplasmic filament which not infrequently passes across the lumen to join with a similar process from a cell of the opposite side. The nucleus is near the base. This type of lining is found chiefly in the tubular rays. At times a fold starting from the periphery of the tube, causes, on transverse section, an appearance suggestive of a polypus projecting into the lumen. This is also covered with a similar type of cell, and the fibrous core, usually hyaline and without nucleus, is joined by a thin velamentous fibrous tissue with the connective tissue at the periphery of the ray. The second type is found in the solid cords. The dominant cell is syncytioid in type and in its functionless state resembles very closely the syncytioid cells of a young placenta that has been retained *in utero* for a short period after loss of function. The third type is less commonly found and resembles a true glandular duct, lined by cubical epithelium. These three types when traced through serial sections pass at times almost imperceptibly the one into the other. At other times the change is abrupt especially after an acute bend or tortuosity of the tube. In serial section many of the rays are found to communicate at one end with the rete ovarii. The distal end of the ray is lost in the periphery of the ovary.

Development of the Epithelial Tissues of the Ovary.—I will not go into details of length and age (probable) of the various fœtuses. These matters have little or no bearing upon the subject. The first sign of the development of the genital organs is found in a heaping up of the epithelium at a spot on the genito-urinary ridge. Immediately under this, at this early stage, lies the Wolffian body with its ducts and glomeruli. This heaping of epithelium constitutes the germinal layer or the germinal ridge. Its origin, so far as is known, is from the peritoneal or mesothelial surface, and it lies to the inner side of the mesonephros. (A second genital ridge appears slightly later to the outer side of the Wolffian body. This eventually gives rise to Müller's duct. Slight confusion arises here owing to the varied nomenclature used. Some authors apply the term germinal epithelium to the whole covering of the Wolffian body, whilst others restrict it to that portion which gives rise to the ovary. I shall use it in this restricted sense.) This is the beginning of the genital gland—the testes or ovary. Soon this epithelium begins to burrow and to arrange itself in columns coursing more or less at right angles to the surface, and running towards a narrow hilus. During this development the ovary has gradually become pedunculated.

If we now turn our attention to the embryos of the cow at various stages of their development, we find that at one time the ovary and testicle form a pedunculated structure which occupies the inner side of the Wolffian body. To the outer side lies the differentiated tissue which is destined to form Müller's duct, the forerunner of the Fallopian tube. Between these two structures lies the large Wolffian body with a wide base of attachment posteriorly. Its main excretory duct, the Wolffian duct, lies slightly to the inner side of Müller's duct. From the inner surface of this excretory (Wolffian) duct many tubules take origin. They run along the outer surface of the Wolffian eminence, then they bend suddenly upon themselves, their lining epithelium begins to stain more readily with eosin, the cells become much larger and the tubules themselves become very much twisted and finally end in a glomerulus, near the inner surface of the Wolffian body. A small artery issuing from the main arterial trunk in front of the vertebra passes into the Wolffian body at its attached border, courses along its inner free margin to end in the vascular tuft of the glomerulus. Separated by only a very short distance from the uppermost glomeruli, the fibrous tissue of the ovary takes its origin from, and becomes continuous with, the fibrous tissue of the Wolffian body. The glomeruli of the latter are more or less arranged in a single row and therefore their convex surface, where it is pierced by the glomerular artery, lies in towards the ovary. Except at both poles of the ovary, the space between Müller's duct and the hilus of the ovary is quite wide and rounded, filled in by the enormously developed tubules of the Wolffian body. So far no differentiation of ovules is visible. The cells of the whole ovarian surface seem to be uniform. They consist of deeply staining cells which resemble more or less ordinary lymphocytes. In an embryo of slightly more advanced age, we find remarkable changes have taken place. The growth of cells from the germinal layer of the ovary is no longer restricted to the immediate surface of the ovary, but has penetrated into the medulla, and fills the hilus and separating the fibrous tissue, which seems to form a capsule for them, they pass, as deeply staining branching cylinders of tissue, out of the hilus and along the Wolffian body between its free border and the glomeruli. These cylinders gradually grow smaller in extent as they travel along the Wolffian body to end at a point near the free border of the mesosalpinx between the outer pole of the ovary and the fimbriated end of the tube. In their passage along the Wolffian body they enter into intimate contact with, and become continuous with, the cavities of at least two or maybe as many as six of the glomeruli of the Wolffian body. They are separate and distinct from the Wolffian duct throughout their whole course, and enter into contact with it only through the interposition of the tubules and glomeruli of the Wolffian body. *There is, therefore, direct continuity between the surface-epithelium of the ovary and the Wolffian duct through the medium of the medullary cords or tubules, the rete ovarii, the Wolffian glomeruli tubules and duct.*

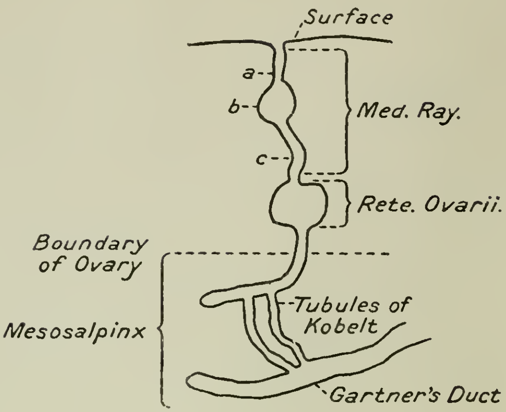
So far as my experience goes, in no other animal can this be studied to such an advantage as in the embryo of the cow. At this stage there is no differentiation of the ovarian epithelial tissue into tubules or ducts. It presents a deeply staining cellular structure, the cells of which possess a minimum of cell-protoplasm with a deeply staining nucleus like that of a lymphocyte. Nothing could be more striking than the contrast between this darkly stained band of germinal epithelium and the adjoining tissues of the Wolffian body. The latter is made up of large cells with a large amount of protoplasm which stains very deeply with eosin. At this stage of development, primordial ova are distinguished within down-growths of surface-epithelium—Pflüger's tubules.

Slightly deeper towards the centre of the ovary, in what I may designate as the second zone, or zone of medullary rays, solid and hollow strands, continuous with Pflüger's tubules, present themselves with primordial ova in their lumina. These are the forerunners of the medullary cords. Still farther towards the centre these tubules in the medullary cords pass insensibly into a richly tubular structure (the rete ovarii) which passes out of the ovary to pursue its course, which I have outlined above, and to enter into contact with the glomeruli and glomerular ends of the Wolffian ducts. In short, what was formerly a mass of darkly staining cells slowly and insensibly differentiates itself into tubules, by a similar process to that by which Pflüger's cords differentiate themselves into Pflüger's tubules, and the medullary cords into medullary tubules. In the ovary, as in the testicle, the epithelial continuity, therefore, is complete from the surface-epithelium of the ovary to the Wolffian duct. The parts nearest the germinal epithelium have to do with the generation of the reproductive elements, the remoter portions are the conduits along which the ova would travel if they pursued the same course as spermatozoa in the male. But in the female, ova are cast off into the peritoneal cavity and the efferent tubules become useless and atrophy completely or in part. Are all these parts maintained in their integrity through fœtal into post-natal and into adult life? The answer is decidedly in the negative, and it is the atrophy which ensues in the ovary owing to the ova pursuing a different route of migration which has led to so much confusion. A study of the adult organ cannot give any connexion whatsoever between these various parts of the one-and-the-same system owing to the complete atrophy of intervening links.

It must not be assumed that the extra-ovarian portion of the canals reaches as complete a development in all other species as it does in the embryo of the cow. For example, in the human subject the extra-ovarian portion of the efferent ducts is relatively small and atrophies easily, so that only that portion remains (for some time) which is necessary to effect a junction with the Wolffian ducts. Such were the conclusions to which my work led me—namely, that there are no parts in the testicle which have not their homologous parts at some time in embryonic life in the ovary. These homologous parts may be clearly represented thus:—

Testicle.		*Ovary.*	
(1) Seminiferous tubules	Of germinal epithelial origin	(1) Pflüger's tubules	Of germinal epithelial origin
(2) Vasa recta		(2) Vasa recta or medullary rays	
(3) Rete testis		(3) Rete ovarii	
(4) Efferent canals ...	Of Wolffian body	(4) Kobelt's tubules of epoöphoron	Of Wolffian body
(5) Vas deferens ...	Of Wolffian duct	(5) Gartner's duct ...	Of Wolffian duct

If we trace the course of the germinal epithelium from its surface to the Wolffian body it could be represented diagrammatically thus:—



The first portion of the down-growth represented by (*a*), (*b*) and (*c*), constitutes the medullary ray. The distal or cortical portion (*a*) atrophies easily and disappears completely. The second portion (*b*) becomes the normal Graafian follicle. The third portion (*c*) atrophies more or less, and may or may not be absorbed. From the lower portion of the rete several tubules are emitted, finally merging into one which passes out of the ovary to unite with the glomeruli of the Wolffian body.

To recapitulate: It has been shown through embryological studies that the ovary at some time in its development possesses a system of tubules, which have their exact homologues in the seminiferous tubules and excretory ducts of the testicle. Embryological studies have also shown that all the structures within the ovary—namely, Pflüger's tubules or cords, the medullary cords and the rete ovarii, to well outside the boundary line of the normal ovary, are of germinal epithelium origin: and it has been demonstrated that the tubules of the epoöphoron, or of the Wolffian body, do not penetrate into the ovary, but are met outside the ovary by the rete ovarii as it passes out of the hilus.

I now wish to take up other facts which have heretofore not been recognized, and which offer incontrovertible arguments for the substantiation of the conclusions stated above.

In the first place, I have found so frequently immature ova within the whole length of the medullary cords, that there can be no doubt that they arose *in situ*. At a certain stage in the development of the ovary, it is not the exception to find this condition of affairs. Their presence is transitory and their life-history is a short one. As we shall see later on, this is the fate of thousands of these ova. They do not all perish. Occasionally an ovum seems to escape. But it is a fact that where one ovum comes to maturity, a thousand are lost in the process. The same statements apply to the rete ovarii, though in a much more restricted sense. In all my specimens of embryos I have found about ten or twelve ova right in the centre of the rete. Inasmuch as these very soon succumb to the general destruction, and as ova have no motive power of their own, and as the character of the tubules would preclude any possible motion being imparted to the ovum, we are forced to the conclusion that this again is incontrovertible evidence of development of ova from the lining both of the medullary cords and of the rete ovarii. Is it not also incontrovertible evidence that these structures are derived from the germinal epithelium, and have retained some of their powers which were impressed upon them in their primitive state? To the arguments of those who claim that both the medullary cords and the rete ovarii are derived from the epoöphoron, I can only add further the statement that it seems quite inadmissible that Nature would allow so important a function as oögenesis to be shared by two structures of totally different origin.

Have these fœtal rests any functions? Without the slightest hesitation one can say that in the human ovary they have no normal function. There cannot be the slightest doubt but that the only normal function they could perform would be the function of oögenesis. But we know equally well that the formation of ova is a function of division and differentiation which in the human species takes place exclusively during fœtal life and the first two years of post-natal life. Scientific opinion upon this point is quite unanimous. But there is not the same unanimity when it comes to the question of the other mammals. In the lowest members of this order, I think it is generally admitted that oögenesis goes on, not only in fœtal and early post-natal years, but throughout the whole of the sexual life. I, myself, am convinced that ova are developed in the bitch throughout her whole life, and that in the case of the cow at particular periods. If such is the case, from what do these ova originate? From a careful study of the bitch's ovary, the different types of tissue which I have described above as being found in the medullary tubes are but different stages in the development of ova. In the second type, where the tissue presents much the character of syncytial cells with vacuoles, it is but the beginning of that development which eventuates in the formation of ova. I am convinced that this goes on throughout at least the sexual life of the bitch, and that ovulation by its stimulating influence, or by the consequent mere increase of vascularity, awakens these oögenetic factors temporarily into activity. I have found ova in all stages of formation. In the vast majority of these developments the ova are quite imperfect and are doomed to a short life. Hundreds of them in one section may be seen as the mere phantoms of ova. If the ovule has developed but slightly (it will have developed a vitelline membrane early in its career) and then succumbs, it will be found with its vitelline membrane somewhat distorted and surrounded by a mass of follicular cells of deeply staining syncytioid characters. If development has gone on further before retrogressive changes set in, the syncytioid cells lose that character and take on the characters of the first type as described above, that is, the ovum, composed now usually only of a vitelline membrane filled with degenerative droplets, is surrounded by a layer of tall, delicate, slender, filamentous cells which stain poorly. This is the prototype of the zona radiata of the discus proligerus.

These facts taken singly or together cannot leave any doubt as to the origin from germinal epithelium of all the tubular and solid epithelial structures found in the subcortical and central zones and hilus of the human ovary. For if such is the case with the higher vertebrates, we cannot but believe that the process is the same in the human species. It seems quite impossible that two tissues so widely different as germinal epithelium on the one hand, and Wolffian ducts on the other, should possess such high potentialities and specialization as are required to develop ova and their necessary adjuncts. True, if we go back far enough, both germinal epithelium and Wolffian ducts were derived from the same cells of the cœlomic cavity. But with that type of argument we need go back but a little further still to realize that all tissues of the body have common parents in an ovum and a spermatozoon.

The atrophy of structures so far described refers only to those superfluous tissues of fœtal origin, but in the dog and in several other animals there takes place a second down-growth of germinal epithelium into the cortex of the ovary. This usually takes place in adult life. The invasions are quite glandular in appearance, never contain ova, and are fruitful sources of new growth with lower animals. Whether such a second invasion takes place in the human subject has never been determined.

In reference to this second invasion of the cortical zone by the germinal epithelium in post-natal or adult life in some animals, von

Winiwarter states: "An important fact remains, we never find in this glandular invasion nuclear elements so characteristically found in germinal cells on their way to become ova. So far as my experience goes this late invasion does not produce ova."

I am convinced that these late characteristic invaginations of the germinal epithelium, at an age when the animal has reached, or almost reached, full development, offer a ready explanation for that strong tendency in the human ovary for the germinal epithelium, when invaginated by any of the causes which will be mentioned later, to burrow and invade deeply, and give rise to a typical growth. Waldeyer vaguely gave expression to the same opinion when he stated that this new formation of tubules from the germinal layer in some of the lower animals was but an expression of an inherited tendency in these cells.

The Interstitial Cells of the Ovary.

In order to avoid confusion, it may be well to state that according to recently accepted views there are two distinct types of cells in the stroma of the ovary. It will not do to speak of them in common as interstitial cells, or as stroma cells, or as connective-tissue cells, because they have distinct separate origins which bind the one type to the epithelial group, and the other to the connective or fibrous-tissue group. The former is of mesothelial, the latter of mesoblastic origin. However, *faute de mieux*, the term "interstitial cells" has passed into common usage, and as a means of distinction the mesoblastic tissue is known as the connective tissue or supporting tissue, or stroma.

Since Limon's and von Winiwarter's articles, an almost infinite number of works have appeared, culminating in Miss Lane-Claypon's and Miss McIlroy's epoch-making article. With a lucidity that deserves the highest praise they have shown that there are two distinct cells in the ovary, the interstitial cell allied to and originating from the germinal epithelium and the connective tissue cell proper derived from the connective tissue of the Wolffian body. The former, therefore, possesses much of the potentiality of the oögenetic tissue, the latter is but a supporting structure. My work upon the human ovary tends in every respect to confirm their work upon the mammalian organ. My specimens fortunately contain three of exceptional importance in this connexion. Two of them are from the human ovary, one of a fœtus of about seven and a half or eight months, the other of a child which died suddenly immediately after birth owing to a diaphragmatic hernia. In each of these specimens, whether due to some peculiarity in the stain, or whether due to the phase of development of these structures, each interstitial cell is so clearly defined and so beautifully stained that it offers the greatest contrast to the slender fibrillar intervening connective tissue. Their origin from the germinal epithelium stands out most clearly, and their differentiation from the follicular cells is as yet not complete. The limit of invasion of these cells in the ovarian tissue is clean cut, and here again offers the most marked contrast to the faintly pink-stained connective tissue. The third specimen is from a rabbit, which had its back broken twenty-four hours before parturition by being clumsily lifted by the neck by one of the orderlies. Immediately paralysis of the hind legs and rear portion of the body set in. Parturition set in next morning, and the rabbit died in the second stage of labour without being able to expel the fœtus. The ovaries in this case were cut. They are from pole to pole just one mass of lutein cells. The outline of the true corpora lutea is well marked by a delicate band of fibrous tissue. Between them and all about them right down to the depths of the hilus, similar cells fill the whole field. Every interstitial cell in the ovary seems to have been converted into a large oval or polygonal cell resembling, in every particular but one, the true lutein cells of the corpora lutea. That one and only distinction consists in a slightly smaller size of the extra-corpus cells. Most minute examination with the highest powers of magnification reveals no other differences. The fine strands of fibrous tissue that interlace with these cells are readily seen. With Sudan III the whole ovary seems a mass of fat.

There can be no doubt that the ovary contains two types of cells, the interstitial and the connective tissue; the former, derivatives of the germinal epithelium and capable of transformation of form and endowed with secretory function; the latter merely supporting structure in the strictest sense of the term.

PART II.

THE ORIGIN OF EPITHELIAL NEW GROWTHS OF THE OVARY.

The work of Part I has led to a generalization the bearing of which is far-reaching, namely, that all epithelial structures within the ovary are derived from a common source—the germinal epithelium.

Division and differentiation have led to wide differences in form and function. But we have seen that the characters—the primitive characters—of this epithelium may make themselves manifest when the proper stimulus comes, and we have seen how imperfectly that assumed function may be performed. This assumption of power is capable only by virtue of the inherited susceptibilities of these cells which are among the oldest in the animal kingdom. With this knowledge afforded by comparative and human embryology, the subsequent discussions will be greatly facilitated.

It would seem at first sight as if we stood to-day just where we did twelve years ago, as regards the histogenesis of simple and malignant tumours of the ovary. The various classifications which have been given have not as a rule been histogenetic, but rather, topical, clinical or histological. Under such systems, the varieties become as a rule (except perhaps the first) so numerous that the subdivision becomes cumbersome. Clinical divisions and pathological subdivisions of any general system have become almost a necessity, but the wholesale multiplication of varieties has much in it that deserves condemnation. I have tried to give a histogenetic subdivision which sacrifices nothing to brevity.

(A) Tumours arising from the germinal epithelium (ovulary; non-ovulary).

(B) Tumours arising from the stroma (ovulary; non-ovulary).

(C) Tumours due to cell inclusions.

"It would be very fine," writes Pfannenstiel, "if when studying each tumour separately, we could place it in its proper histogenetic category." I quite agree with Stratz, that a subdivision of tumours into cystic and solid, is not only unscientific but also impracticable, for neither clinically nor anatomically do tumours allow themselves to be placed in either one of these classes.

What are the structures in the ovary from which epithelial tumours may arise? They are germinal epithelium, follicular epithelium, ova, medullary cords, rete ovarii, corpora lutea, corpora atretica and the interstitial cells. If we include also inclusion tumours, we have covered the whole of the tissues from which they can take origin. But if we ask: can tumours arise from each of these? then the matter becomes one in which you form your own conclusions, either from mere preponderance of opinion or from your own observations. However, it seems advisable to deal with the subject from the view-point of these fixed and known tissues, rather than to take up each class of tumour and try to work back to its origin.

Tumours arising from the Graafian Follicles.

Wendeler has very aptly remarked that it was in the nature of things from the beginning, owing to the cystic formation of most of the tumours, to suppose that they were derived from follicles.

Can the epithelial lining of the Graafian follicle give rise to tumours? In the first place, a distinction must be drawn between the membrana granulosa of a developing Graafian follicle and the single layer of partially developed epithelium which surrounds a primordial ovum in the quiescent state.

If we exclude the condition which is commonly known as "hydrops folliculi," I can say with every emphasis, that I do not believe that tumours can arise from the Graafian follicle in process of development or of degeneration. For years the pathological material from several hospitals, both abroad and at home, has been examined with this end in view, and the result of my observations cannot be expressed in other terms. The varieties of degeneration of the membrana granulosa before or after the death of the ovum are few. The process is singularly simple, and it has never fallen to my lot to see these cells take on characters such as would justify one in assuming that they form the lining of a true cyst.

Are we to classify as tumours the condition known as "hydrops folliculi"? It seems to me that it is a misapplication of a term. Is it even a pathological state? It has happened so often that the small cystic ovary, or fibro-cystic ovary so called, has yielded under the microscope a prolificity of Graafian follicles that is striking. So often is this the case that one grows sceptical of these terms. A careful examination of true cases of hydrops folliculi shows that neither the ovum nor the membrana granulosa have suffered any appreciable change, and I am firmly convinced that the great majority of these ova mature or are cast off by premature rupture of the follicle. In none of these follicles does one find fatty degeneration or secretion in the tunica interna—a condition which forecasts death of the ovum.

Pfannenstiel says they are usually single and one-chambered—i.e., they do not build daughter cysts. Why would they be single if such cysts were of Graafian follicle origin? One would imagine them multiple by the very fact of their origin. Olshausen says when they are multiple, which is seldom, the number is usually small. Most text-books state that the cysts lie side by side and do not communicate, and that the one does not develop out of the other. These arguments seem to have been passed on from text-book to text-book. These two statements, so simple in appearance, would be among the very hardest possible to prove.

Another arbitrary division is found very frequently—namely, that these cysts are always of limited size; for some the fist, for others the fœtal head is the limit. If they exceed this size they pass into another category. This seems the most arbitrary of arbitrary divisions. Now, it is a singular thing that in the many ovaries that have been examined in the past four years from all the species of animals mentioned in the foregoing pages, never has there been a tumour found arising from the Graafian follicles. Hydrops folliculi has been found frequently, but the membrana granulosa was intact. When this had been discharged and had mixed with the liquor folliculi, the cavity became a space lined by flattened cells of the tunica interna. The small cystic ovary of the dog and cat, and many other animals, has quite another origin, as will be described later.

No one will deny the clinical entity known as "hydrops folliculi," but its interpretation admits of more than one opinion. Nagel believes that these cysts are nothing more nor less than unusually large healthy Graafian follicles, and I fully concur. We would hardly classify hydramnios as a new growth, nor does the fœtus succumb even when the quantity of fluid is exceptionally great. Why, then, should the ovum be doomed under somewhat similar circumstances. The fact remains that hydrops folliculi, when it exists, is nearly always multiple, oftentimes classified clinically as the small cystic ovary, yet, according to text-books, the tumour to which hydrops folliculi is supposed to give origin is in the vast majority of cases single, not multiple.

To my mind there is not one case in the literature of a Graafian follicle that is supposed to show developing papilloma, that will stand the test of a scientific criticism. Nagel holds, and I think with perfect justice, that the transformation of the membrana granulosa into a cyst-epithelium owing to the death of the egg, is an impossibility.

In many of the animals in which large and small cysts abound there are many that can with the greatest ease be traced to their origin, and the same applies to my specimens of human ovaries. Yet in not one of these is there any evidence that cysts, as new growths, have arisen from Graafian follicles. It may be objected that the fact that they have not been found in these series is not positive proof that they do not occur. The whole question seems to resolve itself into this: Are we to consider "hydrops folliculi" as a type of new growth or as the product of local change? The question is very difficult. That hydrops folliculi as a clinical entity does exist no one will deny, but that it can reach anything like large dimensions, I think few will be prepared to admit. Through the work of Kroemer we know that the association of cysts of the ovary with chorio-epithelioma of the uterus is quite a common one. We also know that in the ovaries of a pregnant woman, especially if she is somewhat advanced in her pregnancy, we find, not only a large corpus luteum of pregnancy but the other Graafian follicles which contain ova in process of maturation developing a thick layer of lutein cells in their theca interna. It is as though the fœtal metabolism demanded greater lutein secretion and that the theca of these incompletely developed follicles was called on prematurely to do a share of the work. This is the usual course of events as it has been worked out by Seitz and by myself. Such follicles as Limon has pointed out the theca interna of which has undergone, or is undergoing, this lutein change, have their contained ova doomed to destruction.

Chorio-epithelioma acting in a sense like a pregnancy and being derived from a pregnancy, causes all the changes in the ovary that are usually found in a normal pregnancy only to a much greater degree. The ovary becomes distinctly cystic and often of considerable volume. If we examine these cysts, we find that they are mostly, if not all, developed from the corpus luteum of pregnancy and from the Graafian follicles which were maturing when the abnormal impulse overtook them. The truth of the statement lies in the fact that if these cysts are examined in the early stage they are surrounded by layers of lutein cells just as are those Graafian follicles in partial maturation during pregnancy. An impulse to cyst-formation seems to sweep over the ovaries in cases of chorio-epithelioma. These are true Graafian-follicle cysts and not infrequently reach considerable size. Yet—and this is the interesting fact—the large cysts are all devoid of an epithelial lining, being covered either with blood, or serum, or fibrin, or a layer of lutein cells, and only in the smallest can remains of the membrana granulosa be found.

It is very apparent from such cases that a transformation of the lining epithelium of a Graafian follicle into the usual lining of a new growth is an impossibility: that after the death of the ovum the membrana granulosa—a membrana of highly specialized function—has no longer a *raison d'être* and slowly liquefies. If such were not the case, why would not the larger cysts in chorio-epitheliomatous cases still be lined by a membrana granulosa transformed into a one- or two-layered epithelium?

The Follicular Cells of the Primordial Cell-nests.

Do epithelial tumours arise from these cells? Theoretically these cells are so closely allied to the germinal epithelium, they have undergone so little differentiation, that there is no apparent reason why, theoretically, they should not give rise to new growths; yet, practically, such has never been satisfactorily demonstrated. Many authors have published reports of cases in which tumours have been described as having arisen from this structure. The question is a most difficult one to decide, and after all it is a matter of small importance as it will be shown later that epithelial tumours arise either from this primordial follicular epithelium or from invaginations of the germinal epithelium.

Tumours arising out of the follicle-cells of primordial follicles have been described by Gottschalk, Steffeck, Bulius, Pozzi and Beaussenat, Pfannenstiel, v. Velits, v. Kahlden, Hofmeier, Franqué and Limnell, none of which descriptions, however, seem conclusive, except, perhaps, that of Gottschalk. Most of the others, as Waldeyer and Heinrichs state, offer other explanations that are much more readily acceptable.

As stated above, even in the bitch, in which there is normally an invasion of the superficial layer of the ovary by small ducts, which practically never contain normal ova, it is impossible to say under such circumstances whether a tumour, arising in this neighbourhood, originated in one of these small downgrowths or in a real primordial follicle, and so it is in the human ovum. Small cysts situated in the immediate neighbourhood of the surface-epithelium may so mimic the epithelium of the primordial follicle, that it would be quite impossible to state, even at this early stage, with any degree of assurance that it arose from the one or the other of these two sources.

The Origin of Epithelial Tumours from the Germinal Epithelium.

Nothing in the human body can equal the metaplastic power of the germinal epithelium. When we stop to realize that all epithelial tumours of the ovary (except those few which are classified as inclusion tumours) are derived from this epithelium either directly or indirectly through the medium of structures to which it primarily gave rise, we are struck with the latent power of these cells when they are awakened into activity. When we realize that such widely different cells as the apparently simple interstitial cells of the ovary at one end of the list, leads, through many mutations, through the follicular epithelium, membrana granulosa, ciliated epithelium mucous cells to the most highly specialized cell—the ovum—we do cease to wonder at the multiplicity of varieties found among the ovarian new-growths.

We have seen in the first half of this monograph, how by a study of comparative anatomy the whole problem of development and atrophy in the human ovary has taken on a new aspect. In that part of the work, I stated that the amount of atrophy of epithelial structures in the late months of fœtal life and early post-natal life varied greatly not only between different species but between different members of the same species. As examples, I quoted that the difference between the bitch's and the sheep's ovaries in the matter of the amount of apparently functionless structures retained was very great, but that there were also great differences between any two bitches' or any two of any other species. So it is with the human ovary. In some, even in serial sections, I have found not a trace of any fœtal rests except a small rete ovarii. In others, on the other hand, it is impossible to examine any one preparation without finding not only traces but long stretches of fœtal remnants. This applies equally to ovaries of all ages. What the percentage of cases would be in which such structures are found cannot be stated except by making serial sections of a very large number of ovaries at the early adult life. This I have not done, but judging from the frequency with which these structures are found in the ovary before puberty, that is before the ovary is scarred by the rupture of large Graafian follicles, the percentage of cases in which fœtal rests occur must be inordinately high—very much above the figures given by Schickele.

These remnants usually lie deep down in the medulla of the ovary, often in the neighbourhood of the rete ovarii. At times they are in the form of tubules with well-preserved epithelium, at other times the epithelium has remained arrested in a state of partial atrophy. Not infrequently there is a cavity, more frequently they are solid. Often the remains run off from the rete, merely resembling dark lymph-spaces. Towards the surface of the ovary one recognizes these fœtal remnants in the deep crevices or lobulations of the surface of the organ, or by the presence of solid columns of cells, or real ducts which communicate with the surface. If these latter occur before fœtal life, yes, even before full adolescence, they are undoubtedly of fœtal origin, for none of the causes which could give rise to such sinkings have as yet begun to operate.

There are other fœtal remnants that require description. One frequently finds in human ovaries of all ages, certain sectors of the ovary which stand out in marked contrast to the remaining portion of the ovary. Some of these cases will be described. The segment so characteristically changed presents all the appearances of having been arrested in its development, whereby the fœtal structure which should have been absorbed has crystallized and the further development of the segment has not taken place. It is, therefore, a segment which has remained fixed in a stage of imperfect development. A close survey of these cases nearly always leads to the discovery of a vascular defect as an underlying cause. They present an appearance not unlike what might be the late stage of an infarct. The incidence of these cases in those that have come under my observation shows conclusively that this is a common defect. Let me describe some notable cases:—

Case I.—A fœtus which died at full term. The right ovary normal. The left ovary is normal except for a segment about the middle with apex to the medulla. The surface of the ovary at this spot circular and about 1 cm. in diameter, is whiter and of firmer consistence. On macroscopic section no change visible except pin-head cavities. Microscopically, a wedge-shaped portion of the ovary, well defined, is devoid of normal ova or its adjuncts. Instead, the segment is full of glandular or epithelial elements, some of these are typically glandular, others are solid columns which at times degenerate into mere lymph-like spaces. The picture is undoubtedly one of arrested atrophy. Other portions of the ovary are quite normal.

Case II.—A child, aged 1. Right ovary normal. Left ovary contains a segment in the inner pole very similar to that described above. The rest of the ovary normal.

Case III.—Girl, aged 14. Left ovary normal. Right ovary contains at inner pole a wedge of tissue in arrested development. This wedge is filled with epithelial elements and glands. These are frequently broken into short segments. The limits of this wedge are abrupt as if due to an infarct. In many sections the glands contain goblet cells and ciliated epithelium (?).

Case IV.—In this case of a young child, aged 2, an irregular segment of the right ovary seems to have suffered a blight. All about this area the normal structures are found, whereas in this area which, from its outline, gives one the impression that it is due to some vascular disturbance, arrest of development has taken place. The arborescent fœtal structures are lined with cells that stain a deep blue in marked contrast to the pink and blue of the normal tissues.

I think that such a condition whatever its cause offers an explanation for those rare cases in which the ovary remains in its fœtal state, disseminated with canals lined by germinal epithelium, a total absence of ova, a generalized firm fibrous consistence and a proneness to develop multiple cysts. Frommel (?) has published such a case, and one came under my observation some years ago, when working in the laboratories of the Pathological Institute of Freiburg, in Professor Aschoff's service. Quite recently, a second case, somewhat similar, came into my possession. This ovary, differing somewhat from any heretofore described, throws so much light upon the origin of tumours that it will repay a thorough study. It was removed from a young woman aged 23, married three and a half years, no children. Menstruation late in onset had always been scanty. Uterine dysmenorrhœa was not a marked feature. The external genitals were healthy. Owing to severe left-sided pain of a burning character, which gave all the indications of being due to sigmoidal trouble, but which resisted all forms of palliative treatment, an exploratory laparotomy was done. The sigmoid presented nothing pathological on its peritoneal surface. The left ovary was slightly, very slightly, enlarged, but was hard, and almost cartilaginous in consistence. A few small cysts were visible upon the surface. It was deemed advisable to remove this as being possibly a contributing cause. Microscopically, in none of the sections is there an ovum, either in the quiescent or maturing state. The surface-epithelium is unusually tall and columnar. In one of the creases of the surface doubtful cilia are visible. The fibrous stroma is unusually dense and hyaline. The whole ovary is disseminated with small cysts which by their general arrangement run in lines from the surface towards the centre of the ovary. Many of these communicated with the surface-epithelium by a solid strand of cells. In others this was lacking, but by the course of the fibrous tissue, one could easily see that previously such a connexion had existed. The cysts are particularly interesting owing to the variety of their epithelial linings, and owing to the differences in the amount of vascularity of the surrounding fibrous tissue. There are small and large cysts (microscopically); cysts without any apparent epithelial lining, others with a flat lymph-space like covering; still others with cuboidal cells. In this cyst we see tall cylindrical cells, in that one goblet cells, and still further on undoubted ciliated epithelium. In one of the larger cysts the wall has become very vascular and papillary ingrowths are quite frequent. That this is an ovary arrested in its embryonic development, I think, cannot be doubted. It presents all the features of such a condition. How such a condition can arise is indeed quite another problem. And yet I possess among my specimens two which I prize most highly. These were obtained during my work at the Friedrichsheim-Krankenhaus in Berlin. The first was that of a child, aged 5, which had died of chronic nephritis, secondary to scarlet fever. The one ovary was quite normal in every respect. The second was much smaller. The characters of the latter, the evident atrophy of the parenchyma, the shrinkage of the tissues led me to examine the vessels in the hilus. These were unusually small, and cross sections of the main trunks of the vessels farther out in the broad ligament and of the ovarian and uterine vessels of that side showed almost complete closure due either to a developmental defect or to an early obliterative endarteritis.

The second case was that of a girl, aged 18, who died of brain tumour of syphilitic origin. One ovary was perfectly healthy, ovulation had gone on, and there were slight remains of lutein cells and of corpora throughout the ovary. Microscopically this was a perfectly normal ovary. The other ovary was much smaller. The contrast under the microscope could not be more marked. This ovary was like that of a woman aged 50. The fibrous tissue is seemingly more adult in type. There are a few ova which have begun to mature. The majority of these never reach an advanced state of development. The ovary therefore contains retention cavities, the results of these atrophic Graafian follicles. But the most striking feature is the presence of many corpora candicantia, which are similar to those found in the aged who have not sufficient vitality totally to destroy and totally absorb. The arteries in this case also were the seats of a slow chronic obliterating syphilitic endarteritis.

I have quoted this last case as showing the effect of faulty vascularization in the adult, and as throwing some light perhaps upon the previously described cases, which occurred during the developmental or embryonic state, when any change in the blood supply would lead to faulty destruction and absorption of redundant tissues. Of course the only effect such change could have on the adult ovary would be to retard destruction and absorption of functional vessel-scleroses and absorption of the products of corpora lutea and corpora atretica, thereby causing such an ovary to resemble that of an aged woman.

Pfannenstiel has described cases in which the surface epithelium bore patches of ciliated epithelium. Some of these ovaries were quite normal. Others were the seat of tumour growths. Desinety also found islands of ciliated epithelium upon the surface of otherwise normal ovaries. Waldeyer found it in six different cases, in five of which there was cystic formation, and the sixth was the seat of chronic inflammation. Marchand, to explain its presence upon the surface of the ovary, stated that it arose from the fimbria ovarica, and became detached from it. Williams, Walthard, and Kuszman, as previously stated, claimed also that these were isolated portions of the Müllerian duct. In one of my specimens, there are islands of ciliated epithelium in a depression on the surface of the ovary.

To-day it is fully recognized that there is a close relationship between the germinal epithelium and ciliated epithelium, and that the transition from the one type to the other is frequent and easy, and seems but the expression of a character which germinal epithelium once permanently possessed. Pick has drawn attention to the general tendency of the whole of the pelvic peritoneal surface to develop cilia.

In addition to the foregoing cases, which so far as it is possible to ascertain, date back their origin to fœtal life, there are other characteristic invasions of the ovarian stroma by the germinal epithelium, which leave no doubt that they are acquired conditions, due to age changes and to inflammatory changes. Inflammation of the pelvic peritoneum giving rise to chronic perioöphoritis, is a fruitful source of cyst formation. Clinical experience teaches, I think, that new growths are not commonly associated with, nor secondary to, inflammatory changes. These inflammatory cysts usually subside with the subsidence of the inflammation. One can readily conceive that inflammation about an ovary containing many fœtal remnants may act as the stimulus that starts these into activity. In such cases the inflammation would therefore be merely an accidental association.

Tumours arising from Ovules: The Dermoids and Teratomata.

This constitutes perhaps the most interesting chapter of all those dealing with the subject of tumour-formations in the ovary, and of late so much has been added to our knowledge concerning their origin, that to-day there is but little doubt that the ovules by a system of parthenogenesis are responsible for the presence of dermoids and teratomata. The evolution of this idea is interesting and facts that really bear upon the subject are indeed of but very recent date. Previous to this the whole subject lay in the domain of theory, and there are no subjects more productive of controversy than just such.

The earliest theory held that teratomata and dermoids were of the nature of mixed tumours, arising from fœtal rests of already differentiated cells in the ovary. It also held that these were not normally developed in the ovary, but were due to a misplacement from other organs. Of late, this theory was again brought to light by Bandler, who contended that not only was this theory well founded, but that the pronephros and the Wolffian body were responsible for these inclusions. In the light of our present knowledge, we know that not only the theory is wrong, but also that there was a misinterpretation of the facts from which the theory was developed.

Later Wilms pointed out, after a careful examination of dermoids and teratomata, that the former were not the simple mixed tumours they were generally held to be, but that they were mixed in the sense that they might contain structures developed from all the three primitive layers of the fœtus; that the ectodermic layer or ectoblast usually was so predominantly developed as to mask the other two, whereas in teratomata, it was usually the mesoblast which gained the ascendancy. In other words, both these tumour types were really of only one kind, for they both contained products of the three fœtal germinal layers, namely, the ectoblast, mesoblast and endoblast. The difference, therefore, was one of relative quantities of these and not of quality.

But of late, as if stimulated by the warmth of the discussion between these two contending camps, the subject of parthenogenesis has attracted to it a great deal of earnest work. Within the last twelve years we find Delage and Bataillon in France, and Loeb, Wolfsohn, Morgan and Weir in America, busy over this subject, and names of later workers are those of Wedekind, Daudin, Arbacia and Kostanechi. The sum and substance of their work has led to a great deal of weight being added to Pfannenstiel's theory. Through the experimental work of these authorities, it has been established beyond a doubt that parthenogenesis can be provoked experimentally, in echinoderms, worms, molluscs, arthropoda, fish and amphibians. Natural parthenogenesis has also been observed in mammals by Bischoff and Hensen, in batrachians by Bischoff, Leuckart, Born, and

Dehner, and in fish, worms, arthropoda, echinoderms and molluscs by a number of authors.

Lecaillon, who has done a great deal of work upon this subject, states: "We can consider as proved beyond all possibility of doubt that parthenogenesis really takes place naturally in many animals that are of widely different species. Parthenogenesis is really evoked by this fact, and this fact alone, that the unfertilized ovum is endowed with the ability and property to evolve along the lines of embryonic development of segmentation and differentiation, and not because the egg has encountered a special stimulus and special surroundings. This is proved by the fact that in birds the segmentation of non-impregnated ovum takes place as the egg passes along the oviduct. Such segmentation therefore takes place under the same circumstances and surroundings as it does with the impregnated ovum which segments normally.

Pfannenstiel has stated that no dermoid nor teratoma has been found sufficiently early to enable an opinion to be expressed as to its origin. That was reserved for Loeb.

One year later Loeb found that parthenogenesis is a common occurrence in the guinea-pig, and that the ovum is the starting point of this development. He found that parthenogenesis occurs in about 10 per cent. of guinea-pigs before they are 6 months old. Later, the condition is much less frequent. When describing one of these cases he writes: "We see in each case a chorionic vesicle with trophoblast and plasmodia and syncytia penetrating into the neighbouring tissue. There is also a structure present which is probably to be interpreted as the neural tube. As this type of growth occurs in the cortex of the ovary where follicles are normally seen, and are found within follicle-like cavities, they can be derived only from ova developing parthenogenetically. Fertilization can be excluded, as the life history of some of these animals is known and precludes such interpretation." It is very probable that parthenogenetic change sets in soon after ovulation; the altered conditions in the ovaries at that time (variations in blood pressure, in intrafollicular pressure, changes in oxygen supply) provide the necessary stimulus.

The later stages of these developing embryos bear some resemblance to chorio-epitheliomata. These observations demonstrate that chorio-epitheliomata and teratoid tumours that are found in the ovaries are not derived from misplaced blastomeres, as Bonnet and Marchand believed, but that the older view is correct according to which at least the teratoid tumours of the ovary are derived from parthenogenetically developed ova. Loeb might have gone one step further and included the dermoids, for histogenetically they are the same as shown above.

As for those extra-genital cases, there seems as yet no adequate scientific explanation forthcoming. Pfannenstiel maintains that those of the trunk and peritoneal cavity have the same origin as those of the ovary. The assumption is that they arise from ectopic ovarian tissue, which we know is found not only throughout the pelvis, but throughout the abdominal cavity, and it is well known that the seat of the vast majority of these extra-genital dermoids and teratomata are localized in the pelvis. Nagel, Minot and Ribbert have found wandering ova throughout the genito-abdominal cavity. "Grant such a wandering of ova," states Pfannenstiel, "then the propagation of derivative tumours allows of a very ready and easy explanation."

Do pseudomucinous cysts arise from teratomata and therefore from the ova? The frequent association of this type of cyst with the teratomata is more than accidental. Yet we are struck with the frequency with which the dermoids are surrounded by other cysts whose origin is known to be from the germinal epithelium of the ovary. As stated above, Landau, Hanau, Ribbert and Askanazy contend that the pseudomucinous cyst, whether alone or in association with dermoids, is part and parcel of the dermoid and the expression of the development of the intestinal portion of the teratoma or dermoid, just as the teeth, brain and jaw, and hair are the expression of its cephalic portion.

If we look into the question more closely, we find that not only do pseudomucinous cysts surround dermoids, but the incidence of simple serous cysts in association with dermoids is more than a casual association. It would seem that dermoids by their irritation and growth stimulate the surrounding tissues to activity. We have seen how the vast majority of human ovaries contain all the necessary epithelial rests or late invasions for the development of cysts and tumours. We have seen how these rests or invasions, derived as they are from the germinal epithelium, possess the wonderful metaplastic properties of this epithelium. We have even seen how in certain animals it can so develop as to produce ova and pseudo-ova, none of which, however, perhaps ever reach maturity, and lastly I have shown how the germinal epithelium can and does transform itself into mucous cells that have all the characters of the lining cells of the pseudomucinous cyst.

I have found several of my cases of dermoids with mucin mixed with the sebaceous material. Macroscopic section always shows in corroboration of the microscopic description that this is due not to a portion of the original wall of the dermoid being a mucous surface as you would expect to find if the pseudomucinous cyst were part and parcel of the dermoid, but in each case it was quite apparent that the surface covered with mucous cells had once been a cyst separate and apart, and had later been incorporated. This fact taken with the many stated in this chapter cannot but lead, I think, to the conviction that pseudomucinous cysts are not only ovulogenic but are also of germ-epithelial origin.

The Interstitial Cells of the Ovary.

Upon the subject of tumours arising from this tissue, nothing so far has appeared in the literature. Inasmuch as this monograph deals with epithelial tumours of the ovary, and since it has been shown that the interstitial cells of the ovary are of epithelial origin, this tissue must be taken into consideration as a possible factor in the production of epithelial tumours.

It is singular that in many of the so-called peritheliomata the tissues change almost imperceptibly into carcinomata on the one hand and into alveolar sarcomata on the other hand. Particularly is this true of peritheliomata of the mucous membrane of the intestine. More especially are such tumour growths found in the appendix vermiformis. I have seen several such in which it was quite impossible to state whether the new growth was of the nature of a carcinoma, or of a perithelioma or of a sarcoma, for all three types existed in the same specimen. There is a striking fact in connexion with the ovary. In our series of malignant diseases of the ovaries at the Royal Victoria Hospital—a series extending over ten years—peritheliomata are eight times more frequent than are the sarcomata. In fact sarcoma of the ovary is in my experience a rare tumour of the ovary.

In view of these facts, I think the theory quite justified, that inasmuch as peritheliomata occupy morphologically a position midway between carcinomata and sarcomata, their relative frequency in the ovary might readily be explained by assuming that they arise from the interstitial cells which occupy a position morphologically between the epithelial and connective-tissue cells. The incidence of peritheliomata of the ovary in the ratio of eight to one of sarcoma has something more in it than will be accounted for by the law of accidental occurrence.

Such a preponderance of peritheliomata in the ovary is found in no other organ in the human body. In fact the ratio is usually eight sarcomata to one perithelioma. To me it is quite doubtful whether such a cell as the interstitial cell could completely abdicate its primitive characteristics and so deny its origin as to develop into a sarcoma. We must not look upon the interstitial cell as being of a nature of a connective tissue cell. It has a much more exalted function. We have seen how periodically when in the neighbourhood of maturing Graafian follicles it can awaken into activity and assume secretory functions and serve very materially in the maintenance of equilibrium in the internal economy. Its position, morphologically speaking, between the connective tissue and the epithelial structures makes it pre-eminently the tissue responsible for the great frequency of perithelioma in the ovary.

BIBLIOGRAPHY.

Askanazy. *Bibl. Med. Abt. C.*, Stuttgart, 1905, Heft 19.
Balfour. *Quart. Journ. Microsc. Soc.*, 1878, xviii, pp. 383-438.
Bandler. *Arch. f. Gyn.*, 1900, lx, p. 377, lxi, p. 445 ; *Monatschr. f. Geb. u. Gyn.*, xiv.
Bataillon. *Arch de Zool. exp. et gen.*, Par., 1910-11, xlvi, p. 103 ; *Compt. rend. Acad. des Sci.*, Par., 1911, clii, pp. 928, 1120, 1271.
von Beneden. *Arch. de Biol.*, 1880, i, p. 475.
Bischoff. *Thèse de Par.*, 1843.
Bonnet. *Ergeb. d. Anat. u. Entwick.*, 1900, xiii ; *Monatschr. f. Geb. u. Gyn.*, xi.
Born. *Arch. f. Anat. u. Phys.*, 1874, pp. 118-151.
Bornhaupt. "Untersuchungen über die Entw. des urogenit. Systems beim Hühnchen," Diss. inaug. (Dorpat.), Riga, 1867.
Borsenkow. *Zeitschr. f. wissensch. Zool.*, lviii.
Braun, Max. *Arb. Zool. Zoot. Inst.*, Würzburg., 1877, iv.
Bulius. Hegar's *Beitr. z. Geb. u. Gyn.*, 1898, i, p. 138 ; *Zeitschr. f. Geb. u. Gyn.*, 1888, xv, p. 365.
Chiarugi. *Inst. Anatomico di Siena*, 1885.
Coert. "Proefschrift," Leiden, 1898.
Daudin. *Bull. Scient. de la France et de la Belgique*, Par., 1909, xliii, pp. 297-372.
Delage. *Compt. rend. Acad. des Sci.*, Par., 1909, cxlix, p. 890 ; *Arch. d. Zool. exper. et gen* , Par. (1907) 1908, 4 sér., vii, pp. 445-508.
Diepgen. Hegar's *Beitr. z. Geb. u. Gyn.*, 1904, viii.
Egli. "Beitrage zur Anatomie und Entwickelung der Geschlechtsorgane," Diss. Inaug., Zurich, 1876.
Fischel. *Verhandl. d. deutsch. Gesellsch. f. Path.*. Berl., 1903 ; *Arch. f. Gyn.*, 1879, xv, p. 198.
Flaischlen. *Zeitschr. f. Geb. u. Gyn.*, 1881, vi, p. 126.
Flemming. *Arch. f. mikros. Anat.*, 1887, xxix, pp. 389-463.
Foulis. *Quart. Journ. Micros. Sci.*, 1876, xvi, p. 190.
Franqué. *Zeitschr. f. Geb. u. Gyn.*, 1899, xxxix.
Friedmann. *Arch. f. mikros. Anat.*, 1898, lii.
Frommel. *Zeitschr. f. Geb. u. Gyn.*, 1890, xix.
Genhard. "Path. Anat. d. Weibl. Sexualorgane," Leipz., 1899.
Goodall. "Involution of the Puerperal Uterus," Montreal, 1910.
Gottschalk. *Arch. f. Gyn.*, 1899, lix, p. 676 ; *Verhandl. d. Deutsch. Gesellsch. f. Gyn.*, Giesen, 1901 ; *Zeitschr. f. Geb. u. Gyn.*, lviii, H. 2.

Hartz. *Arch. f. mikros. Anat.*, 1883, xxii, pp. 374-407.

Heinrichs. Martin's text-book, "Krankheiten der Eierstöcke."

Hensen. *Zeitschr. f. Anat. u. Entwick.*, 1876, i.

Hertwig, O. "Ueber den Befruchtungs und Theilungsvorgang des Thierischen Eies," &c., Jena, 1887; "Experimentelle Studien am Thierischen Eie," &c., Jena, 1890.

His. *Arch. f. mikros. Anat.*, 1865, i, p. 151.

Hofmeier. "Handbuch der Krankheiten der weiblichen Geschlechtsorgane," Leipz., 1898.

Janosik. *Arch. f. mikros. Anat.*, 1896, xlviii, p. 169; *Sitz. Berl. Acad. Wien*, 3 Abth. 1885, xci; 1887, xcvi: 1890, xcix.

v. Kahlden. *Beitr. z. Path. Anat. u. allg. Path.*, 1902, xxxi, p. 1; *Zentralbl. f. path. Anat.*, iv.

v. Klebs. *Virchow's Archiv*, 1863, xxviii, pp. 301-336.

Klob. "Path. Anat. der weiblichen Geschlectsorgane," Wien, 1864.

Kölliker. "Entwickelungsgeschichte," 2te Aufl., 1879; *Verhandl. d. anat. Gesellsch.*, Kiel, 1898; *Würzburger Sitzungsber.*, Juni, 1898.

Kossmann. *Monatschr. f. Geb. u. Gyn.*, 1895, i, p. 97; *Verhandl. d. Deutsch. Gesellsch. f. Chir.*, 1901.

Kostanecki. *Bull. Internat. Acad. d. Sci. d. Cracovie*, 1909; *Arch. f. mikros. Anat.*, Bonn, 1908, lxxii, p. 327.

Koster. *Arch. néerl. de Sci. exactes*, 1869, iv, p. 363.

Kroemer. *Arch. f. Gyn.*, 1899, lvii, pp. 322-423; Veit's "Handbuch der Gynäkologie," 1908.

Landau. *Berl. klin. Wochenschr.*, 1904, xli, p. 162.

Lane-Claypon. *Journ. Obst. and Gyn. of Brit. Emp.*, 1907, xi, pp. 205-214; *Brit. Med. Journ.*, 1905, ii, p. 18.

Laulanie. *Compt. rend. de la Soc. de Biol.*, 1886, 8 sér., iii, pp. 87, 132, 280; 1888, 8 sér., v, p. 4.

Lecaillon. *Arch. d'Anat. Micros.*, Par., 1910-11, pp. 511-638; *Compt. rend. de la Soc. de Biol.*, 1910, lxix, pp. 123, 125.

Leopold. *Beitr. z. Geb. u. Gyn.*, 1903, ii; *Arch. f. Gyn.*, 1874, vi, pp. 189-278.

Limnell. *Arch. f. Gyn.*, 1901, lxiii, pp. 547-605.

Limon. *Thèse de Nancy*, 1901; *Arch. de l'Anat. Micros.*, 1902, v.

Lockhart. *Canada Lancet*, Toronto, 1909, xlii.

Loeb, L. *Zeitschr. f. Krebsforsch.*, Berl., 1911, xi; *Journ. Morphol.*, Philad., 1911, xxii; *Journ. Amer. Med. Assoc.*, Chicago, 1911, lvi, p. 1326.

McIlroy. *Trans. Glasgow Obst. and Gyn. Soc.*, 1911, p. 57; *Brit. Med. Journ.*, 1909, ii; *Proc. Roy. Soc. Med.*, 1910-11, iv (Sect. Obst. and Gyn.), p. 226.

MacLeod. *Arch. de Biol.*, 1880, i, pp. 241-278.

Malassey et De Sinety. *Arch. de Physiol.*, 1879, vi, p. 624.

Martin. "Die Krankheiten der Eierstöcke," 1899.

Martin and Jung. "Handbuch der Gynäkologie," Berl., 1906.

Marchand. *Verhandl. d. Deutsch. path. Gesellsch.*, 1899; *Zeitschr. f. Geb. u. Gyn.*, 1895, xxxii, pp. 405-472; 1898, xxxix, pp. 173-258; *Monatschr. f. Geb. u. Gyn.*, 1895, i, p. 419.

Mathieu. *These de Nancy*, 1898.

Mayweg. Inaugural Dissertation, Bonn, 1868.

Mériel. *Ann. de Gyn. et d'Obstet.*, Par., 1910, vii.

Meyer. *Ergeb. d. allg. Path. u. path., Anat.*, IX Jahrg., 1903; *Zentralbl. f. Gyn.*, 1907, xxxi, p. 203; *Zeitschr. f. Geb. u. Gyn.*, 1905, lvi, p. 210.

Mihalkovics. *Internat. Monatschr. f. Anat. u. Histol.*, 1885, ii.

Minot. *Anatomischer Anzeiger*, 1894, ix.

Morgan. *Science*, New York and Lancaster, Pa., 1909, xxix; *Anat. Record*, Philad., 1909, iii.

Nagel. *Arch. f. mikros. Anat.*, 1888, xxxi, and 1895, xlvi; *Arch. f. Gyn.*, 1887, xxxi, pp. 327-362, 1894, xlv, p. 453, and 1905; *Anatomischer Anzeiger*, 1889 ("Die Weibl. Geschlectsorgane," Handbuch der Anat. des Menschen, von Bardeleben, 1896).

Neumann. *Arch. f. Gyn.*, 1899, lviii, p. 594; *Monatschr. f. Geb. u. Gyn.*, 1896, iii, p. 387; *Virchow's Archiv*, 1886, civ.

Ohlshausen. "Die Krankheiten der Ovarien," Stuttgart, 1886; *Deutsch. med. Wochenschr.*, 1902.

Paladino. "Ulteriori ricerche sulla distruzione del parenchima ovarico nei mammiferi," Napoli, 1887.

Pfannenstiel. In Veits' "Handbuch der Gynäkologie," 1908; *Verhandl. d. Deutsch. Gesellsch. f. Gyn.*, xi.

Pflüger. "Die Eierstocke der Säugetiere und des Menschens," Leipz., 1863.

Pick. *Berl. klin. Wochenschr.*, 1900 and 1904; *Zentralbl. f. Gyn.*, 1903, xxvii, pp. 1033-1110.

Plato. *Arch. f. mikros. Anat.*, 1897, l, pp. 640-85.

Poirier et Charpy. "Traité de l'Anat. humaine," 1910, v, fasc. 1er.

Pozzi et Beaussenat. *Rev. de Gyn. et de Chir. Abdom.*, 1897, i, p. 245.

Rabl. *Wien. Gyn. Gesellsch.*, *Zentralbl. f. Gyn.*, 1899, xvii (Anat. Heft 1 Abth.), 1898, ii.

Regaud et Policard. *Compt. rend. de la Soc. des Anatomistes*, 1901.

Regaud et Senat. *Thèse de Lyon*, 1900.

Ribbert. "Geschwulstlehre," 1904; *Deutsch. med. Wochenschr.*, 1905.

Rielander. "Das Paroöphoron," Hab.-Schr., Marburg, 1904.

Rindfleisch. "Lehrbuch der pathol. Gewebelehre."

Rokitansky. *Allgem. Wien. med. Zeitung. Vierten Jahrg.*, 1859.

Romiti. *Arch. f. mikros. Anat.*, 1874, x, p. 200.

Rouget. *Journ. de la Physiologie*, 1858, pp. 320, 479, 735; *Compt. rend. Acad. des Sci.*, 1879, lxxxviii, p. 602.

Roux. *Virchow's Archiv*, 1888, cxiv, pp. 113, 246.

Schickele. *Beitr. f. Geb. u. Gyn.*, 1911, xvi, p. 130; *Verhandl. d. Deutsch. Gesellsch. f. Gyn.*, 1905.

Schottländer. *Arch. f. mikros. Anat.*, 1888, xxxi, pp. 426-82; *Monatschr. f. Geb. u. Gyn.*, 1905, xxi, p. 622.

Schrön. *Zeitschr. f. Wissensch. Zool.*, 1862, xii.

Schulin. *Arch. f. Mikros. Anat.*, 1881, xix, pp. 442-512.

Seitz. *Arch. f. Gyn.*, lxxvii, Heft 2; *Zentralbl. f. Gyn.*, 1905, xxix, pp. 257, 578.

Semon. *Zeitschr. f. Naturwissensch.*, Jena, 1887, xxi.

Slavianski. *Bull. Soc. Anat.*, Par., 1873, xlviii, p. 844.

Sobotta. *Arch. f. mikros. Anat.*, 1896, xlvii (Anat. Heft, 1897, viii).

Steffeck. *Zeitschr. f. Geb. u. Gyn.*, 1890, xix.

Stoeckel. *Fritsch's Festchrift*, Leipz., 1902; *Arch. f. mikros. Anat. u. Entwick.*, 1898, liii, pp. 357-84.

Stratz. "Der Geschlechstreife Eierstock," 1898; *Zeitschr. f. Geb. u. Gyn.*, 1893, xxvi, p. 1.

Tourneux. *Journ. de l'Anatomie*, 1888, xxiv; *Journ. de l'Anatomie et de la Physiol.*, 1879, xv, p. 306.

Valentin. *Muller's Archiv*, 1838, p. 526.

Veit. "Handbuch der Gynäk.," 1910, iv, Fasc. 1.

v. Velits. *Arch. f. Gyn.*, 1906, lxxix, pp. 523-604; *Zeitschr. f. Geb. u. Gyn.*, 1889, xvii, p. 232.

Virchow. *Verhandl. d. Gesellsch. f. Geb.*, Berl., 1849.

Waldeyer. "Eierstock und Ei." Leipz., 1870; *Arch. f. Gyn.*, 1870, i, p. 252.

Walthard. *Zeitschr. f. Gyn.*, 1903, xlix, pp. 233-329.

Weir, J. *Dietetic and Hyg. Gaz.*, New York, 1905, xxi.

Weldon. *Quart. Journ. of Micros. Sci.*, 1884, xxiv, p. 171.

Wendeler. Martin's text-book, "Die Krankheiten der Eierstöcke," 1899; *Arch. f. mikros. Anat.*, 1895, xlv, p. 167.

Williams. *Johns Hopkins Hosp. Bull.*, 1891, No. xviii.

Wilms. "Die Mischgeschwülste," Leipz. 1902; "Ueber Dermoide und Terat.," *Deutsch. Arch. f. klin. Med.*, 1895, lv, pp. 289-392; *Arch. f. Gyn.*, 1900, lxi, p. 203; *Monatschr. f. Geb. u. Gyn.*, 1899, ix, p. 285; Martin's text-book, "Die Krankheiten der Eierstocke und Nebeneierstocke," 1899.

Wilson, E. *Columb. Univ. Biol. Ser.*, New York, 1900, iv.

Wilson, T. *Birmingham Med. Review*, 1906, lviii, p. 678.

v. Winiwarter. *Anatomischer Anzeiger*, 1908 and 1912; *Arch. de Biol.*, Liège et Par., 1900, xvii, p. 178, and 1910-11, xxv, pp. 169-267.

Wolfsohn. *Bull. Biol. Woods Holl.*, Mass., 1907, xiii.

DISCUSSION.

Dr. Herbert Williamson: The paper we have listened to to-night is one of great importance. We are to be congratulated because its first publication is at a meeting of our Society, and I am sure we are all grateful for the gift Dr. Goodall has sent us from Canada. I desire to express my admiration of both the patient industry with which he has pursued his task and the importance of the results he has achieved. These results have been obtained in the main by following two methods of research: (1) The constant checking of conclusions by the study of ovaries of mammals lower in the scale; (2) by the study of serial sections. These two methods of investigation have enabled Dr. Goodall to throw light upon what was previously dark and obscure. To my mind, if Dr. Goodall has rightly interpreted what he has seen, the paper establishes two theses of great importance: (*a*) That structures previously regarded as mesonephric are, in reality, derivatives of the germinal epithelium, and (*b*) that the common epithelial tumours of the ovary must all be regarded as oöphoronic and none of them as Wolffian. I regret very much that we have no drawings nor photographs of the sections upon which such important conclusions are based, and I hope that there will be some fund available to enable Dr. Goodall to remedy this omission. If correct deductions have been drawn, the paper gives the *coup de grâce* to the Wolffian theory, a theory which has been dying slowly for years. Keith has already shown that what we regarded as parovarian cysts are in reality cysts of the fimbria ovarica and are derived from prolongations of the rete ovarii into their structure; so that the familiar large broad ligament cysts are in reality oöphoronic. It is curious that no mention of Keith's work is to be found in the paper. I regard as of special importance Dr. Goodall's observations upon the downgrowths of germinal epithelium which occur in adult life in bitches and other animals; downgrowths which are glandular in appearance but never give rise to true ova. Similar downgrowths occur in the human ovary; they were described by Waldeyer in 1870, and he taught that most ovarian tumours had their origin in these structures. My attention was drawn to the subject fifteen years ago by a paper by Walthard, and in 1906 I published photographs of sections of ovaries, normal in their naked-eye characters, which in my opinion clearly demonstrated the origin of pseudo-mucinous cystadenomata from the germinal epithelium. I am glad to find the author declines to accept the theory of a teratomatous origin for cyst adenomata. When we consider the potentialities of the germinal epithelium; when we see it assume many varieties of form and even become ciliated; when we have traced every stage between it and the typical cystadenoma, any such hypothesis is totally unnecessary. We sometimes find true intestinal epithelium in a teratoma, but we find intestinal villi and crypts on a true muscle wall. I have stained a number of cystadenomata by van Gieson's stain, but I have failed to find muscle in the walls of the loculi. We not infrequently find teratomatous loculi in a cystadenoma, but they originate as separate tumours; a specimen in the museum at St. Bartholomew's Hospital shows two such tumours separated by $\frac{3}{4}$ in. of ovarian stroma. The mode of origin of ovarian teratomata is still in doubt, and I gather that Dr. Goodall's work has thrown no light upon the problem. Personally I see no difficulty in accepting the theory of parthenogenesis, and Loeb's observations upon guinea-pigs lend support to this view. I am interested in Dr. Goodall's remarks upon lutein cells, his work shows that, whatever be their origin, they do not arise from the membrana granulosa; I do not think he definitely stated it anywhere, but from one or two remarks he seems to regard them as arising from interstitial cells. He is not quite correct in stating that so far nothing has appeared in the literature upon the origin of tumours from the interstitial cells; it has been suggested that certain rare malignant tumours composed of lutein-like tissue arise from these cells. Dr. Goodall's suggestions concerning peritheliomata are interesting, but they appear to be merely speculations and not based upon anything seen in his sections.

The PRESIDENT: We are all indebted to Dr. Lockyer for reproducing the gist of Dr. Goodall's paper in a very successful way. I have been unable to obtain anything written by Loeb on the parthenogenesis in guinea-pigs which has been mentioned. Can Dr. Lockyer give further information concerning the nature and degree of completeness of this phenomenon? Parthenogenesis is a well known mode of normal reproduction. According to Loeb, the females of some aphides can propagate their species for four years, and probably indefinitely, without the intervention of the male. Loeb and others have with great patience and ingenuity discovered conditions in which a development of the unfertilized eggs of the sea urchin and starfish is induced with certainty as far as the pluteus stage exactly as if they had been fertilized. In natural parthenogenesis a normal individual is produced, and, so far as I know, in all artificially-induced parthenogeneses a development, partial or complete, of a perfect individual is effected. It seems therefore questionable whether a dermoid tumour, with its heterogeneous mixture of many sorts of tissues altogether wanting in any fixed relationship to each other, and without the least resemblance to the individual from which it grows, can accurately be described as an example of parthenogenesis. Such a tumour may certainly be described as a parthenogenesis which has gone wrong, but this is true of all new growths—simple, malignant, and teratomatous alike. All arise from the cells of the body by a process which is not stimulated by any sexual influence, but the suggestion that any of them is an example of parthenogenesis leaves out of account altogether the cause of the erratic growth, which is the crux of the whole question of tumour development.

Mr. GORDON LEY: I cannot understand if, as Dr. Goodall says, the ovarian stroma grows out along the surface of the Wolffian body, how it is that the ovary becomes a pedunculated organ. The evolution of a mesentery is an upgrowth of tissue, and cannot result from downgrowth. I, myself, am of opinion from study of human embryos, that the hilum of the ovary is an upgrowth of connective tissue, not a downward projection of germinal cells. Pathology appears to me firmly to establish the fact that, whatever their origin, the tubules found in the ovary and tubules found in the broad ligament are of the same nature, and as it is apparently admitted by everyone that the tubules in the broad ligament are Wolffian, one must assume a like origin for similar pathological conditions in the ovary.

Dr. CUTHBERT LOCKYER: It has been a great pleasure as well as a privilege to present to this Section an abstract of Dr. Goodall's paper on "The Origin of Epithelial Tumours of the Ovary." It would have been a great pity if such an important and praiseworthy piece of work, performed by a British subject, had only seen the light of day through the medium of a foreign press. For my own part, I wish to state that I consider Dr. Goodall's work is of very high importance and will have a far-reaching effect upon our views relating to epithelial new growths of the ovary. Dr. Goodall has shown that a clear conception of these growths cannot be obtained without a previous knowledge of the comparative embryology of the ovaries. His painstaking study of 15,000 serial sections taken from 127 individuals forms the most important part of his work, and the details of this are set forth in the first part of his paper. Owing to the extensive atrophy of epithelial structures which occurs in the evolution of the adult human ovary, we can obtain no knowledge from the study of these organs alone of the nature of the medullary-ray system nor of the gland-like structure known as the rete ovarii. The only information hitherto at our disposal, here in London, on the latter structure has been that provided by Professor Keith, who taught us the proper conception of rete testis cysts. The existence of an epithelial tubular system running from the germ-epithelium on the surface of the cortex, through the ovary and into the mesosalpinx, to join up with the Wolffian duct, is fresh knowledge. The statement that this epithelial tubular system is ovulogenetic so long as it is enclosed in the ovary and can therefore be considered to be oöphoronic, is also an addition to our knowledge. The exclusion of the epoöphoron from taking any share in providing the ovary and its hilum with tubules is also new. Hilum-cysts—the serous papilliferous cysts—are no longer to be considered as Wolffian structures, but as oöphoronic. These are only a few of the conclusions which we must draw from Dr. Goodall's work. The gist of his teaching seems to be that, whilst all epithelial ovarian tumours are derived from germ epithelium, only such parts of the latter as should normally atrophy, and for some reason do not, are capable of tumour formation. Those parts which persist and perform a useful function do not lead to the formation of new growths; e.g., take the Graafian follicle. Goodall will not allow that a large hydrops folliculi is a new growth, the granulosa does not line a true cyst. The common multilocular cystadenoma never arises from a follicle. Only the ovum itself can produce a neoplasm and that by parthenogenesis, which results in an embryoma. In one particular alone do I find Dr. Goodall less dogmatic in connexion with the follicles. He speaks of the lutein cystoma found in association with a vesicular mole as a "true cyst."

I was relieved to find that the author is inclined to put his veto upon the theory which claims that a multilocular pseudomucinous cystadenoma is the expression of the intestinal portion of an embryoma. He has proved to his own satisfaction that, when a simple teratomatous cyst is found within a multilocular pseudomucinous cyst, it is the result of the fusion of the two. My own investigations support this finding. That ovarian perithelioma is derived solely from the interstitial cells of the ovary is a pious opinion which will need a lot of proving. I have no doubt that the Fellows of this Section are grateful to Dr. Goodall for allowing me to read to them an abstract of his most valuable work.

The PRESIDENT suggested that the thanks of the Section should be conveyed to Dr. Goodall by Dr. Lockyer for his valuable contribution, and this was agreed to.

Primary and Secondary Carcinoma of the Ovary: A Statistical Record from the Pathological Institute of the London Hospital.

By GORDON LEY, F.R.C.S.

OUR present knowledge of carcinoma of the ovary is so scanty and incomplete in regard to its origin, its frequency as a primary tumour, and its method of metastasis, when primary, that I feel that this paper and further work on which I am engaged in conjunction with Dr. Russell Andrews are justified. I am producing this paper first for the reason that it is composed of material gleaned from the post-mortem room and therefore entirely above suspicion. Every case has been examined by skilled pathologists and the site of the primary growth definitely settled.

Where doubt has arisen as to whether an ovarian or visceral growth has been the primary, the microscopical evidence has been given precedence over the macroscopical. This doubt has arisen in four of the cases: 179, 1909; 590, 1915; 381, 1916; and 309, 1918. In two of these cases the large bowel was the seat of the primary growth, in one the bile passages, and in one a bilateral pseudomucinous cyst. It will be noticed that in these four cases the carcinoma had arisen in columnar-celled, mucus-forming epithelium. The majority of the text-books and literature on the subject lay stress on this difficulty and state that carcinomata arising from bowel epithelium are stained by muci-carmine, whereas ovarian carcinomata are not. This statement is untrue. The epithelium of pseudomucinous cysts is stained deeply by muci-carmine, as is also that of carcinomata arising from pseudomucinous cysts (fig. 1).

The material from which this paper is composed is taken from the records of the Pathological Institute of the London Hospital. These records, since the year 1909, have been a model of accuracy under the directorship of Professor Hubert M. Turnbull. To him and his assistant directors, Dr. G. B. Bartlett and Dr. W. W. Woods, my thanks are due for kind assistance and for records without which it would have been impossible to produce this work. My best thanks are also due to Mr. J. R. Ford for his excellent drawings and paintings of microscopical preparations.

During the eleven years, 1909 to 1919 inclusive, 324 necropsies were performed on women affected with carcinoma. In the following Table (p. 97) these cases are arranged in groups according to the site of the primary growth. Each of these primary groups is subdivided into secondary groups, of cases which resemble each other in the absence of metastases or in the like distribution of metastases. In this table the term "organs" designates, for the sake of brevity, not only all the viscera but the bones. In 264 cases the ovaries were not the seat of carcinoma. These cases are set aside. In 60 cases the ovaries were affected. These form the subject matter of my paper. In 25 cases the ovaries were affected primarily. In 38 they were affected secondarily; in 3 of these latter the primary growth was in the opposite ovary.

PRIMARY OVARIAN CARCINOMA—25 CASES.

A satisfactory classification of the origin of these tumours is extremely difficult. Previously they have been divided into solid and cystic. This classification is unsatisfactory as primary solid tumours may become cystic from necrosis, and primarily cystic tumours may become solid by the extreme luxuriance of intra-cystic growth. It seems best to classify them according to their histogenetic origin into:—

(A) *Oöphoric.*—(1) Arising *de novo*; ? site. (2) Arising from pseudomucinous cysts. (3) Arising from teratomatous (dermoid) cysts.

(B) *Epoöphoric and Paroöphoric.*—(1) Arising from Wolffian relics in the ovary, hilum of ovary, or broad ligament.

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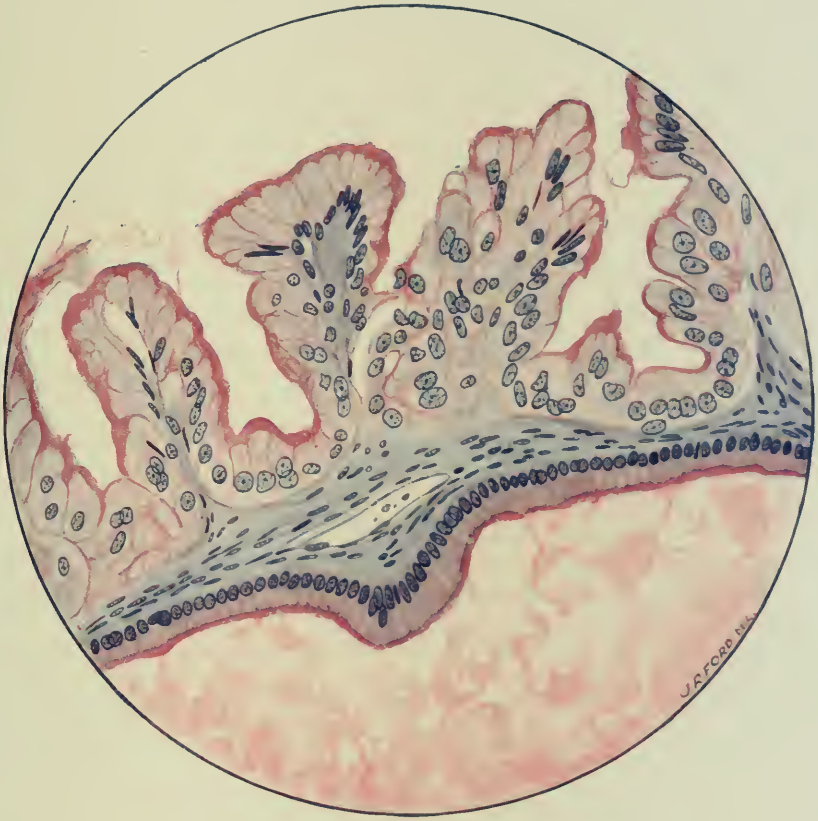

FIG. 1.

Precarcinomatous area in carcinomatous pseudomucinous cyst (muci-carmine).

GORDON LEY:
Primary and Secondary Carcinoma
of the Ovary.

	Colon and rectum	Extra-hepatic bile passages and caruncula major of Santorini	Gall bladder	Pancreas	Stomach	Ovary	Uterus	Breast	Œsophagus	Lung- and bronchi	Nose	Nasopharynx	Larynx	Tongue	Thyroid	Thymus	Pituitary gland	Vulva	Vagina	Small intestine	Omentum	Urinary bladder	Kidney	Suprarenal gland
Total number of cases ..	106	17	13	11	57	25	25	23	7	8	1	1	3	1	3	1	1	1	1	3	2	4	5	5
Cases without metastases	51	7	2	3	7	3	11	6	4	4	—	1	1	—	1	—	1	—	1	2	1	3	—	—
Cases with metastases in glands alone	14	3	—	—	8	—	9	5	2	1	—	—	2	1	—	—	—	1	—	—	—	1	—	—
Cases with metastases in peritoneum alone	4	—	1	1	6	4	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in organs alone	9	1	—	1	—	—	1	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	1	2
Cases with metastases in glands and peritoneum	4	—	1	1	12	4	1	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
Cases with metastases in glands and organs	3	2	4	—	6	1	—	6	1	3	—	—	—	—	1	1	—	—	—	—	—	—	2	1
Cases with metastases in peritoneum and organs	2	2	—	1	—	4	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in glands, peritoneum and organs	8	—	3	2	8	6	2	2	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—
Cases with metastases in ovaries alone	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries and glands	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries and peritoneum	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries and organs	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries, glands and peritoneum	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Cases with metastases in ovaries, glands and organs	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries, peritoneum and organs	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Cases with metastases in ovaries, glands, peritoneum and organs	5	2	1	2	8	1	1	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1

(A1) *Carcinomata arising de novo.*—With regard to this type, little can be said in the present paper. Macroscopically they are usually solid, medullary or scirrhous, tumours, with or without cystic degenerated areas, and are often bilateral, one tumour being usually larger than the other. They are of relatively infrequent occurrence. Thus no tumour of this type appears in this series. Several ovarian tumours removed by operation have been placed in this class in the records of the institute. I have myself obtained others at operation. A statement of the evidence which shows that these tumours are not secondary carcinomata is beyond the scope of this paper.

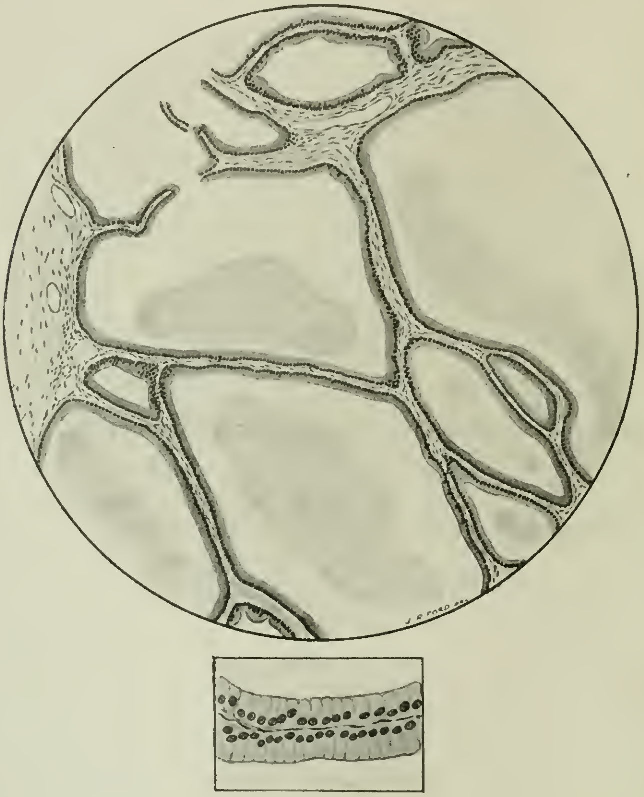

Fig. 2.

Simple multilocular pseudomucinous adenomatous cyst.

(A2) *Carcinomata arising from Pseudomucinous Cysts.*—Pseudomucinous cysts are of frequent occurrence. With regard to their origin, I am strongly of opinion that they are teratomatous. I believe that they represent the posterior end (hind gut) of the embryo just as the so-called "dermoid" represents essentially the cephalic end of the embryo. My reasons for this statement are: (i) the epithelium is typical of gut and resembles no epithelium found in the normal ovary. (ii) Loculi lined by skin containing hair, &c., are of not infrequent occurrence in these tumours. (iii) Unstriped muscle is not infrequent in their walls. (iv) Loculi lined by cells of this type are by no means infrequent in "dermoid" cysts. These cysts are multilocular as a rule. When benign, the loculi are lined by a single layer of columnar cells, the cells being

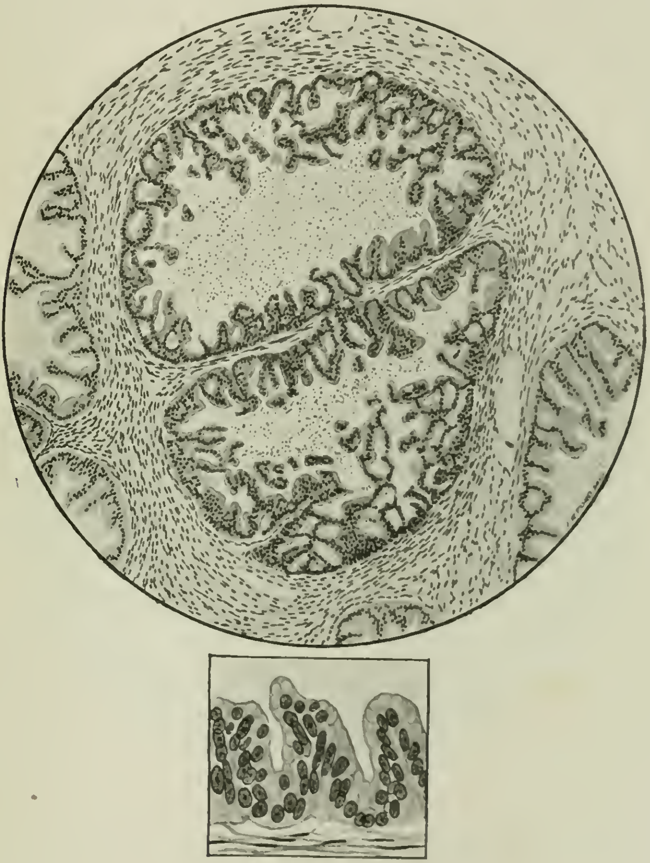

Fig. 3.

Multilocular pseudomucinous adenomatous cyst with cellular papillæ (pre-carcinomatous).

regular in size and shape, the nuclei being basal (fig. 2). The transition from a benign to a malignant, carcinomatous, state passes through a papilliferous stage in which microscopical villus-like or branching cellular papillæ project into the loculi (fig. 3). The cells covering these papillæ are atypical and their nuclei vary in level being by no means always basal. The final, carcinomatous, state is marked by solid or partly solid masses replacing parts or, more rarely, the whole cystic mass; by extreme papilliferous change; by distinctly atypical cells with nuclei arranged at all levels; and frequently by the loculi being filled by shed cells (fig. 4).

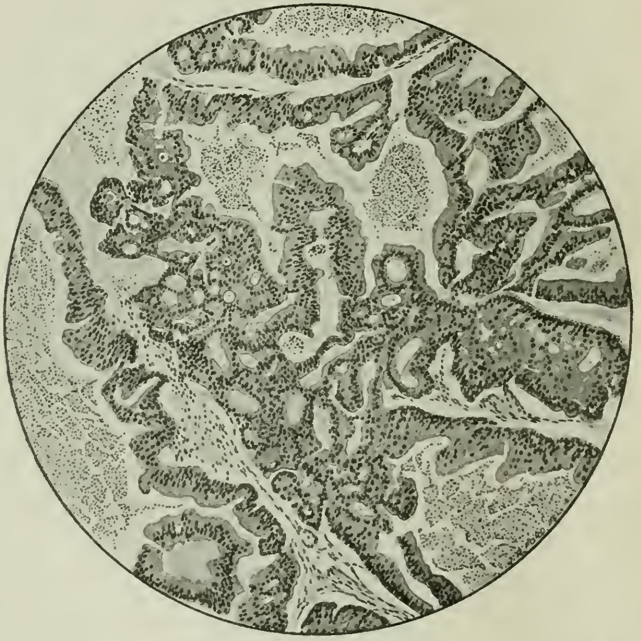

Fig. 4.

Tubular and papillary-cystic columnar-celled carcinoma of ovary (pseudomucinous adenomatous cyst).

(A3) *Carcinomata arising in Teratomatous Cysts.*—A squamous and polygonal-celled, horny carcinoma occasionally arises in the skin lining a "dermoid" cyst. These growths, many of which have been recorded, are comparatively rare. No case occurs in this series. One would expect to obtain similar malignant, teratogenous tumours from other constituents of the teratomatous cyst; but of such tumours I can obtain no record.

(B1) *Carcinomata arising in Wolffian Tubules.*—Cysts of the Wolffian tubules are of frequent occurrence. They are found in the ovary (fig. 5), generally in the hilum, and in the broad ligament (fig. 6). The tubules of the Wolffian duct system are lined by a single layer of narrow columnar cells with large central nuclei. When situated in the broad ligament they are surrounded by a relatively broad coat of unstriped muscle fibres. The cysts arising in these tubules are frequently bilateral, this feature becoming more frequent as the condition approaches towards carcinoma. The changes occurring in these cysts are three: Club-shaped papillary fibromata projecting into the cyst cavity (fig. 7). These are rarely large, usually less in size than a grain of wheat, hard and pearly white. Microscopically they are seen to consist of coarse fibrous tissue covered by a single layer of cells

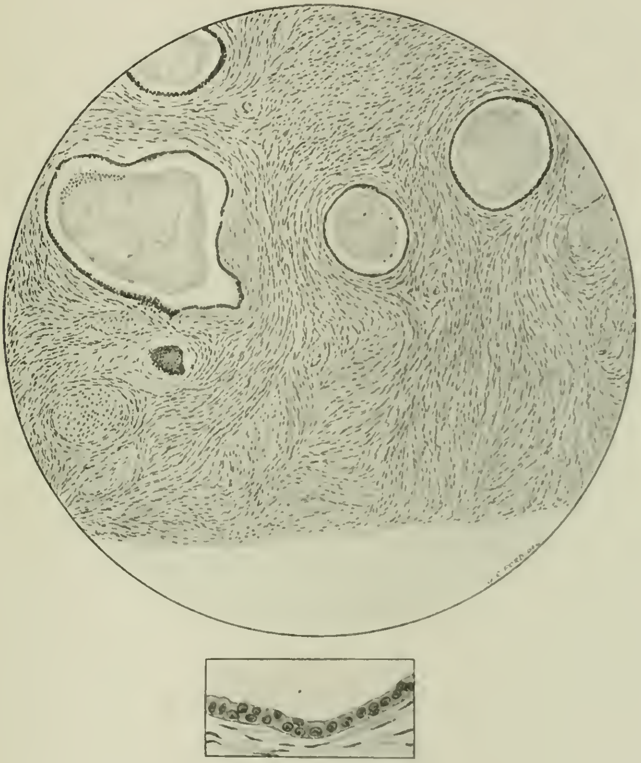

Fig. 5.

Wolffian tubules in cortex ovarii.

similar to those lining the cyst. Dendritic fibromata projecting into the cyst cavity (fig. 8). These are cauliflower-like, white or pink warts varying considerably in size, sometimes forming large villous masses. Microscopically they have a fibrous core and are covered by a single layer of cells similar to those lining the cyst. In most examples occasional atypical cells are seen, the condition gradually passing to the carcinomatous state (fig. 9) in which the papillæ are covered by one or many layers of atypical cells varying from columnar to polygonal. These cysts are frequently multiple; many tubules taking on the same change, so that a multilocular mass is formed. Not infrequently a papillary mass inside a small loculus grows so rapidly that the loculus bursts, the papillary mass then appearing to be an extracystic growth from an adjacent loculus. The proof of its having been originally

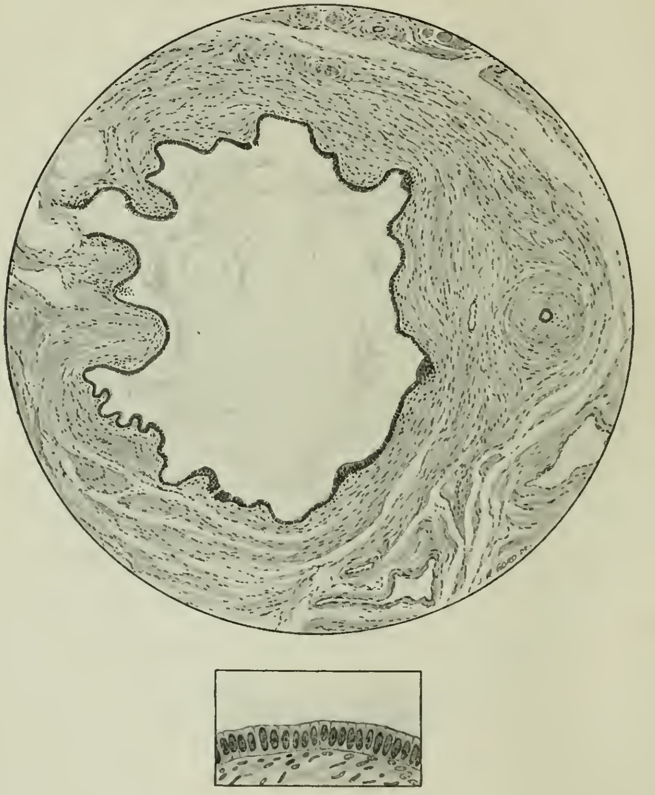

Fig. 6.

Wolffian tubule and cystic ovarian tubule in mesosalpinx.

intracystic lies in the finding of the shrunken wall of the loculus at the base of the papillary mass; this I have observed. These tumours with extracystic papillary processes are not of necessity carcinomatous. It is stated that benign metastases may arise in the peritoneum in association with these cysts; this condition I have not seen. When carcinomatous the tumour may form a cystic mass with intracystic, and, possibly, extracystic papillæ, or may form a solid mass, the papillary processes being so closely packed as to completely fill the loculi.

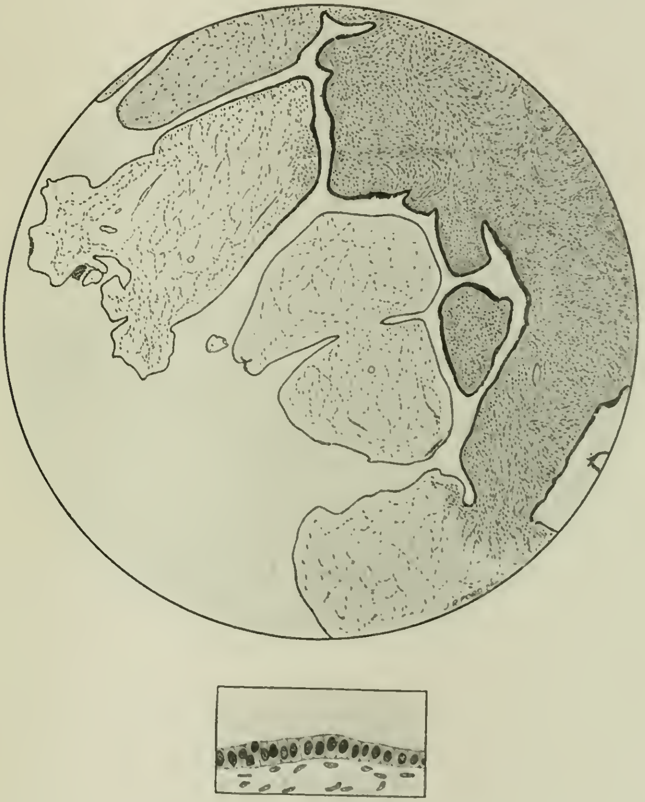

Fig. 7.

Wolffian cyst, with intracystic club-shaped papillæ.

In this series of 25 cases of primary ovarian carcinomata there were 16 of Wolffian origin, 9 of pseudomucinous cyst origin.

Wolffian Carcinomata.

Among the 16 Wolffian carcinomata, the average age incidence was 54, the oldest patient being 74 and the youngest 40. In 8 cases the tumour was unilateral and in 8 bilateral. One of the unilateral cases had a simple Wolffian cyst in the opposite ovary. In one case the opposite ovary contained a secondary nodule of growth, and in one there was carcinomatous infiltration of the surface of the opposite ovary.

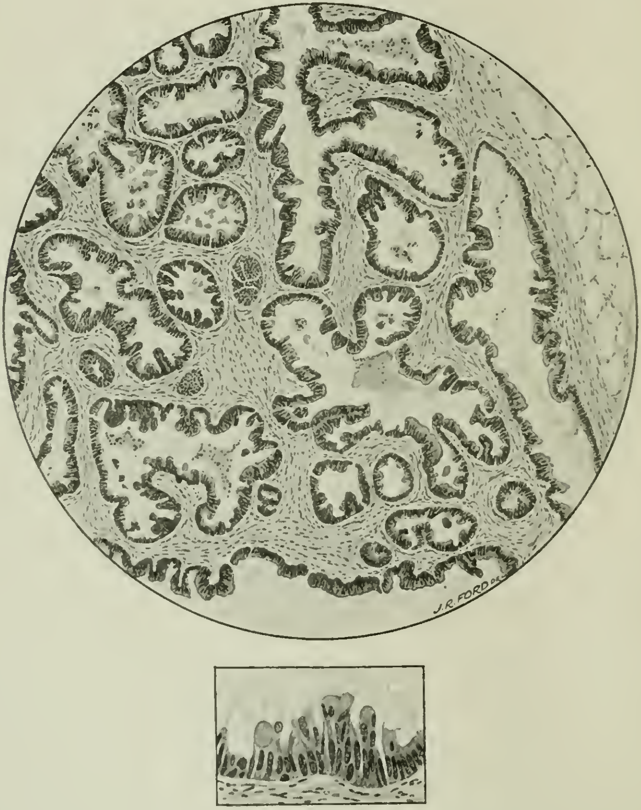

FIG. 8.

Wolffian cyst with intracystic dendritic papillæ; precarcinomatous.

From the naked-eye descriptions, the tumours were clearly solid in 2 cases, papillary-cystic in 4 cases and solid papillary-cystic in 6 cases, the cysts being completely filled by packed papillary processes. In the remaining 4 cases it is impossible from the description to decide to which of the classes the tumours belonged.

Microscopically the tumours were almost without exception partly solid, partly intracystic papillary, columnar and polygonal-celled carcinomata.

Metastases were present in every case at post-mortem. They were situated in the peritoneum only in 2 cases; in the peritoneum and glands in 4 cases; in the peritoneum and organs in 2 cases; in the peritoneum, glands and organs in 7 cases; and in the glands and organs in 1 case.

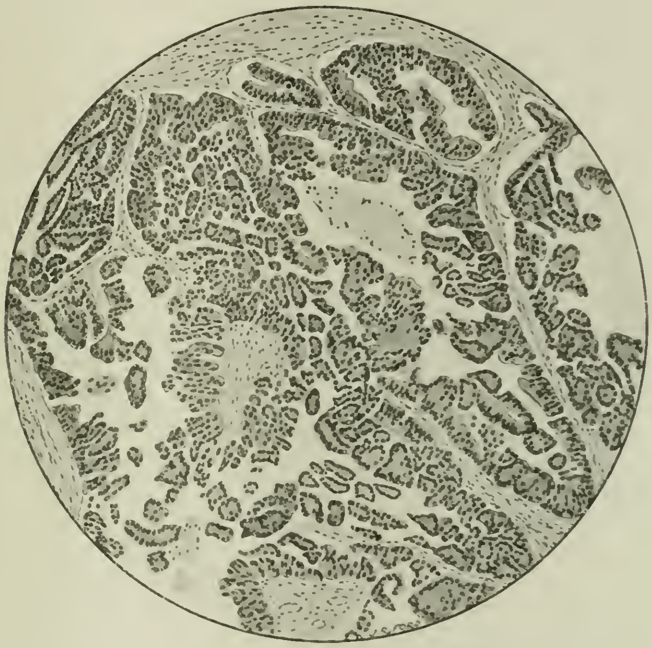

FIG. 9.

Papillary-cystic columnar and polygonal-celled carcinoma of ovary (Wolffian cyst).

The peritoneum was therefore affected in all but 1 case, and the glands in all but 4 cases. The glands were affected in the following order. There were metastases, in lumbar glands, 1 case; in lumbar and iliac glands, 2 cases; in lumbar and cœliac glands, 2 cases; in lumbar, iliac, cœliac, pancreatic and hepatic glands, 1 case; in lumbar, iliac, cœliac, pancreatic, and inguinal glands, 1 case; in lumbar, iliac, cœliac, pancreatic, gastro-coronary and thoracic glands, 1 case: in lumbar, iliac, cœliac, gastro-coronary, hepatic, mesenteric and inguinal glands, 1 case; in lumbar, iliac, cœliac, pancreatic, hepatic, mesenteric, gastro-coronary, thoracic, cervical, axillary, and inguinal glands, 1 case; in lumbar, cœliac, pancreatic, hepatic, gastro-coronary, thoracic, cervical and axillary glands, 1 case; in iliac, cœliac, thoracic and axillary glands, 1 case. The corresponding Fallopian tube was affected in three cases.

Pseudomucinous Carcinomata.

Among the 9 pseudomucinous carcinomata the average age incidence was 44; the oldest patient being 62 and the youngest 26. In 8 the growth was unilateral and in 1 bilateral. In one of the unilateral cases there was a secondary nodule on the surface of the opposite ovary.

The tumour was described macroscopically as solid, medullary, mucoid, with necrotic cystic areas, in 4 cases. As a multilocular pseudomucinous cyst, with solid or semi-solid areas, in 5 cases. In 2 cases the cyst had burst.

Microscopically it was described, without exception, as a partly tubular, partly papillary-intracystic, columnar-celled, mucoid carcinoma.

Metastases were present in only 5 of the cases at post-mortem. They were confined to the peritoneum in 2 cases. They affected both peritoneum and organs in 3 cases. The peritoneum was therefore affected in all cases in which there were metastases. There was no case in which glandular involvement was present.

Summary.

It will thus be seen that—

(1) The majority of primary ovarian carcinomata arose either in Wolffian relics or in pseudomucinous cysts (hypoblastic teratomata), those arising in Wolffian relics being the more numerous.

(2) The age-incidence of Wolffian carcinomata was a decade later than that of the pseudomucinous carcinomata.

(3) The malignancy as demonstrated by metastasis was much greater in the Wolffian than in the pseudomucinous carcinomata.

(4) In both, metastasis was most frequently peritoneal.

(5) In the Wolffian carcinomata the lumbar glands were first involved; later the iliac, cœliac and pancreatic glands.

(6) No pseudomucinous cyst gave rise to glandular metastasis.

SECONDARY OVARIAN CARCINOMATA—38 CASES.

The site of the primary growth was, in order of frequency, as follows: Colon and rectum, 11 cases; stomach, 10 cases; gall-bladder, 2 cases; extrahepatic bile passages and caruncula major of Santorini, 2 cases; pancreas, 2 cases; breast, 4 cases; ovary, 3 cases; suprarenal gland, 2 cases; uterus, 1 case; kidney, 1 case.

There appears to be no special tendency for deposits to occur in the ovary from any one particular primary site unless it be possibly from the suprarenal gland. In carcinomata of the suprarenal gland, the ovary was involved in 1 out of 2½ cases (actually 2 out of 5), in carcinomata of the colon and rectum in 1 out of 10 cases and in carcinomata in each of the other sites 1 out of 6.

The ovarian secondary deposits were bilateral in 19, and unilateral in 19 cases. In 3 of the latter the primary growth was in the opposite ovary so that carcinoma was bilateral in 22 cases, unilateral in 16.

The Size of the Secondary Growths.

In 25 of the 38 cases the ovary or ovaries affected were not enlarged, and in a further 3 cases only very slightly so. In none of these 28 would the ovary or ovaries have formed clinical tumours. In 25 of these the condition was associated with more or less diffuse carcinomatosis of the peritoneum, the ovarian metastases being surface nodules forming a part of the generalized dissemination. In three only was there no carcinomatosis of the peritoneum.

In 10 cases the ovary or ovaries were sufficiently enlarged to form a clinical tumour. In one of these the cause of the enlargement was a benign ovarian cyst, the secondary carcinoma being a small nodule on the surface of the cyst. Excluding this case the remaining 9 have to be considered.

Among these 9 the primary growth was situated in the colon and rectum in 4 cases (170, 1909; 58, 1910; 397, 1914; 381, 1916); in the gall-bladder 2 cases (1160, 1909; 347, 1911); in the extrahepatic bile passages 1 case (309, 1918); in the stomach 1 case (373, 1913), and in the suprarenal gland 1 case (1229, 1909). The condition was unilateral in 5 cases, bilateral in 4 cases.

From the macroscopic description the growth was medullary in 2 cases, cystic in 5 cases, scirrhous in 1 case, and in 1 case impossible to classify.

Microscopically the growth conformed to the primary in type and generally tended to be scirrhous. It was definitely so in 2, and medullary in 1. This conforms with my experience that there is almost without exception a greater amount of fibrous tissue in secondary ovarian growths than in primary. The growth or growths were associated with carcinomatosis of the peritoneum in 5 cases. In 4 cases there was no carcinomatosis of the peritoneum.

Mode of Production of Ovarian Metastases.

The method of metastasis in 31 of the 38 cases was probably by peritoneal implantation. In these 31 cases carcinomatosis of the peritoneum, usually diffuse, was noted and nodules in the pouch of Douglas were often more frequent than elsewhere in the peritoneum. The method of deposition was probably by the fall of cancer cells from other regions where the growth had reached the peritoneal surface. These cases included 5 clinical tumours (170, 1909; 1160, 1909; 1229, 1909; 381, 1916; 309, 1918).

In 3 of the remaining 7 cases (58, 1910; 119, 1911; 385, 1918) the spread was direct. It resulted from adhesion of a primary growth of the bowel to the left ovary. These three included one clinical tumour (58, 1910).

In 3 cases (995, 1909; 347, 1911; 373, 1913) the spread was probably by lymphatic permeation or metastasis; glandular involvement being very marked in all. Two of these were cases with clinical tumours (347, 1911; 373, 1913).

In 1 case (397, 1914), the method of spread was doubtful, being possibly by peritoneal implantation, possibly direct. This was a case of clinical tumour.

Summary.

(1) The figures give no proof that ovarian metastasis is more frequently associated with primary carcinoma in any particular site.

(2) The ovarian metastases were unilateral and bilateral in an equal number of cases.

(3) The method of invasion of the ovaries was: by implantation in 31 of these 38 cases, i.e., 81·5 per cent.; direct in 3 cases, i.e., 7·9 per cent.; by permeation of, or metastasis along, lymphatics, in 3 cases, i.e., 7·9 per cent.

(4) Tumours of clinical significance occurred in 10 cases, i.e., 28·9 per cent.

(5) In 7 of the 9 ovarian tumours of clinical significance, the growth in the ovary or ovaries completely overshadowed the primary growth.

(6) Of the 31 cases in which metastasis was by implantation, the tumours were of clinical significance in 5 cases, i.e., 16 per cent. Of the 3 cases in which the invasion was direct, the tumour was of clinical significance in 1 case, i.e., 33 per cent. Of the 3 cases in which invasion was by lymphatic permeation or metastasis, the tumour was of clinical significance in 2 cases, i.e., 66 per cent.

CONCLUSION.

The following conclusions can I think be derived from this paper:—

(1) That primary carcinoma of the ovary is of comparatively frequent occurrence.

(2) That it frequently arises in a pre-existing benign ovarian cyst.

(3) That it most frequently arises in Wolffian relics, situated in the hilum or even in the cortex of the ovary, the carcinoma being usually preceded by a benign cyst. That it next most frequently arises in a pseudomucinous, adenomatous cyst.

(4) That the classification into cystic and solid carcinomata is unsatisfactory, those arising in pre-existing cysts being frequently solid, those originally solid becoming cystic as the result of necrosis.

(5) The age-incidence of Wolffian carcinomata is a decade later than that of carcinomata arising in pseudomucinous cysts.

(6) Wolffian carcinomata are as often bilateral as unilateral. Carcinomata arising in pseudomucinous cysts are rarely bilateral.

(7) Malignancy as shown by metastasis is greater in Wolffian carcinomata than in carcinomata arising in pseudomucinous cysts.

(8) In both types of primary ovarian carcinoma the peritoneum is the most frequent seat of secondary deposit.

(9) Glandular metastasis is common in association with Wolffian cysts. It is rare in association with pseudomucinous cysts. In the material analysed, glandular metastasis in association with pseudomucinous cysts did not occur.

(10) When the glands are infiltrated, the lumbar and iliac glands are affected first.

(11) Staining with mucicarmine is useless as a method of differentiating primary pseudomucinous carcinomata of the ovary from tumours in the ovary secondary to carcinomata of the alimentary tract. Mucicarmine stains the cells lining pseudomucinous cysts and the epithelium of carcinomata of the bowel with equal intensity.

(12) Secondary carcinomata of the ovary do not appear to be especially associated with primary carcinomata in any particular site.

(13) In more than 75 per cent. of cases, secondary ovarian carcinomata are of no clinical significance. In less than 25 per cent. they are of clinical significance, and in almost all such cases overshadow the primary growth.

(14) Fifty per cent. of secondary ovarian carcinomata are bilateral both where the tumours are of clinical significance and where they are not.

(15) The incidence of bilateral secondary ovarian carcinomata is similar therefore to that of bilateral primary Wolffian carcinomata but is very much greater than that of bilateral primary carcinomata arising in a pseudomucinous cyst.

(16) In about 80 per cent. of cases the method of secondary invasion of the ovaries appears to be implantation. The method of invasion in the remainder is divided equally between direct spread and lymphatic permeation or metastasis.

(17) The incidence of secondary ovarian tumours of clinical significance is much greater when invasion is either by lymphatics or by contact than when invasion is by peritoneal implantation.

The summary of the cases analysed is given in the following appendix:—

APPENDIX.[1]

Wolffian Carcinomata.

P.M. 829, 1909: Female, aged 40. Bilateral primary carcinomata of epoöphoron (left greater than right). Secondary carcinoma of lumbar, iliac, cœliac, pancreatic, hepatic, mesenteric, gastro-coronary, thoracic, cervical, axillary and inguinal lymph glands. Secondary carcinomatous nodules under right parietal pleura. Secondary subpleural carcinomatous nodules in left lung. Secondary subcapsular carcinomatous infiltration of liver. Microscopic: (1) Primary papillary-cystic and solid, cubical and polygonal-celled carcinoma of ovary (Wolffian); (2) secondary papillary-cystic and solid, cubical and polygonal-celled carcinoma in capsule of liver and adherent diaphragm; (3) secondary papillary carcinoma in cervical lymph gland.

P.M. 940, 1909: Female, aged 66. Secondary carcinomatous invasion of lumbar lymph glands in region of left renal artery. Secondary carcinomatous nodule in pelvic colon. Atrophic right ovary. Operation: Removal of primary carcinoma of left ovary. (S.D. 629, 1909) Microscopic: (1) Secondary acinar cubical-celled carcinoma of pelvic colon; (2) secondary acinar, columnar and cubical-celled carcinoma of lumbar lymph gland. (S.D. 629, 1909) Papillary-cystic and solid, primary carcinoma of left epoöphoron. Microscopic: Primary papillary-cystic and tubular, columnar and polygonal-celled carcinoma of left ovary (Wolffian).

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[1] The letters P.M. (post mortem) and S.D. (Surgical Department) together with the figures following them refer to the indices of the Pathological Institute of the London Hospital.

P.M. 636, 1910: Female, aged 59. Solid, nodular, primary carcinoma of left ovary (size of walnut), adherent to posterior wall of uterus. Papillomatous projections on surface of tumour. Small mucinous cysts in tumour tissue. Carcinomatous nodules on visceral and parietal peritoneum. Great thickening of parietal peritoneum and of great omentum. Secondary carcinoma of lumbar, cœliac, pancreatic, gastro-coronary, hepatic, tracheal, right axillary and right and left cervical lymph glands. Carcinomatous infiltration of musculature of hepatic flexure of colon. Secondary carcinoma of visceral and parietal pleuræ of left side. Secondary carcinoma of visceral pericardium. Microscopic: (1) Primary papillary, tubular and solid, columnar and cubical-celled carcinoma of left ovary (Wolffian); (2) secondary papillary, tubular and solid, columnar and cubical-celled carcinoma of peritoneum of uterus; (3) secondary papillary, tubular and solid, columnar and cubical-celled carcinoma of cervical lymph gland; (4) secondary papillary, tubular and solid, columnar and cubical-celled carcinoma of gastro-hepatic lymph gland.

P.M. 504, 1911: Female, aged 54. Bilateral, papillary-cystic primary carcinomata of epoöphora (each 5 cm. diameter). Projection of carcinomatous papillæ into peritoneal cavity. Granular friable growth on peritoneum in pelvis. White nodular growth over whole of peritoneum with extreme matting of intestines. Carcinomatous nodules on superior surface of diaphragm and on both parietal and visceral pleuræ. Microscopic: (1) Primary tubular and papillary, cubical and polygonal-celled carcinoma of ovary (Wolffian); (2) secondary, tubular and papillary, cubical and polygonal-celled carcinoma of peritoneum; (3) secondary tubular and papillary, cubical and polygonal-celled carcinoma of pleura.

P.M. 947, 1911: Female, aged 63. Medullary, papillary-cystic, primary carcinoma of left epoöphoron (4 cm. by 2·5 cm. by 2·5 cm.). Extreme atrophy of right ovary. Carcinomatosis of peritoneum. Carcinomatous nodule beneath diaphragmatic surface of liver. Carcinomatous nodule in one upper lumbar and in one cœliac lymph gland. Microscopic: (1) Primary papillary-cystic, columnar-celled carcinoma of ovary (Wolffian).

P.M. 549, 1912: Female, aged 48. Solid, papillary-cystic, primary carcinoma of left epoöphoron (8 cm. by 6 cm.). Distension of left Fallopian tube by growth. Numerous secondary nodules throughout pelvic peritoneum. Matting of great omentum by secondary nodules. Numerous secondary nodules on under surface of right dome of diaphragm. Fibrous carcinomatous adhesions about pelvic colon. Large secondary masses in right and left lumbar and left iliac lymph glands. Microscopic: (1) Primary papillary-cystic, columnar, cubical and giant-celled carcinoma of ovary (Wolffian); (2) secondary papillary-cystic, columnar, cubical and giant-celled carcinoma of lumbar lymph gland; (3) secondary, papillary-cystic, columnar, cubical and giant-celled carcinoma of iliac lymph gland.

P.M. 439, 1913: Female, aged 43. Secondary carcinomatous nodules on and in mesentery of pendant coils of small intestine in pelvis. Secondary nodule in fat of mesentery of rectum. Carcinomatous nodules on peritoneum, beneath laparotomy incision. Operation, removal of uterus and appendages. (S.D. 766, 1913.) Bilateral, solid, rarely cystic, primary carcinomata of epoöphora (right, 7 cm. by 6 cm. by 4 cm.: left, 11 cm. by 6 cm. by 5 cm.); warty, carcinomatous nodules on surface of tumours and on fundus of uterus. Microscopic: (1) Papillary-cystic and tubular, columnar-celled carcinoma of ovary (Wolffian); (2) columnar-celled cyst of ovary with intracystic, dendritic, papillary fibromata (Wolffian).

P.M. 752, 1913: Female, aged 57. Secondary ulcerated carcinoma in submucosa of posterior wall of urinary bladder. Secondary carcinoma of left lumbar and right and left internal and external iliac lymph glands. Operations: Removal of ovarian tumour at Torquay Hospital in 1907; removal of ovarian tumour in London Hospital in 1908; removal of rectum, pelvic colon and uterus (S.D. 2597, 1913). Microscopic: (1) Secondary papillary and tubular, cubical, spheroidal and polygonal-celled epoöphoric (Wolffian) carcinoma in urinary bladder; (2) Secondary, papillary and tubular, cubical, spheroidal and polygonal-celled, carcinoma of lumbar lymph gland. (S.D. 2597, 1913) Secondary carcinoma of serosa and muscularis of rectum. Microscopic: Secondary, papillary and tubular, columnar-celled carcinoma invading rectum (primary in epoöphoron).

P.M. 818, 1913: Female, aged 50. Papillary-cystic, primary carcinoma of left epoöphoron (21 cm. by 16 cm. by 13 cm.). Secondary carcinoma of left internal iliac, cœliac, right axillary, œsophageal, right and left bronchial lymph glands and in lymph glands at bifurcation of trachea. Numerous nodules of secondary carcinoma (up to 2 cm. by 1 cm.) throughout liver. Carcinomatosis of visceral and parietal pleura especially on right side. Carcinomatosis of both lungs. Carcinomatous nodules scattered over peritoneum. Small epoöphoric cyst in hilum of right ovary. Microscopic: (1) Tubular and papillary-cystic, short columnar and polygonal-celled carcinoma of ovary (Wolffian); (2) secondary, papillary, polygonal-celled carcinoma of liver; (3) secondary, tubular, polygonal-celled carcinoma of pleura and lung; (4) secondary, tubular and papillary polygonal-celled carcinoma of lumbar lymph gland; (5) benign epoöphoric cyst in hilum of right ovary (Wolffian).

P.M. 436, 1914: Female, aged 46. Medullary, primary carcinoma of right epoöphoron (5 cm. by 4 cm.). Medullary and papillary-intracystic, primary carcinoma of left epoöphoron (8 cm. by 8 cm.). Nodular, secondary, carcinomatous infiltration of tissues of both broad ligaments and of pelvic cellular tissue. Carcinomatosis of peritoneum in Douglas's pouch. Carcinomatosis of whole of peritoneum, especially on diaphragm and around pyloric end of stomach. Carcinomatosis of wall of descending and pelvic colon, and of subjacent retro-peritoneal tissue. Secondary carcinoma of lumbar, iliac, cœliac, pancreatic, gastro-hepatic and tracheal lymph glands. Carcinomatosis of left pleura. Microscopic: (1) Solid, alveolar, spheroidal, polygonal and giant-celled carcinoma of ovary (Wolffian); (2) secondary, solid, alveolar, spheroidal, polygonal and giant-celled carcinoma of lumbar lymph gland; (3) secondary, solid, alveolar, spheroidal, polygonal and giant-celled carcinoma of omentum; (4) secondary, solid, alveolar, spheroidal, polygonal and giant-celled carcinoma of pleura.

P.M. 2, 1917: Female, aged 56. Papillary-cystic and solid, primary carcinoma of right epoöphoron (9 cm. diameter). Confluent, secondary, carcinomatous nodules all over peritoneum of pelvis. Carcinomatosis of parietal and visceral peritoneum and of sausage-shaped omentum. Operation, removal of left ovarian cyst. (S.D. 1665, 1916.) Microscopic: (1) Papillary-cystic columnar and cubical-celled carcinoma of ovary (Wolffian); (2) secondary papillary-cystic columnar and cubical-celled carcinoma of floor of pelvis. (S.D. 1665, 1916) Papillary-cystic and solid, primary carcinoma of left epoöphoron (15 cm. diameter). Microscopic: Papillary and tubular, columnar-celled carcinoma of ovary (Wolffian).

P.M. 351, 1917: Female, aged 60. Bilateral, partly solid, partly cystic, primary carcinoma of ovaries (right, 5 cm. in diameter; left, 4 cm. in diameter). Direct carcinomatous invasion of left Fallopian tube and left ureter. Carcinomatous plaques and nodules, some cystic, over parietal and visceral peritoneum. One polycystic secondary nodule (1·2 cm. in diameter) projecting on mucosa of lower ileum. Secondary carcinoma, often polycystic, filling right and left lumbar, right and left internal, external and common iliac, cœliac, hepatic, and right and left inguinal lymph glands. Nodules of secondary carcinoma in mesenteric and gastro-coronary lymph glands. Nodular and diffuse carcinomatosis of lower half of left parietal pleura.

P.M. 265, 1918: Female, aged 61. Necrosed, medullary, primary carcinoma filling Douglas's pouch and covering uterus and appendages. Whole of right Fallopian tube and small fragment of right ovary identified. Secondary medullary, carcinomatous plaque covering under-surface of right dome of diaphragm and greater part of left dome. Carcinomatosis of parietal and visceral peritoneum and omentum. Secondary carcinomatous mass (10 cm. by 3 cm.) in ascending mesocolon. Secondary carcinoma in lumbar and cœliac lymph glands. Microscopic: (1) Papillary and solid, columnar, cubical and polygonal-celled carcinoma of ovary (Wolffian): (2) secondary, intracystic-papillary and solid, columnar, cubical and polygonal-celled carcinoma of retroperitoneal tissue; (3) secondary, solid, diffuse, spheroidal-celled carcinoma of cœliac lymph gland; (4) secondary, intracystic-papillary and solid, columnar, cubical and polygonal-celled carcinoma of ascending mesocolon.

P.M. 275, 1918: Female, aged 49. Papillary-cystic, primary carcinoma of left epoöphoron (14 cm. in diameter). Secondary, carcinomatous nodule (2·5 cm. by 1 cm.) in right ovary. Secondary carcinoma of lumbar, right internal and external iliac, cœliac, pancreatic and right and left inguinal lymph glands. Diffuse carcinomatosis of peritoneum, especially of great omentum. Carcinomatosis of liver, extending from capsule to depth of 2·5 cm. Microscopic: (1) Intracystic-papillary, columnar and cubical-celled carcinoma of left ovary (Wolffian); (2) secondary, intracystic-papillary, columnar, cubical, polygonal and giant-celled carcinoma of right ovary; (3) secondary, tubular, solid and intracystic-papillary, cubical and polygonal-celled carcinoma of inguinal lymph gland; (4) secondary, tubular, solid and intracystic-papillary, cubical and polygonal-celled carcinoma of omentum.

P.M. 360, 1919: Female, aged 45. Secondary carcinomatous nodules (0·5 cm. by 0·3 cm.) on peritoneum of Douglas's pouch and in lower border of great omentum. Secondary nodule in mesentery of iliac colon. Medullary secondary carcinoma in right and left lumbar, right and left external iliac, left internal iliac, cœliac, pancreatic and hepatic lymph glands. Operation: Removal of right and left carcinomatous ovarian cysts. (S.D. 940, 1919.) Microscopic: (1) Secondary, solid and papillary, rarely tubular, polygonal-celled carcinoma of cœliac lymph gland; (2) secondary, solid and papillary, polygonal-celled carcinoma of left lumbar lymph gland; (3) secondary, tubular and intracystic-papillary, short columnar and polygonal-celled carcinoma of pancreatic lymph gland. (S.D. 940, 1919) Bilateral, papillary-cystic, primary carcinomata of epoöphora (17 cm. by 11 cm., and 7 cm. by 5·5 cm.). Secondary, solid, carcinomatous nodule (3·5 cm. by 2 cm.) on ostium of Fallopian tube. Microscopic: (1 and 2) Bilateral, papillary-cystic and solid, cubical and columnar-celled carcinoma of ovaries (Wolffian); (3) secondary, papillary-cystic and solid, cubical and columnar-celled carcinoma of Fallopian tube.

P.M. 361, 1919: Female, aged 74. Secondary carcinomatous infiltration of surface of right ovary (4 cm. by 2 cm. by 1 cm.). Numerous, medullary, partly hæmorrhagic, carcinomatous masses (up to 6 cm. by 5 cm.) on peritoneum of pelvis. Medullary, white, secondary nodule (4 cm. by 3 cm.) on peritoneum of right dome of diaphragm. A few small secondary nodules in capsule of liver. Operation: (1) Removal of primary cystic carcinoma of left ovary (S.D. 505, 1910). Operation: (2) Removal of pelvic secondary mass (S.D. 941, 1919). Microscopic: (1) Secondary, solid and intracystic-papillary, spheroidal and small polygonal-celled carcinoma of surface of ovary; (2) secondary, solid and intracystic-papillary, spheroidal and small-polygonal-celled carcinoma of pelvic peritoneum; (3) secondary, solid and intracystic-papillary, spheroidal and small polygonal-celled carcinoma of right ovary. (S.D. 505, 1910) Cystic and solid, primary carcinoma of left epoöphoron (20·5 cm. in diameter). Microscopic: (1) Solid, almost solid papillary, rarely tubular, small columnar and polygonal-celled carcinoma of ovary (Wolffian). (S.D. 941, 1919) Partly necrosed, secondary, carcinomatous mass (16 cm. by

12·5 cm. by 9·5 cm.) from peritoneum of pelvis. Microscopic: Secondary, solid and almost solid, papillary, small columnar and polygonal-celled carcinoma of peritoneum.

CARCINOMATOUS PSEUDOMUCINOUS CYSTS.

P.M. 1118, 1909: Female, aged 30. Carcinoma of stump of pedicle of left ovarian cyst. Right ovary normal. Secondary, slightly ulcerated, constricting, annular, mucous carcinoma of rectum, at level of cervix uteri. Small secondary carcinomatous nodule in serosa of pelvic colon. Operation: Removal of primary carcinoma of left ovary (S.D. 420, 1909). Microscopic: (1) Secondary, tubular, columnar-celled carcinoma of pedicle of ovarian cyst; (2) secondary, tubular, columnar-celled, mucous carcinoma of rectum; (3) secondary, tubular, columnar-celled, mucous carcinoma of serosa of pelvic colon. (S.D. 420, 1909) Multilocular, partly cystic, partly solid carcinoma of left ovary (17·7 cm. by 15·5 cm. by 10 cm.). Microscopic: Primary, papillary and tubular, columnar-celled, carcinomatous, pseudomucinous cyst of ovary.

P.M. 1132, 1910: Female, aged 38. No carcinoma found. Operation: Removal of carcinomatous cyst of ovary (S.D. 1218, 1910).—(S.D. 1218, 1910) Multilocular, solid and cystic, partly necrotic, carcinoma of ovary (20·5 cm. by 10 cm.). Microscopic: Primary, multilocular, papillary and tubular, columnar-celled, carcinomatous, pseudomucinous cyst of ovary.

P.M. 1195, 1911: Female, aged 30. Primary, multilocular, carcinomatous cyst of right ovary. Pseudomyxoma peritonei. No rupture detected in cyst. Corpora fibrosa in small left ovary. Microscopic: (1) Primary, multilocular tubular, columnar-celled, carcinomatous, pseudomucinous cyst of ovary; (2) degenerate columnar cells in nodular, organizing, fibrinous exudate on peritoneum.

P.M. 315, 1914: Female, aged 52. No carcinoma found. Corpus luteum in right ovary. Operation: Removal of left appendages and resection of small intestine (S.D. 1019, 1914).—(S.D. 1019, 1914) Areas of solid, primary carcinoma in multilocular, pseudomucinous, adenomatous cyst of ovary (24 cm. in diameter). Microscopic: (1) Multilocular, pseudomucinous, adenomatous cyst of ovary; (2) tubular and papillary, columnar-celled, carcinomatous, pseudomucinous cyst of ovary.

P.M. 419, 1914: Female, aged 44. Secondary carcinomatosis of whole of peritoneum, with firm, nodular, carcinomatous growth throughout sausage-shaped omentum. Localized, carcinomatous infiltration of capsule of liver in region of attachment of falciform ligament. No trace of either tube or ovary found. Operation: Removal of ovarian cyst (S.D. 1627, 1913). Microscopic: (1) Secondary, solid, spheroidal and polygonal-celled, scirrhous carcinoma of omentum (primary in ovary). (S.D. 1627, 1913.) Areas of primary carcinoma in pseudomucinous, adenomatous cyst of ovary (25 cm. in diameter). Microscopic: Tubular and papillary, columnar-celled, carcinomatous, pseudomucinous cyst of ovary.

P.M. 173, 1915: Female, aged 59. Cystic and solid, primary carcinomatous, pseudomucinous cyst of right ovary (9·5 cm. by 7 cm. by 5 cm.). Direct, secondary, carcinomatous infiltration of right broad ligament. Infiltration by carcinoma of adherent cæcum. Diffuse, carcinomatous thickening of parietal peritoneum. Close set nodules over visceral peritoneum of intestines, stomach and liver. Secondary carcinoma around hepatic portion of inferior vena cava. Atrophy of left ovary. Microscopic: (1) Papillary-cystic and tubular, columnar-celled, carcinomatous, pseudomucinous cyst of ovary; (2) secondary, diffuse infiltrating, giant-celled carcinoma of parietal peritoneum.

P.M. 590, 1915: Female, aged 62. Necrosed, solid, medullary, partly cystic, primary carcinoma of right ovary (25 cm. by 18 cm.). Solid, medullary, partly cystic, primary carcinoma of left ovary (10 cm. by 10 cm.). Ulcerated, secondary carcinomatous ulcer (3·5 cm. in diameter) in iliac-colon 45 cm. above rectum, adherent to right ovarian growth. Miliary carcinomatosis throughout peritoneum. Secondary, carcinomatous nodule in liver. Scirrhous, carcinomatous adhesions around spleen. Microscopic: (1) Papillary-cystic and tubular, columnar-celled, carcinomatous, pseudomucinous cyst of ovary; (2) secondary, papillary-cystic and tubular, columnar-celled carcinoma of iliac colon; (3) secondary, papillary-cystic and tubular, columnar-celled carcinoma of liver.

P.M. 671, 1915: Female, aged 56. Degenerate, solid, medullary, mucous, primary carcinoma of right ovary (9 cm. by 6·5 cm. by 6·5 cm.). Secondary nodule (3·5 cm. by 2 cm. by 1·4 cm.) of medullary, mucous carcinoma in left ovary. Carcinomatosis of peritoneum. Scirrhous carcinomatous nodule (3·5 cm. by 2·5 cm. by 1·5 cm.) in tail of pancreas. Secondary nodule of carcinoma, constricting lumen of small intestine, 7 cm. above ileocæcal valve. Scirrhous, secondary nodule in and beneath capsule of right lobe of liver. Microscopic: (1) Papillary-cystic and tubular, columnar-celled, carcinomatous, pseudomucinous cyst of right ovary; (2) secondary, papillary-cystic and tubular, columnar-celled carcinoma of left ovary; (3) secondary, papillary-cystic and tubular, columnar-celled carcinoma of liver; (4) secondary, papillary-cystic and tubular, columnar-celled carcinoma of pancreas.

P.M. 272, 1917: Female, aged 26. No secondary carcinoma. No abnormality of right ovary. Operation: Removal of carcinomatous ovarian cyst (S.D. 539, 1917).—(S.D. 539, 1917) Area of primary carcinoma in multilocular, pseudomucinous, adenomatous cyst (30 cm. by 29 cm.). Microscopic: (1) Tubular and papillary, columnar-celled, carcinomatous, pseudomucinous cyst of ovary.

SECONDARY CARCINOMATOUS NODULES IN OVARY.

P.M. 194, 1909: Female, aged 42. Ulcerated, scarred, scirrhous, primary carcinoma of greater curvature of stomach, infiltrating mucous membrane. Permeation of lymphatics of lesser curvature to liver and diaphragm. General carcinomatosis of peritoneum. Secondary carcinoma of lymph glands of lesser and greater curvature of stomach and of cœliac lymph glands. Carcinomatous infiltration of cortex of ovaries (4 cm. by 3 cm.). Microscopic: (1) Solid, spheroidal-celled, scirrhous carcinoma of stomach; (2) secondary, solid, spheroidal-celled, scirrhous carcinoma of lymph gland of lesser curve of stomach; (3) secondary, solid, spheroidal-celled, scirrhous carcinoma of ovary.

P.M. 945, 1909: Female, aged 42. Large, annular, constricting, ulcerated, mucous, primary carcinoma in transverse colon. Carcinomatosis of peritoneum. Masses of secondary carcinoma between diaphragm and liver, surrounding spleen and in Douglas's pouch. Secondary carcinoma of lumbar, cœliac, mesenteric, bronchial, tracheal and left supraclavicular lymph glands. Subcapsular, carcinomatous invasion of liver. Nodular, secondary carcinoma of both ovaries (5 cm. by 4 cm. by 4 cm.). Microscopic: (1) Tubular, mucous carcinoma of transverse colon; (2) secondary, tubular, mucous carcinoma of ovary.

P.M. 955, 1909: Female, aged 51. Discrete nodule of primary carcinoma in left breast over fifth intercostal space. Large mass of carcinomatous lymph glands in left posterior cervical triangle, reaching scapula and invading cervical muscles. Massive, confluent, carcinomatous lymph glands in right posterior triangle and in both axillæ. Carcinomatous infiltration of tracheal, cœliac and lumbar lymph glands. Secondary carcinomatous nodules in cystic ovaries. Microscopic: (1) Primary, solid, alveolar and acinar, spheroidal-celled, medullary carcinoma of left breast; (2) secondary, solid, alveolar and acinar, spheroidal-celled, medullary carcinoma of cervical lymph gland; (3) secondary, solid, alveolar and acinar, spheroidal-celled, medullary carcinoma of left ovary: (4) secondary, solid, alveolar and acinar, spheroidal-celled, medullary carcinoma of right ovary.

P.M. 1142, 1909: Female, aged 45. Large, scirrhous, primary carcinoma occupying whole of left breast. Infiltration of cutaneous lymphatics around left nipple. Small, secondary, carcinomatous nodules in left axillary, right and left cervical, œsophageal, gastric, cœliac, lumbar and mesenteric lymph glands. Numerous submucous secondaries in appendix and in lower 7·5 cm. of ileum. Small submucous nodules scattered throughout jejunum and ileum. Subperitoneal secondaries in Douglas's pouch. Miliary secondary deposits in pericardium. One small secondary nodule in liver. Carcinomatous infiltration of enlarged red spleen. Minute secondaries in medulla of suprarenals. Carcinomatous nodules in right ovary. Submucous, carcinomatous nodules in stomach. Scirrhous secondary deposits throughout marrow of femur, sternum and ribs; ebonizing secondaries in lumbar spine. Microscopic: (1) Solid, rarely tubular, scirrhous carcinoma of left breast; (2) secondary, solid, rarely tubular, scirrhous carcinoma of iliac lymph gland; (3) secondary, solid, rarely tubular, scirrhous carcinoma of right ovary; (4) secondary, solid, rarely tubular, scirrhous carcinoma of lumbar lymph gland; (5) secondary, solid, rarely tubular, scirrhous carcinoma of left cervical lymph gland; (6)

secondary, solid, rarely tubular, scirrhous carcinoma of marrow of femur; (7) secondary, solid, rarely tubular, scirrhous carcinoma of spleen.

P.M. 289, 1910: Female, aged 37. Scar of ulcer midway on lesser curve with scarred ulceration of pyloric end of stomach. Scirrhous carcinomatous infiltration of whole of submucosa of stomach, spreading through muscle to subserosa. Submucous infiltration of lower 3 cm. of œsophagus. Secondary carcinoma in lymph glands of lesser curve and in cœliac, pancreatic, lumbar, œsophageal, supraclavicular and right and left cervical lymph glands. Carcinomatosis of peritoneum. Secondary carcinomatous nodules in bodies of second, third and fourth lumbar vertebræ, and one secondary nodule in manubrium sterni. Secondary carcinomatous nodules in right ovary. Microscopic: (1) Primary, solid and tubular, spheroidal-celled carcinoma of stomach; (2) secondary, solid and tubular, spheroidal-celled carcinoma of cervical lymph gland; (3) secondary, solid and tubular, spheroidal-celled carcinoma of right ovary.

P.M. 864, 1910: Female, aged 31. Ulcerated primary carcinoma on posterior surface of pyloric canal, 4 cm. from sphincter. Submucous infiltration of pyloric canal and of first part of duodenum. Carcinomatosis of peritoneum, especially of Douglas's pouch. Secondary carcinomatosis of mesenteric, cœliac, lumbar and omental lymph glands, in small lymph glands of lesser curvature of stomach and in large lymph gland of cardia; also in lymph glands of posterior and superior mediastina, and in a mass of glands in left posterior cervical triangle. Small secondary nodules in visceral pleuræ. Small, secondary nodules in liver. Secondary nodule (1 cm.) in left ovary. Microscopic: (1) Solid, spheroidal-celled carcinoma of stomach; (2) secondary, solid, spheroidal-celled carcinoma of liver; (3) secondary, solid, spheroidal-celled carcinoma of left cervical lymph gland; (4) secondary, solid, spheroidal-celled carcinoma of mesenteric lymph gland.

P.M. 123, 1911: Female, aged 46. Scirrhous primary carcinomatous infiltration of pylorus and of 10 cm. of pyloric end of stomach: superficial ulceration of growth on greater curvature. Submucous and subserous carcinomatous spread in stomach. Secondary carcinoma of cœliac, gastro-hepatic and upper lumbar lymph glands. Carcinomatous nodule in left supraclavicular lymph gland. Carcinomatosis of peritoneum, especially of diaphragm and in Douglas's pouch. Sausage-shaped omentum. Carcinomatosis of parietal pleura. Numerous secondary nodules, with central necrosis, in fatty liver. Small secondary nodule (size of pea) in both ovaries. Microscopic: (1) Tubular, small cubical and columnar-celled carcinoma of stomach.

P.M. 1136, 1911: Female, aged 34. Large, ulcerating, purulent, medullary, annular, primary carcinoma of pelvic colon. Carcinomatosis of peritoneum. Extensive carcinomatosis of Douglas's pouch. Carcinomatous infiltration of great omentum. Secondary carcinoma of lumbar, iliac, cœliac, hepatic, lymph glands, and glands of lesser and greater omenta. Numerous carcinomatous secondaries in liver. Diffuse carcinomatous infiltration of liver spreading from lymphatics around gall-bladder. Secondary, carcinomatous nodules in both ovaries. Microscopic: (1) Primary, solid, rarely tubular, polygonal-celled carcinoma of colon; (2) secondary, solid, rarely tubular, polygonal-celled carcinoma of liver.

P.M. 1191, 1911: Female, aged 64. Large, primary carcinomatous ulcer of pelvic colon, 28 cm. from anus. Nodule of carcinoma in site of left ovary, in base of ulcer. Microscopic: (1) Secondary, tubular and papillary, columnar-celled, mucous carcinoma of ovary.

P.M. 1221, 1911: Female, aged 41. Scirrhous, constricting, primary carcinoma (2·5 cm. long) involving common bile and cystic ducts. Infiltration of surrounding tissues and walls of hepatic and cystic ducts and gall-bladder. Cholelithiasis. Numerous carcinomatous secondaries throughout liver. Scirrhous nodules in omentum, mesentery, and in subserous tissue of cæcum and ascending colon. Implantation nodules in Douglas's pouch. Small, scarred, carcinomatous nodule in appendix. Large, secondary carcinomatous nodule in right suprarenal gland. Carcinomatous infiltration of hepatic, cœliac, upper lumbar, gastric, bronchial and lower tracheal lymph glands. Carcinomatous infiltration of lymphatics of visceral pleura and of under surface of diaphragm. Secondary carcinomatous nodule (2 cm. in diameter) in right ovary. Microscopic: (1) Primary, solid, rarely tubular, cubical, spheroidal and polygonal-celled, scirrhous, rarely mucous, carcinoma of common bile-duct; (2) secondary, solid, rarely tubular, cubical, spheroidal and polygonal-celled, scirrhous, rarely mucous, carcinoma of suprarenal gland; (3) secondary, solid, rarely tubular, cubical, spheroidal and polygonal-celled, scirrhous, rarely mucous, carcinoma of liver; (4) secondary, solid, rarely tubular, cubical, spheroidal and polygonal-celled scirrhous, rarely mucous, carcinoma of ovary; (5) secondary, solid, rarely tubular, cubical, spheroidal and polygonal-celled, scirrhous, rarely mucous, carcinoma of lumbar lymph gland.

P.M. 793, 1912: Female, aged 50. Scirrhous, primary carcinoma (3 cm. in diameter) of breast beneath right nipple. Multiple, secondary, carcinomatous nodules throughout both lungs. Carcinomatosis of parietal and visceral pleuræ. Carcinomatous nodules in bronchial, mediastinal, right supraclavicular and lower cervical lymph glands, also in gastro-hepatic, cœliac, pancreatic and upper lumbar lymph glands and in glands of lesser and greater curve of stomach. Carcinomatous nodules and diffuse carcinomatosis throughout liver. Secondary scirrhous nodule in right suprarenal gland. Carcinomatous nodules in spleen. Carcinomatous nodules in lower part of shaft of right humerus. Sclerosing carcinoma in neck and upper part of shaft of right femur and in lumbar vertebræ. Secondary carcinomatous nodules in peritoneum of Douglas's pouch with scirrhous carcinomatous nodules in both ovaries (right 2·5 cm. by 2·5 cm.; left 2·5 cm. by 2·5 cm.).

P.M. 197, 1913: Female, aged 48. Ulcerated primary carcinoma of vaginal portion of cervix uteri. Carcinomatous infiltration of whole thickness of cervical and uterine walls. Masses of nodular growth in both broad ligaments.

Direct spread of carcinoma to adjacent ovaries (5 cm. greatest diameter) and along walls of both Fallopian tubes. Discrete carcinomatous nodules on upper part of vagina. Extensive carcinomatosis of pelvic peritoneum. Carcinomatous nodules scattered over rest of peritoneum; larger nodules (up to 2 cm.) in omentum. Massive carcinomatous secondaries in iliac (4 cm.), lumbar (3 cm.), and in cœliac lymph glands (2 cm.); also in bronchial and lower tracheal lymph glands; one nodule in right supraclavicular lymph gland and one minute nodule in lower œsophageal lymph gland. Multiple, small, secondary nodules (1 cm.) throughout liver. A few small carcinomatous nodules in kidneys. Numerous large, secondary nodules (3 cm.) throughout both lungs. Microscopic: (1) Primary, solid, small, polygonal-celled carcinoma of vaginal portion of cervix uteri invading corpus; (2) secondary, solid, polygonal-celled carcinoma of liver.

P.M. 646, 1913: Female, aged 38. Diffuse, scirrhous, primary carcinoma of whole of right breast. Carcinomatous infiltration of skin, subjacent fasciæ and pectoral muscles spreading to left side of chest and down right rectus sheath. Nodules of secondary carcinoma in left breast. Secondary carcinoma in right and left axillary, bronchial, mediastinal, cœliac, left hepatic, pancreatic, lumbar, iliac, inguinal and mesenteric lymph glands, and in lymph glands on lesser curvature of stomach. Scirrhous carcinomatous thickening of right parietal pleura. Secondary carcinomatous nodules on both surfaces of both domes of diaphragm. Carcinomatosis of parietal and visceral peritoneum. A few small, subperitoneal, carcinomatous nodules in pancreas. Secondary, carcinomatous nodules in suprarenal glands. Minute, secondary nodules in liver. Secondary carcinomatous nodules in both ovaries (left, 4 cm. by 2·5 cm.; right, 4 cm. by 2 cm.). Microscopic: (1) Secondary, solid and tubular, polygonal celled, scirrhous carcinoma of liver.

P.M. 93, 1914: Female, aged 45. Primary carcinoma of head of pancreas. Complete obliteration of common bile-duct, in its transpancreatic course. Mass of firm, seminecrotic growth (8 cm. in diameter) surrounding extrapancreatic common bile-duct, lower portion of hepatic duct and whole of cystic duct. Carcinomatous infiltration of tissues in hilum of liver, and carcinomatous invasion of portal and hepatic veins. Carcinomatous invasion of wall of duodenum, puckering mucosa. Direct, carcinomatous invasion of liver in region of gall-bladder; a few secondary nodules scattered throughout liver. Matted mass of secondary carcinoma in cœliac, pancreatic and lumbar lymph glands; secondary carcinoma in glands of lesser curvature of stomach and in lower œsophageal and left supraclavicular lymph glands. Slight carcinomatous invasion of lower part of inferior vena cava. Multiple carcinomatous nodules on peritoneum, most marked in pelvis. Secondary nodule on surface of unenlarged right ovary. Microscopic: (1) Tubular and solid acinar, spheroidal and polygonal-celled carcinoma of head of pancreas, invading common bile-duct; (2) secondary, solid, polygonal-celled carcinoma of œsophageal lymph gland; (3) secondary, solid and acinar, polygonal-celled carcinoma of liver.

P.M. 590, 1914: Female, aged 63. Large, primary carcinomatous ulcer (8·5 cm. by 6 cm.) riding on lesser curve of stomach near pylorus. Secondary carcinoma of cœliac lymph glands and glands of greater and lesser curvature of stomach. Extensive, lymphogenous carcinomatosis of visceral and parietal peritoneum. Numerous submucous secondaries (up to 2 cm. by 2·5 cm.) throughout small and large intestine, very numerous in lower colon. Diffuse carcinomatous infiltration of submucosa of appendix, and of walls of ureters in their middle third. Carcinomatous infiltration of both surfaces of diaphragm, of left parietal pleura and of lymphatics of posterior border of lower lobe of left lung. Implantation secondary carcinoma in Douglas's pouch. Small nodules of growth on walls of Fallopian tubes. Diffuse carcinomatous infiltration without enlargement of ovaries. Microscopic: (1) Secondary, solid, spheroidal-celled carcinoma of lymph gland: (2) secondary, solid, spheroidal-celled carcinoma of ovary; (3) secondary, solid, spheroidal-celled, scirrhous carcinoma of submucosa of appendix; (4) secondary, solid, spheroidal-celled, scirrhous carcinoma of submucosa of ileum; (5) secondary, solid, spheroidal-celled, scirrhous carcinoma of diaphragm.

P.M. 439, 1915: Female, aged 49. Ulcerated primary carcinoma (3 cm. by 2·3 cm.) in caput caeci. Massive, secondary, carcinomatous infiltration of subjacent peritoneum. Scirrhous infiltration of whole of parietal and visceral peritoneum and sausage-shaped omentum. Secondary carcinomatous nodule in cœliac lymph gland (0·9 cm. by 0·4 cm.). Secondary carcinomatous nodules (up to 0·7 cm. by 0·1 cm.) on left visceral and parietal pleuræ. Secondary carcinomatous infiltration of body of uterus. Secondary carcinomatous infiltration of ovaries (right, 4·5 cm.; left, 5 cm.). Microscopic: (1) Tubular, columnar-celled, mucous carcinoma of cæcum: (2) secondary, tubular, columnar and cubical-celled, mucous carcinoma of left ovary; (3) secondary, tubular, cubical-celled, mucous carcinoma of endo- and myo-metrium.

P.M. 205, 1916: Female, aged 47. Primary carcinomatous ulcer (4·5 cm. in diameter) saddling lesser curve of pyloric canal and just involving pyloric sphincter. Carcinomatous infiltration of subjacent serosa and of whole wall of lesser curve of stomach. Secondary carcinomatous infiltration of gastro-coronary, cœliac, right and left lumbar lymph glands. Diffuse carcinomatosis of parietal and visceral peritoneum and sausage-shaped omentum. Especially marked carcinomatosis of peritoneum of Douglas's pouch. A few small nodules projecting into mucosa of ileum. Small nodules of secondary carcinoma in left diaphragmatic pleural adhesions. Carcinomatous infiltration of both ovaries (right, 5 cm. by 3 cm. by 2 cm.; left, 4·5 cm. by 4 cm. by 2·5 cm.).

P.M. 297, 1916: Female, aged 43. Non-ulcerated, scirrhous, primary, carcinomatous plaque (5 cm. sagittally by 3 cm. from right to left by 0·9 cm. thick) saddling lesser curve 8 cm. from cardia and 5 cm. from pyloric sphincter. Scirrhous carcinoma filling glands of lesser curve of stomach, also cœliac, hepatic and lumbar lymph glands and two lymph glands in left supraclavicular fossa. Partial replacement of gastro-coronary and right supra-pancreatic lymph glands by secondary carcinoma. Almost complete replacement of lymph glands in great omentum by secondary carcinomatous growth. A few miliary, puckered, secondary, carcinomatous nodules in utero-vesical and Douglas's pouches. Closely set osteoplastic, secondary, carcinomatous deposits in neck and upper two-thirds of shaft of femur; few nodules in lower third. Nodules of osteoplastic growth throughout nedullary cavity of humerus and in manubrium sterni. Almost complete replacement by growth of bodies of lumbar vertebræ. Osteoplastic, secondary, carcinomatous nodules in medulla of ribs. Secondary nodules (up to 1·5 cm.) in both ovaries. Microscopic: (1) Solid, trabecular, rarely acinar and tubular, spheroidal-celled, scirrhous, rarely mucous, carcinoma of stomach; (2) secondary, solid, trabecular, spheroidal-celled carcinoma of bone marrow.

P.M. 461, 1916: Female, aged 68. Small, annular, primary, carcinomatous ulcer (2·5 cm. longitudinally) of ascending colon. A few secondary carcinomatous nodules in cœliac lymph glands. Numerous secondary carcinomatous nodules in liver. A few secondary carcinomatous nodules (up to 1·7 cm. by 0·5 cm.) on peritoneum of Douglas's pouch. Secondary nodule (1·7 cm. by 0·7 cm.) in left ovary.

P.M. 506, 1916: Female, aged 31. White, medullary, often softened and cavitated, primary carcinoma of left suprarenal gland (9 cm. by 6 cm.) compressing and invading upper pole of left kidney. Direct carcinomatous invasion of posterior wall of cardia and of greater part of body of stomach. Direct, carcinomatous invasion of left dome of diaphragm. Secondary, carcinomatous nodules in left kidney. Carcinomatous invasion of left renal vein. Secondary carcinomatous nodules filling right and left lumbar, right and left common, external and internal iliac lymph glands; also supra-pancreatic, infra-pancreatic, cœliac, mesenteric, hepatic, gastro-coronary, lower œsophageal, anterior and superior mediastinal lymph glands, also glands of tracheal bifurcation and right and left bronchial glands. Secondary carcinomatous nodules in left psoas muscle and in retroperitoneal tissues of left iliac fossa. Direct carcinomatous invasion of body and tail of pancreas. Secondary carcinomatous nodules throughout liver. Carcinomatous plaques on pleural surfaces of right and left domes of diaphragm. Numerous nodules of secondary carcinoma in left parietal pleura. Closely set miliary, secondary nodules all over visceral pleura of lower lobe of left lung with sparse nodules over remainder of left and whole of right visceral pleura. Carcinomatosis of great omentum and of mesenteries of large intestine. Numerous secondary carcinomatous nodules over visceral peritoneum, especially in uterovesical and Douglas's pouches. Two secondary carcinomatous nodules (1 cm. in diameter) in left ovary. Microscopic: (1) Secondary, solid, spheroidal and polygonal-celled carcinoma of ovary; (2) secondary, solid, spheroidal and polygonal-celled carcinoma of liver; (3) secondary, papillary and solid, spheroidal, and polygonal-celled carcinoma of omentum.

P.M. 535, 1917: Female, aged 40. Diffuse, scirrhous, primary carcinoma of whole of pyloric canal and pyloric antrum extending along lesser curve to œsophagus. Carcinomatous infiltration of muscularis over lesser curvature and posterior wall of stomach. Nodules of carcinomatous growth on peritoneum of lesser curve. Carcinomatosis of lymph glands of lesser curve of stomach and of gastro-coronary, subpyloric, cœliac, right and left lumbar, right and left common and external iliac lymph glands; also of lower œsophageal, paratracheal and tracheal bifurcation lymph glands. Very soft, white, osteoclastic secondaries scattered throughout sternum, lumbar vertebræ and ribs extending into intercostal spaces. Diffuse carcinomatous infiltration of peritoneum of Douglas's pouch. Numerous nodules of pink, medullary growth (up to 1·2 cm.) in both ovaries (right, 4 cm. by 2·5 cm. by 2 cm.; left, 4·5 cm. by 3 cm. by 2·2 cm.).

P.M. 306, 1918: Female, aged 57. Partly necrosed, solid, primary carcinoma (9 cm. in diameter) in centre of posterior part of right kidney. Carcinomatous infiltration of perirenal tissues in region of pelvis. Carcinomatosis of right and left lumbar, iliac, cœliac, para-œsophageal, bronchial, para-tracheal and superior mediastinal lymph glands. Numerous secondary nodules in retroperitoneal tissues, one infiltrating lower half of right suprarenal gland. Secondary nodules in right ovary (4 cm. by 2·5 cm. by 1·5 cm.). Two miliary, secondary nodules on serosa of uterus. Microscopic: (1) Solid, polygonal-celled carcinoma of right kidney; (2) secondary, solid, polygonal, often vacuolated, spheroidal-celled carcinoma of lumbar lymph gland.

P.M. 385, 1918: Female, aged 47. Ulcerated, constricting, annular, primary carcinoma (5 cm. longitudinally by 2 cm. internal circumference) of iliac colon 36 cm. above anus. Direct carcinomatous invasion of adherent left ovary. Direct carcinomatous invasion of mesentery of ileum, 35 cm. above ileocœcal valve. Secondary carcinomatous nodule (1·2 cm. by 0·8 cm. by 0·3 cm.) projecting through mucosa of ileum at this point. Secondary carcinomatous nodule in liver (3 cm. by 2 cm.).

P.M. 188, 1919: Female, aged 35. Scirrhous primary carcinoma with necrotic centre, involving whole thickness of neck of pancreas at its junction with head. Carcinomatous infiltration of surrounding tissues and of posterior serous surface of stomach. Carcinomatosis of serous surfaces of extrahepatic bile-ducts and gall-bladder. Secondary carcinoma in pancreatic, cœliac, gastric, lumbar, iliac, œsophageal, bronchial and left supraclavicular lymph glands. Carcinomatosis of peritoneum most marked on diaphragm, in omentum and in Douglas's pouch. Carcinomatosis of visceral and parietal pleuræ most marked on diaphragmatic surfaces. A few carcinomatous nodules in substance of lungs. Very numerous carcinomatous nodules throughout liver. A few secondary carcinomatous nodules in spleen. Many secondary carcinomatous nodules in kidneys and suprarenal glands. Many carcinomatous nodules in marrow of femur and vertebræ. Secondary carcinoma in cystic, slightly enlarged, ovaries. Microscopic: (1) Tubular, cubical and columnar-celled, scirrhous carcinoma of neck of pancreas; (2) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of liver; (3) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of spleen; (4) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of suprarenal; (5) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of kidney; (6) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of ovary; (7) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of femur; (8) secondary, tubular, cubical and columnar-celled, scirrhous carcinoma of vertebræ.

P.M. 466, 1919: Female, aged 60. Scirrhous, primary carcinomatous infiltration of pyloric end of stomach. Secondary, scirrhous, carcinomatous nodule in head of pancreas. Secondary carcinomatous infiltration of peri-pancreatic tissues and mesentery of small intestine. Carcinomatous infiltration of lumbar, iliac, and cœliac lymph glands. Carcinomatous infiltration of mesenteries of cæcum, ascending colon and of perirectal tissues. Carcinomatous infiltration of pericardial, mediastinal and peri-œsophageal tissues. Carcinomatosis of peritoneum of Douglas's pouch. Carcinomatous infiltration of surface of both ovaries. Microscopic: (1) Solid, spheroidal and polygonal-celled, scirrhous carcinoma of stomach: (2) secondary, solid, spheroidal and polygonal-celled, scirrhous carcinoma in mesentery of colon.

CLINICAL CARCINOMATOUS TUMOURS IN OVARY.

P.M. 170, 1909: Female, aged 33. Ulcerating, constricting, mucoid, primary carcinoma of termination of pelvic colon. Large, solid, mucoid, secondary carcinoma in right ovary, with broken-down area perforated into peritoneum. Two small submucous secondaries in jejunum and colon. Microscopic: (1) Tubular, polygonal and cubical-celled, mucous carcinoma of pelvic colon with secondary in right ovary.

P.M. 1160, 1909: Female, aged 67. Scirrhous primary carcinoma of extremely contracted gall-bladder invading liver substance. Cholesterin stones in gall-bladder and cystic duct. Large secondary carcinomatous nodule at root of mesentery. Secondary carcinomatous infiltration of cœliac and lumbar lymph glands. Small secondary nodule compressing right ureter at brim of pelvis. Submucous, secondary nodules in jejunum, cæcum and pelvic colon. Scattered, small secondary nodules throughout peritoneum, particularly in pelvis. Large, degenerated, secondary carcinomatous masses in cystic ovaries (right, size of large apple; left, of hen's egg). Microscopic: (1) Primary, tubular, columnar-celled carcinoma of gall-bladder; (2) secondary, tubular, columnar-celled carcinoma of cœliac lymph gland; (3) secondary, tubular, columnar-celled carcinoma of lumbar lymph gland; (4) secondary, tubular, columnar-celled carcinoma of ovary.

P.M. 1229, 1909: Female, aged 50. Pigmented primary carcinoma (size of walnut) in right suprarenal gland. Carcinomatosis of peritoneum; masses of secondary, carcinomatous growth in sausage-shaped omentum; polypoid growth scattered over peritoneum; minute, black-pigmented, secondary, carcinomatous nodules on peritoneum; cystic degeneration of many growths; secondary polypoid growths on both surfaces of right dome of diaphragm and on pleura. Secondary carcinoma of lower œsophageal lymph glands. Secondary, moist, semitransparent, homogeneous, white, gelatinous, medullary tumour of left ovary, with numerous, small pin-head, cystic spaces (ovary of size of Jaffa orange). Microscopic: (1) Primary, solid, acinar, polygonal, rarely cubical-celled, pigmented carcinoma of right suprarenal gland; (2) secondary, tubular and papillary, columnar and cubical-celled carcinoma of left ovary; (3) secondary, intracystic-papillary, columnar, cubical and polygonal-celled carcinoma of omentum; (4) secondary, papillary, columnar, cubical and polygonal-celled carcinoma of mesentery; (5) secondary, solid, polygonal and giant-celled, pigmented carcinoma of small intestine; (6) secondary, papillary and solid, polygonal-celled, pigmented carcinoma of œsophageal lymph gland.

P.M. 58, 1910: Female, aged 36. Annular, constricting, primary carcinoma of pelvic colon. Necrotic and cystic secondary carcinoma of left ovary filling pelvis and occupying abdominal cavity as far as umbilicus. Submucous, secondary carcinomatous nodules in appendix. Follicular cysts in right ovary. Microscopic: (1) Ulcerated, tubular, columnar-celled carcinoma of pelvic colon; (2) secondary, tubular, columnar and cubical-celled carcinoma of left ovary; (3) secondary, tubular, columnar-celled carcinoma of appendix.

P.M. 347, 1911: Female, aged 28. Hæmorrhagic, medullary, mucoid, primary carcinoma (14 cm. in diameter), entirely replacing and filling gall-bladder. Multiple secondary carcinomatous nodules throughout liver. Secondary carcinoma of cœliac and gastro-hepatic lymph glands. Mass of secondary growth (16 cm. by 12 cm. by 9 cm.) in anterior and superior mediastina, invading parietal pericardium. Similar growth invading pleura of left lung. Masses of secondary, carcinomatous growth in left fifth and right seventh rib. Mass of secondary growth in right ovary. Microscopic: (1) Primary, acinar, polymorphous-celled, medullary carcinoma of gall-bladder; (2) secondary, acinar, polymorphous-celled, medullary carcinoma of liver; (3) secondary, acinar, polymorphous-celled, medullary carcinoma of ovary.

P.M. 615, 1911: Female, aged 45. Ulcerated, tightly-constricting, primary carcinoma of cæcum. Secondary involvement of lower 3 cm. of ileum. Invasion by growth of adherent omental fat and of two adherent loops of small intestine. Localized carcinomatosis of peritoneum. Nodule of secondary carcinoma in adherent right ovarian cyst.

P.M. 373, 1913: Female, aged 46. Scirrhous, primary carcinoma of stomach: leather-bottle stomach with large ulcer (6 cm. by 6 cm.) situated on lesser curve 2 cm. from pylorus and 2·5 cm. from œsophagus. Infiltration of wall of stomach by growth (1·2 cm. thick). Secondary carcinoma of lymphatic glands in great, gastro-hepatic and gastro-splenic omenta and in subpyloric and retropyloric groups, also in lumbar and left supraclavicular lymph glands. Nodules of secondary carcinoma in a lower œsophageal lymph gland and in a gland of hilum of left lung. Operation: Removal of both uterine appendages (S.D. 1220. 1913). (S.D. 1220, 1913) Œdematous, solid, rarely cystic, secondary carcinoma of ovary (18 cm. by 9 cm. by 12 cm.). Œdematous, solid, secondary carcinoma of ovary (15 cm. by 9 cm. by 9·5 cm.). Microscopic: Secondary, solid, spheroidal and polygonal-celled, scirrhous carcinoma of ovary (primary stomach).

P.M. 397, 1914: Female, aged 42. Secondary, scirrhous, carcinomatous nodule in left lateral vaginal fornix. Secondary, carcinomatous infiltration of vaginal wall. Operation: Removal of pelvic colon, rectum, uterus and both ovaries together with coccyx (S.D. 1336, 1914). Microscopic: (1) Secondary, solid and tubular, cubical and columnar-celled carcinoma of vaginal wall. (S.D. 1336, 1914) Ulcerated, annular, constricting, primary carcinoma of pelvic colon 24 cm. above anus. Direct, secondary, carcinomatous invasion of outer coats of adherent loops of colon, 9 cm. and 12 cm. from anus. Bilateral, solid, partly cystic, secondary carcinoma of ovaries (larger, 7 cm. by 4 cm.). Microscopic: (1) Tubular, columnar-celled, scirrhous carcinoma of pelvic colon; (2) secondary, tubular, columnar-celled, scirrhous carcinoma of right ovary; (3) secondary, tubular, columnar-celled, scirrhous carcinoma of left ovary.

P.M. 381, 1916: Female, aged 68. Ulcerated, annular, primary carcinoma (2·5 cm. longitudinally by 3·8 cm. circumferentially) in pelvic colon opposite brim of true pelvis. Secondary carcinomatous nodule in left lumbar gland at origin of inferior mesenteric artery. Secondary carcinomatous nodules in liver. Secondary carcinomatous nodules in Douglas's pouch. Cystic, secondary carcinoma of right ovary (10 cm. by 7 cm. by 5 cm.). Atrophy of left ovary. Microscopic: (1) Solid and tubular, polygonal and columnar-celled carcinoma of pelvic colon; (2) secondary, papillary-cystic and tubular, columnar-celled carcinoma of ovary; (3) atrophy of left ovary.

P.M. 309, 1918: Female, aged 45. Papillary, primary carcinoma of mucosa of common hepatic duct, completely obliterating lumen (2·5 cm. long by 0·4 cm. thick by 2·2 cm. internal circumference). Papillary carcinoma of mucosa of cystic duct. Medullary carcinoma (3·5 cm. in diameter) of wall of fundus of gall-bladder invading lumen. Secondary carcinoma of iliac and lumbar lymph glands with one nodule in lymph gland of tracheal bifurcation. Secondary nodules in capsule of anterior surface of liver over gall bladder. Pin-head and minute secondary carcinomatous nodules on visceral pleuræ. A few secondary carcinomatous plaques on diaphragm and on peritoneum of right renal pouch and pouch of Douglas. Secondary, necrotic, cystic, deeply bile-stained carcinomata of right (15 cm. by 12 cm. by 11 cm.) and left ovary (14 cm. by 8 cm. by 8 cm.). Microscopic: (1) Intracystic-papillary and tubular, columnar-celled carcinoma of bile passages; (2) secondary, intracystic-papillary and tubular, columnar-celled carcinoma of ovary; (3) secondary, intracystic-papillary and tubular, columnar-celled carcinoma of lymph gland.

DISCUSSION.

Mr. CECIL ROWNTREE: This paper affords an excellent illustration of how unsafe it may be to draw conclusions from the study of material obtained from a single source. I was surprised to hear how small had been the incidence of cancer of the ovary secondary to cancer of the breast. The explanation probably is that cases of cancer of the breast in an advanced stage are rarely admitted to the wards of a general hospital. My own experience, gained by long association with the Middlesex Hospital and its special cancer wards and with the Cancer Hospital, has convinced me that cancer of the ovary is secondary to cancer of the breast more commonly than to cancer of any other organ. I have no figures to put before you, but my impression is that our post-mortem results show such a preponderance of cases secondary to the breast, that this organ provides as many secondary tumours in the ovary as all others put together. With regard to the actual mode of growth I have been very much struck by the luxuriance of the growth on the ovary itself as compared with the rate of growth of the nodules on the neighbouring peritoneum. It is quite usual to see a mass, 2 in. in diameter, growing from each ovary with dozens of tiny nodules no more than a few millimetres in diameter scattered round it on the peritoneum. I have often wondered whether this is due to the fact that the ovarian epithelium provides especially favourable conditions for the growth of the cancer cell or whether the endothelium of the peritoneum is especially inimical to its growth, for the presumption is that all the pelvic structures are inseminated by cancer cells to an equal extent.

The paper was also discussed by Dr. CUTHBERT LOCKYER and Dr. H. RUSSELL ANDREWS.

Mr. GORDON LEY (in reply) thanked Dr. Russell Andrews and Dr. Cuthbert Lockyer for their very kind remarks on his paper. With regard to what Mr. Rowntree had said, he reminded him that the material of a hospital post-mortem room was very different from the material of a cancer ward, and that both, particularly the latter, had their inaccuracies.

8

Section of Obstetrics and Gynæcology.

President—Mr. J. D. MALCOLM, F.R.C.S.Ed.

Specimen of Douche Nozzle removed from the Female Bladder.[1]

Exhibited by T. G. STEVENS, M.D.

THIS is the third case of the kind which had come under my care. The patient was admitted to the Hospital for Women, Soho Square, in December, 1919, with the history that the douche nozzle had been in the bladder since September, almost three months. The patient was giving herself a vaginal douche with this nozzle loosely fixed to a Higginson's syringe and passed up the urethra by mistake. Owing to the pain of the passage the syringe was hurriedly jerked away, leaving the nozzle in the bladder. As usual in these cases, the story was hardly credited, but the nozzle was felt at once on passing a sound into the bladder. There was a very severe cystitis; urine could only be retained a few minutes and was foul and ammoniacal and contained a large amount of pus and mucus.

I removed the nozzle by incising the bladder *per vaginam.* The incrustation with phosphatic crystals made the nozzle so rigid that it could not be turned round in the bladder and consequently it had to be cut in half before it could be removed. In spite of the severe cystitis the bladder was sewn up at once with catgut, the vagina having been separated $\frac{3}{4}$ in. all round the opening. The vaginal incision was sutured separately and a self-retaining catheter was placed in the bladder. The catheter was removed on the tenth day, the urine being then almost free from pus, and the incision appeared to have healed. A few days later a doubtful leakage occurred, but after that there was no further trouble. The patient left the hospital three weeks after the operation, able to hold urine all night without inconvenience.

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[1] At a meeting of the Section, held April 8, 1920.

The other two cases were much more recent in that the nozzle had only been in the bladder three days in each case. The severe cystitis in the present case made no difference to the healing of the bladder incision.

DISCUSSION.

Dr. AMAND ROUTH: I think Mr. Stevens' method of removing the vaginal nozzle from the bladder is the best one. If the bladder and vagina are aseptic the incision heals spontaneously.

Dr. HERBERT SPENCER: I think Mr. Stevens did right in incising the bladder from the vagina rather than from the abdomen. But it might have been possible to remove the gum-elastic nozzle through the urethra by means of a Kelly's tube, cutting the nozzle in two with scissors or a wire.

Dr. LAPTHORN SMITH: I would like to commend very heartily the method adopted by Mr. Stevens in removing the douche nozzle from the bladder. The two other alternatives—namely, by dilating the urethra and by suprapubic cystotomy—have both serious objections. The tube was incrusted with phosphatic deposit, and in dragging it through the urethra the latter would almost surely have been lacerated and rigors might have followed. A very considerable amount of dilatation would have been required, after which paralysis of the sphincter vesicæ with incontinence of urine might have followed. The suprapubic route has less objection, but in view of the septic condition of the bladder, and the chance, however slight, of making a slight opening of the peritoneal cavity leading to peritonitis, makes the vaginal route greatly preferable. Even if the peritoneum is not injured, as it should not be, still there is an almost inevitable infection of the incision, especially if a drainage tube has to be left in the bladder until the cystitis has been cured. The vaginal route has the very great advantage of drainage by gravity if the opening in the vagina and bladder is left open with a drainage tube in it. Although the most recent text-books say that the greatest difficulty arises from the opening closing up too soon, I have known cases in which a vesico-vaginal fistula has failed to close over and over again in the most expert hands. I had one such case which had failed five times before I saw the patient, and which failed the sixth time in my hands owing to the catheter which had been tied in slipping out in the night. When the bladder distended the stitches cut through the scar tissue. The experience of that case led me to devise a new method and to search many instrument shops for a better self-retaining catheter. Not till I had found one did I operate for the seventh time, and with complete success. I may add in passing that last year I had need of such an instrument, and it took me nearly a month of hunting in London to find one. It is such a precious instrument that I would like to show it at the next meeting. Since I have used it and no other I have never failed to close a vesico-vaginal fistula. The method of operating which I employed was to make the incision in the vagina half an inch to the right of the middle line, and after carefully separating the vagina from the bladder over an inch and a half of surface to make the incision in the bladder half an inch to the left of the middle line. In a case like Mr. Stevens' one only has to bring the two openings opposite each other and remove the foreign body through an ample opening to avoid injury when the valve-like flaps at once slide back to their original position. Even without stitches the valvular opening would close without leakage, but in this I take no chances, but carefully close the bladder with a fine running suture of six-day catgut through the muscular wall only. Then the vagina is closed with another running catgut suture, and finally the vagina and bladder are brought firmly together with one or two medium silkworm gut sutures which have been introduced before the two rows of catgut are placed, but which are not tied until the last. In the case of a fistula with scar tissue around the fistula this is, of course, removed before any sutures are introduced. Then comes what I consider the most important step of all, the insertion of a glass self-retaining catheter with a fine rubber tubing on it, which conducts the urine drop by drop as it is secreted, into a chamber under the bed. By this means the bladder is at rest and empty for a week, there never being as much as five drops in it at a time.

Mr. STEVENS (in reply): I agree that if possible foreign bodies should be removed from the bladder via the urethra. In the case of bodies as long as 6 in. this has been proved to be impossible, as they cannot be turned in the bladder so as to draw them out by the narrower end. I do not see how a Kelly's speculum could be used, as a pair of scissors could not be manipulated to cut the foreign body in half in the narrow space available. Therefore in each of these cases I have found vaginal cystotomy the best method and the one least likely to do damage to the bladder.

Cystic Embryoma of Ovary.

By J. D. BARRIS, F.R.C.S.

THE specimen was removed by my friend and colleague, Mr. Roberts, at St. Bartholomew's Hospital on February 25, 1920, from a woman aged 27, who had been married one year and had not been pregnant. Menstruation was unaffected, the last regular period ended on February 1. The operation was performed on account of repeated attacks of abdominal pain since Christmas, 1919. On opening the abdomen he found and removed a multilocular pseudo-mucinous cyst of the right ovary about the size of a man's fist. The cyst was surrounded by many adhesions that glued it to the back of the uterus and the right side of the pelvis. The appendix, which showed chronic inflammatory changes, was also adherent to it.

The specimen I show to-night consists of the distal portion of the left Fallopian tube and the left ovary. The left ovary has been laid open, and shows an upper cystic and a lower solid portion representing the unaffected part of the ovary. The cystic portion in the recent state was about the size of a small hen's egg. It will be noticed that it has considerably shrunk in the process of preserving the specimen. It was lined by dark adherent blood-clot, and contained, lying freely within it when it was first opened, a small oval yellow structure, attached at one end by a narrow pedicle to the inner wall of the cyst. I am unable to give the precise spot of its attachment, as the pedicle broke when the cyst was opened. This oval structure has been laid open and will be seen to contain sebaceous material and a few hairs.

Microscopic section of this structure shows a fibrous tissue wall lined by a thin layer of squamous epithelium; the containing cyst wall is composed of ovarian tissue lined by a thin layer of compressed connective tissue and there is no definite cell lining.

The specimen appeared worth bringing before your notice for two reasons: Firstly, it is unusual in that it represents a dermoid, which is pedunculated; secondly, this pedunculated structure was situated within a cyst cavity lined by old blood-clot.

I shall be grateful to know whether any other members of this Society have come across a pedunculated dermoid and also for their opinions as to the nature and origin of the cyst cavity surrounding it.

Dr. HERBERT SPENCER: This specimen would appear to be very rare. Amongst about sixty dermoid tumours of the ovary removed I have not met with a case in which the dermoid cyst was intracystic and pedunculated. Possibly the ovarian tumour was a composite tumour; of these I have seen a few examples, and the cyst enclosing the dermoid cyst may have resulted from the breaking down of septa of the multilocular portion.

Case of Combined Antepartum Hæmorrhage: Placenta Prævia and Accidental Hæmorrhage.

By EARDLEY HOLLAND, F.R.C.S.

PLACENTA prævia is said to occur once in one thousand cases, and accidental hæmorrhage, if examples of slight partial separation of the placenta are included (such as cases of isolated retroplacental hæmatoma), once in five hundred cases. The combination of the two should, therefore, occur about once in half a million cases. I have never before heard of a case showing a combination of the two.

The patient, a primigravida aged 28, at the full term of an apparently normal pregnancy, was admitted to the City of London Maternity Hospital on January 20, 1920. She began to bleed, without any obvious cause, at 10 a.m., and was sent into hospital by her doctor at 2.30 p.m. Her pulse was 80 and of low tension, and her temperature 97° F.; she seemed more anæmic than was warranted by the statements about the amount of blood she had lost. On examination the cervix admitted two fingers, and the placenta could be felt lying in the lower segment with its edge just overlapping the margin of the cervix. A catheter specimen of urine contained albumin in a dense cloud. She was not bleeding on admission, so she was given morphia and watched. I saw her at 5 p.m., and was able to confirm the diagnosis of placenta prævia; at the same time I was struck by the physical character of the uterus, which was hard and irritable, although the patient was having no labour pains. This, in conjunction with the general condition of the patient, with the albuminuria and with the absence of fœtal heart sounds, in a case of *antepartum* hæmorrhage would have led without hesitation to the diagnosis of accidental hæmorrhage, had not the placenta been plainly felt in the lower uterine segment. At the time I did not suspect the combination of the two forms of hæmorrhage.

As the head was presenting, treatment by external podalic version and bringing down a leg was decided on. External version failed, because of the hard, irritable state of the uterus, so I did internal version and brought down a leg, on which continuous weight-traction was made. The patient delivered herself of a dead fœtus at 8.30 p.m. The placenta came away almost immediately, together with a large dark clot of a volume of about 14 oz.

Examination of the placenta revealed a typical, sharply localized, depressed area, with flattened cotyledons, in the middle of the maternal surface and occupying about one-third of its area. Some dark clot was still occupying parts of this depressed area, and a closer inspection revealed some adherent laminæ of clot of greater age. The opening in the bag of membranes was at the margin of the placenta, adjacent to a tongue of blood-infiltrated placenta typical of the separated area of a placenta prævia. The albumin quickly diminished in amount after delivery, and had disappeared from the urine by the eighth day post partum.

I realize that, in certain cases of placenta prævia, concealed intra-uterine hæmorrhage, retroplacental and otherwise, does occur, owing to the fact that the presenting, or compressing, part of the fœtus does not completely compress the separated area of placenta. That the present case was not of this nature is proved by the fact that a zone of normal placenta intervened between the separated prævial area and the localized depressed area occupied by the retroplacental clot.

This was a true case of placenta prævia combined with toxæmic retroplacental hæmorrhage. There is no inherent reason why such a combination should not occur; according to the laws of chance, it should occur once in every half million cases of pregnancy, or, according to the present birth-rate, twice per annum in the United Kingdom.

Dr. HERBERT WILLIAMSON: I have seen one similar case. The patient was a primigravida, and when admitted into hospital was semi-conscious, and losing profusely from the vagina. There was general œdema and the urine contained a large amount of albumin. She died shortly after admission and before delivery. Post mortem the placenta was found situated partially over the lower uterine segment, both its lower and its upper portions were detached and the uterus contained a large mass of blood-clot.

Specimen of Infected Fibromyoma of the Puerperal Uterus associated with General Peritonitis and removed by Hysterectomy.

By EARDLEY HOLLAND, F.R.C.S.

THE patient, a multipara, aged 35, was admitted to the London Hospital on November 19, 1919. She had been delivered three weeks previously of a child which weighed 6½ lb., and lived only one day; the uterus, after delivery, remained large, the placenta did not come away, so a twin was diagnosed and the patient left to nature. However, at the end of three days her doctor removed the placenta manually, and diagnosed a fibroid tumour. The patient did not become acutely ill until two days before admission, when she began to get acute abdominal pain, vomiting and high temperature.

On admission she was acutely ill, and presented the signs and symptoms of an acute generalized peritoneal infection. A tender, hard, round tumour was felt rising out of the pelvis, the size and shape of a uterine pregnancy of twenty-four weeks' duration. I operated at once,

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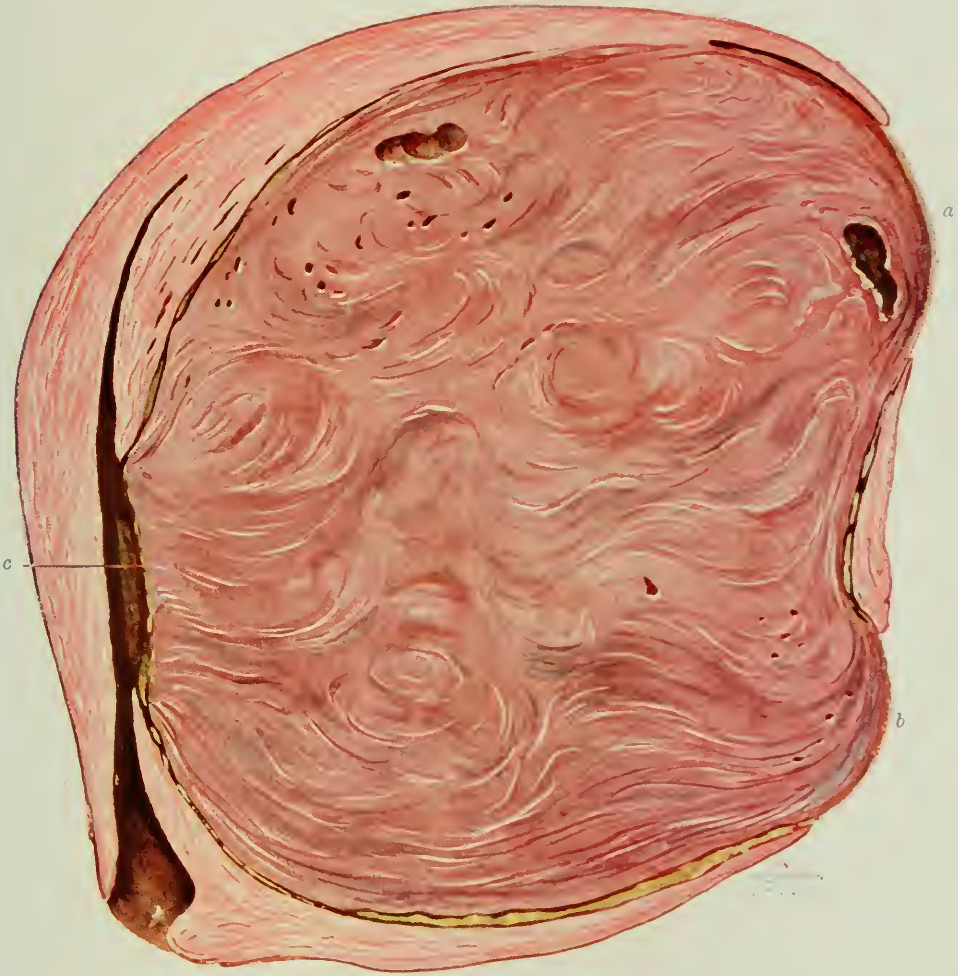

Hemisection of infected puerperal uterus, with a necrobiotic fibromyoma in anterior wall. Note pus-track between tumour and capsule, and three perforations of capsule, one into uterine and two into peritoneal cavity.

HOLLAND:

Specimen of Infected Fibromyoma of the Puerperal Uterus associated with General Peritonitis and removed by Hysterectomy.

found generalized peritonitis, and removed the enlarged uterus by subtotal hysterectomy. The anterior surface of the tumour presented two perforations, from which pus was oozing. The patient died in thirty-six hours.

A post-mortem examination was made the next day, of which the following are the notes: "Drainage of abdominal cavity through slit cervix uteri and vagina, and also through a laparotomy wound. Dilatation and purulent inflammation of lower 3 cm. of cervical canal. Slight laceration of the lower vagina. Large quantity of fibrinous deposit with little pus all over abdominal cavity. Anæmia and slight cloudy swelling of myocardium. Slight atheroma; few small fatty plaques in aortic commissure, with very few fat flecks in rest of aorta, coronaries and carotids. Partial compression collapse of inferior parts of lower lobes of lungs. Slight acute diaphragmatic pleurisy in both lower lobes. Caseous calcareous tubercular nodule in right paratracheal gland. Marked anæmia with some fatty infiltration of liver (3 lb. 3½ oz.). Œdematous spleen (5¼ oz.) with some greyish hyperplasia of pulp. Anæmia and severe cloudy swelling of kidneys (10 oz.). Marked general anæmia. No physiological activity in fibrous breasts. No pigmentation of nipples. Caries of teeth. A thin, normally developed woman."

Description of the Specimen.—The uterus was hardened in formalin and then bisected sagitally. The uterus is almost spherical in shape, with a diameter of 17 cm. The whole anterior wall is occupied by a single, spherical, interstitial fibromyoma, 14·5 cm. in diameter (Plate). The fibromyoma is foul-smelling, and its cut surface has a dirty greyish-pink colour, and a tough whorled surface with a few small areas of myxomatous cystic degeneration in it. The flattened, elongated uterine cavity, 13 cm. long, is spread out on its posterior surface. The muscular capsule of the tumour is thin. At the lower end of the uterus is the cut surface of the cervical canal. The peritoneum covering the uterus is white, with a deposit of yellow fibrin. In the middle of the anterior wall of the uterus (Plate, *a*, *b*) are two ruptures, or perforations, of the whole thickness of the uterine wall (i.e., capsule of the fibromyoma), 3 cm. and 3·5 cm. in diameter respectively. Through these perforations project part of the underlying fibromyoma, covered with a yellow slough. Through the capsule of the posterior wall of the fibromyoma is another perforation, 5 cm. in diameter (Plate, *c*); as this part of the capsule forms the anterior wall of the uterine cavity, the perforation is naturally through the endometrium and into the uterine cavity. Between this perforation and the already mentioned two perforations in the anterior wall is a continuous purulent track lying between the fibromyoma and its capsule, providing a direct communication between the cavity of the uterus and the general peritoneal cavity. The endometrial surface is brown and ragged ; the placental site cannot be identified with certainty, but it appears to have been in the anterior wall, i.e., over the posterior aspect of the fibromyoma.

The special point about the specimen is the occurrence of perforation of the capsule of the tumour, on the one hand into the infected uterine cavity, and on the other hand into the general peritoneal cavity, forming a direct track of infection leading to general peritonitis. The perforations are no doubt due to acute necrosis from a virulent infection acting on the capsule, rendered tense by the rapid swelling of the fibromyoma it enclosed.

The PRESIDENT : I suggest that the contraction of the uterine wall would increase the effect of the inflammatory swelling of the septic tumour in favouring a necrotic change in the uterine wall.

A Report upon Two Specimens showing : (1) A Fibro-myo-lipoma of the Uterus ; (2) Extensive Fatty Degeneration of a Uterine Fibromyoma.

By HERBERT WILLIAMSON and R. ST. L. BROCKMAN.

THE first specimen is No. 3001 in St. Bartholomew's Hospital Museum. The description in the catalogue is as follows : "A pedunculated fibrous tumour of the uterus with a small well-defined, encapsuled fatty tumour embedded in it near the centre of the mass. It was removed by operation from a woman, aged 50. Its growth was not accompanied by any symptoms, but it suddenly protruded from the vagina during an action of the bowels."

The specimen was exhibited at a meeting of the Pathological Society of London in 1861 by the late Sir Thomas Smith, who described it as "an adipose tumour the size of a pigeon's egg, completely embedded in the substance of a larger tumour, and surrounded by a fibrocellular capsule from which it was easily shelled out."

As far as we have been able to ascertain, the specimen is unique amongst uterine fibro-myo-lipomata in that it is encapsuled and

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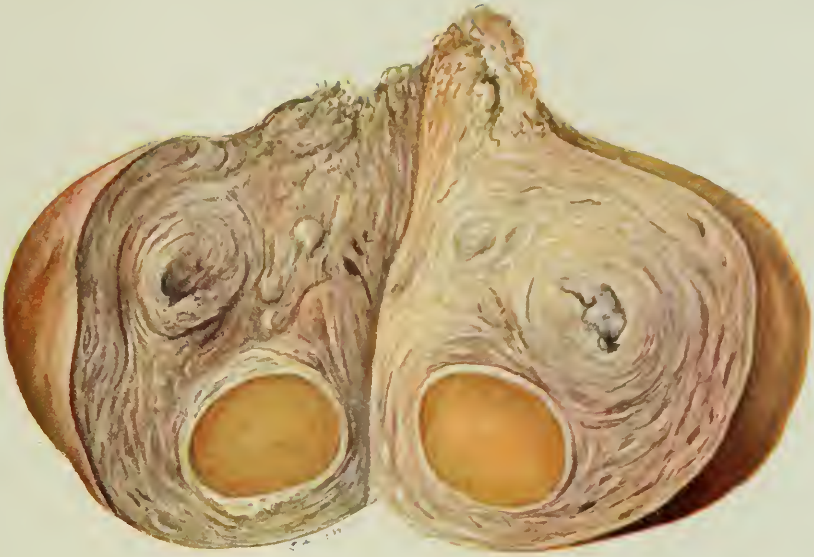

Fig. 1.
Encapsuled fibro-myo-lipoma of the uterus.

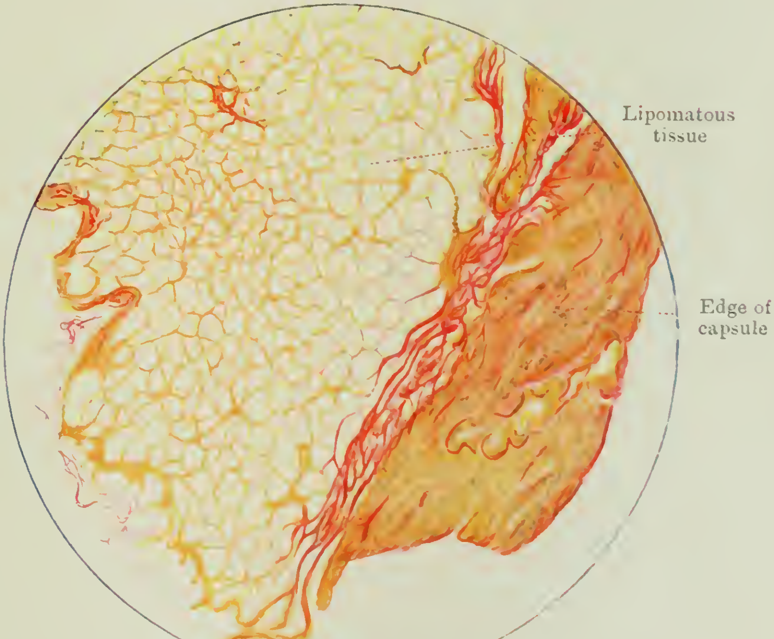

Fig. 2.
Microscopic section of encapsuled fibro-myo-lipoma of the uterus.

WILLIAMSON and BROCKMAN:
A Report upon a Specimen showing a Fibro-myo-lipoma of the Uterus.

clearly defined from the surrounding fibromuscular stroma. As it is often referred to in the literature, and as no account of its microscopic appearance has hitherto been published, we are exhibiting it to-night. The specimen is an ovoid tumour, measuring 7 cm. by 6 cm. by 9 cm. At the upper extremity are seen the remains of a pedicle by which it was attached to the uterine wall; the surface is for the most part smooth, but one or two rounded lobules project near the lower pole. It has been bisected, and the cut surfaces show a dense fibrillated stroma, and embedded near the centre of the lower half a yellow encapsulated mass measuring 3 cm. by 2·5 cm. The mass is separated by a definite white capsule from the surrounding tissues, it has the characteristic appearance of fat, and oil globules can be scraped from its surface. A little above this mass is a small irregular cavity, 1 cm. by 1 cm., which in the recent state is said to have contained "serous fluid" (*see* Plate, fig. 1).

Microscopical Appearances.—The sections have been difficult to prepare because the specimen has been immersed in spirit for nearly sixty years, and the tissues have become very hard. The yellow mass is composed of a network of fibromuscular strands, and between these strands of tissue are fat cells. The fat cells are rounded and polygonal, and, where the fat has been dissolved out by the reagents used in preparing the sections, appear as spaces surrounded by a thin envelope of cell substance, in which a nucleus is embedded. The masses of fat cells are separated by strands of fibromuscular tissue, which van Gieson's stain shows to consist of muscle bands with a small proportion of collagen fibres. These bands show hyaline changes but no fatty degeneration. In sections stained by Sudan III fat can still be seen occupying the interior of the rounded cells just described. A section through the capsule shows that it is composed of fibromuscular tissue (*see* Plate, fig. 2).

We have found descriptions of only six similar tumours :—

(1) Seegar (1853).[1] A widow, aged 53, had suffered from severe hæmorrhage for a year. A tumour, the size of a child's head, was found protruding from the vulva. The pedicle was ligatured and the mass removed. It consisted of "a fatty tumour covered by a firm cellular membrane and traversed by fibrous tissue bands. Parts of the tumour were soft, others firm and poor in blood-vessels." No microscopic description is given.

(2) Paget (1861).[2] This is the specimen shown to the right.

[1] *Zeitschr. f. Wundaerzte u. Geb.*, 1853, v, p. 24.

[2] (Sir) T. Smith, *Trans. Path. Soc. Lond.*, 1861, xii, p. 148.

(3) Kelly and Cullen (1894).[1] The patient was aged 50, but no details of the clinical history are given. The uterus measured 32 cm. by 32 cm. by 36 cm. The enlargement was due to an interstitial myoma. Microscopic examination showed hyaline degeneration and small areas of adipose tissue; the latter consisted of groups of three to eight or more fat cells principally in close proximity to the vessels. A drawing of the microscopic section is given on p. 164 of the authors' book.

(4) Kelly and Cullen (1895).[2] The patient was aged 29, but no further clinical details are given. The uterus was large and multinodular, the largest myoma measuring 13 cm. by 20 cm. by 28 cm. Some of the nodules show much hyaline degeneration with liquefaction. In several sections small groups of fat cells were found. Microscopic drawing on p. 165 of the book quoted.

(5) Knox (1901).[3] The patient was aged 63 and the mother of thirteen children. The menopause occurred at the age of 50; three years later a slight serous vaginal discharge was noticed which soon ceased, but returned again after an interval of eight years. For two weeks before operation there had been moderate but continued hæmorrhage. After hysterectomy a tumour, 10 cm. by 10 cm. by 13 cm., was found in the posterior wall of the uterus. On section the cut surface showed a network of white glistening bands and in the interstices yellow areas. Microscopic sections showed collections of large fat cells enclosed in a supporting framework of smooth muscle and fibrous tissue. Drawings of the tumour and microscopic section given.

(6) Gordon Ley (1914).[4] The patient was aged 54 and had borne children. The menopause occurred six years previously, and one year before operation bleeding commenced and gradually became more continuous. After subtotal hysterectomy a tumour, 12 cm. in diameter, was found embedded in the posterior wall of the uterus composed of nodules—some white, some white with yellow streaks, some yellow. Microscopic examination showed that for the most part the section was made up of fat envelopes separated by narrow strands of collagen fibres and muscle. Drawings of microscopic sections given.

(7) Lockyer (1918).[5] The patient was aged 51 and had borne one

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[1] "Myomata of the Uterus," p. 162.

[2] Loc. cit.

[3] *Johns Hopkins Hosp. Bull.*, xii, p. 318.

[4] *Proc. Roy. Soc. Med.*, 1913-14, vii (Sect. Obst. and Gyn.), p. 150.

[5] "Fibroids and Allied Tumours," 1918, p. 65.

child. Uterine hæmorrhage commenced three years before operation; the losses were frequent and profuse. Pan-hysterectomy was performed. The uterus contained two tumours, one in the body the other in the posterior wall of the cervix. Fat was present in the cervical tumour only. The author says "the sections show large fat globules within muscle fibres . . . but the large globules of fat are quite different from the small granules seen in fat necrosis." Colour illustrations of the tumour and sections are given.

Dr. Lockyer generously presented one half of his specimen to St. Bartholomew's Hospital Museum, and we have cut other sections. These sections show typical signet-ring fat cells in interstices between fibromuscular bands. We saw in addition large globules of fat as shown in the illustration in Dr. Lockyer's book, but our impression is that they are not within, but lying upon, the muscle cells, and consist of fat liberated from the lipoma cells in the preparation of the section. We regard this tumour as a true fibro-myo-lipoma.

We think these tumours should be described as fibro-myo-lipomata, because this name is a statement of fact and involves no theory of origin. Ley uses the term "lipomatosis," and states that the distribution of the lipomatous tissue and the absence of young fat-granule cells are strongly in favour of a fatty metamorphosis of the fibrous stroma. Lockyer prefers the term "fatty metamorphosis": he believes that the metamorphosis chiefly affects muscle cells, and regards the presence of the fat as "evidence of vital but depraved activity." We find no conclusive evidence to prove either of these views, but see no difficulty in accepting them, since the metamorphosis of the muscle and fibrous tissue cells of fibromyomata into sarcoma cells has been proved. Neither Ley nor Lockyer, however, has described transition forms between the characteristic fibrous tissue or muscle cell and the typical fat cell, and in the absence of such transition forms their thesis cannot be taken as proved. Knox evidently regards fibro-myo-lipomata as mixed tumours and remarks that the presence of lipomatous tissue in fibromyomata supports the theory of Cohnheim which attributes their development to embryonal tissue-rests.

From the clinical point of view it is interesting to note that with one exception (Case IV) all the patients from whom the tumours were removed were over fifty years of age, and in the majority of patients bleeding after the menopause was the symptom which made them seek advice.

Addendum.—Since the above was written our attention has been called to a specimen, No. 1419.1, in the museum of the Royal College of Surgeons. It is thus described in the catalogue: "A uterus in the wall of which there has grown a spheroidal tumour 10 cm. in diameter. Except at its lower end the growth, which has been bisected and opened out, and from which the uterine tissue has widely retracted, consists almost solely of fat. A myo-lipoma. Microscopic examination shows that the fat has been formed in the connective-tissue between the muscle cells, the bundles of which are widely parted as a result." The tumour was removed by Mr. Albert Carless from a woman, aged 66, who had never borne children. The tumour had been known to exist for sixteen years, but had never caused symptoms. Unfortunately the microscopic section cannot be found.

The second specimen is of a totally different nature and is an instance of extensive fatty degeneration. Such extensive degeneration is rare in fibromyomata and we show the specimen not merely as a contrast to the last but also on account of its intrinsic interest. The tumour was removed from a woman aged 39. She has been married for eleven years but had never been pregnant. For three months before operation she had suffered from severe menorrhagia but had no other symptoms. The uterus after removal by subtotal hysterectomy was enlarged to a size of 16 cm. by 10 cm. by 14 cm., by the presence of multiple fibromyomata.

The specimen shown is a transverse section through the mass; the cavity of the uterus is seen in its centre. Multiple fibromyomata are present in the uterine wall, and in the recent state the largest of these appeared to consist of yellow fat, whilst oil globules could be scraped from its surface and with Sudan III it stained a sunset-red. Unfortunately in the preparation of the specimen most of the fat has been dissolved out and these appearances have been lost. It can be seen however that its tissues are semi-translucent and consist of a white fibrous-looking framework with yellowish-pink material in the interstices. A second smaller nodule situated below the larger tumour and more deeply in the uterine wall shows a ring of yellow homogeneous fat-like material.

The microscopic sections exhibited stain very faintly with hæmatoxylin and show hyaline degeneration with fragmentation and disappearance of nuclei. These hyaline areas are infiltrated with fat which is seen in the form of small globules lying both within the remains of the muscle cells and in the hyaline substance without them. The deposit of fat is usually absent in the immediate vicinity of the blood-vessels. Fat cells are not found in any part of the sections. Fatty degeneration, sufficiently extensive to be visible to the naked eye, is rare in uterine fibromyomata, and very few instances have been placed on record. Cullen has given in one short paragraph the essential features of the process: "In nearly every myoma there is a hyaline degeneration in some part of the growth. This is usually diffuse but in a certain percentage of the cases a large circumscribed area undergoes this degeneration. In the centre of such a block of hyaline tissue there is a gradual melting away and we find nothing but a few threads of connective tissue and, in the spaces between these, free fat looking very much like melted butter. In this free fat cholesterin crystals are frequently present."[1]

DISCUSSION.

Dr. LOCKYER: Four of the examples of fatty metamorphosis of fibroid tumours mentioned by the authors are cited in my monograph on "Fibroid and Allied Tumours." From investigations carried out on the specimen which I removed myself, I came to the conclusion that the condition was not neoplastic—i.e., that the fat present did not represent a lipoma but was an example of metamorphosis. I further convinced myself that the change affected the muscle fibres, because, in some of the smaller and more discrete fat-globules, oval bodies, stained by logwood, which resembled muscle nuclei, were present. In the larger globules, which were massed together, these "nuclear" bodies were not to be seen. I regarded the "nuclei" in the globules as evidence of a transition between muscle-tissue and fatty tissue. This investigation was carried out in 1914, and the slide from which the drawings were made has been lost. Dr. Williamson appears not to have found the "nuclear" bodies in question, so the point whether my observations were correct or rightly interpreted remains at present *sub judice*. Nevertheless, I do not share Dr. Williamson's opinion that the term "lipo-fibromyoma" is non-committal since it implies, to my mind, that the fatty deposit is of the nature of a new growth and will be accepted as such. I used the term "metamorphosis" in order to distinguish this condition from the fatty degeneration which follows on hyaline change, and which leads up to red, yellow, and white necrosis. In such cases the degeneration-product is in the form of fine lipoid granules, no large sunset-red globules and signet rings are to be found.

Mr. T. G. STEVENS: I see no difficulty in regarding this specimen as an unusual example of fatty metamorphosis in the connective tissue cells of a fibromyoma. I cannot see any reason for regarding the lesion as a new formation—a lipoma—even though it forms an encapsuled mass. The encapsulation seems to be more apparent than real, as strands of fibrous and smooth muscle tissue can be seen running into the fatty mass from the periphery. Such a growth as a myo-lipoma is unknown, and this specimen certainly consists of fat, smooth muscle and fibrous tissue. It ought to be placed in the same category as the other specimens which have been described, and in which the fatty change was diffused through the tumour.

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[1] *Johns Hopkins Hosp. Bull.*, 1901, xii, p. 321.

The PRESIDENT: The naked-eye and microscopic appearances of Paget's case seem to indicate so clearly that there was a new growth of fatty tissue that I cannot avoid the conclusion that in that case there was a true fatty neoplasm, even although the origin of the fat cells is obscure. Such cases are very rare, and I agree that further light is necessary before a final opinion can be expressed.

Dr. WILLIAMSON (in reply): When Dr. Lockyer has examined the sections we have cut from a different part of his specimen I have no doubt that he will agree that it belongs to the group of tumours we have been describing to-night. We prefer the term fibro-myo-lipoma to others suggested, because it is a simple statement of fact; these tumours consist of fibrous tissue, muscle cells and fat cells: these facts and nothing more than these facts are indicated in the name. The fat cells may be formed by metamorphosis of muscle or fibrous tissue cells, or they may be new growths. At present we do not know which view is correct, but doubtless further research will clear the matter up.

Case of High Ammonia Coefficient.

By Lady BARRETT, C.B.E., M.S.

THE patient, Mrs. W., aged 22, munition worker, was transferred from the Medical Wards of the Royal Free Hospital to the Maternity Block on August 29, 1918, suffering from persistent vomiting. Her last regular period began on May 25, 1918.

The patient first felt ill on the night of July 14. On this day she started night work at the factory and felt ill and faint throughout the night. The following evening she was unable to go to work as she was vomiting every five minutes. Her doctor diagnosed the case as a recurrence of "Spanish 'flu" which she had had a fortnight before and recommended her to go to the Royal Free Hospital. On July 27 she was admitted to the hospital suffering from persistent vomiting. The vomiting was treated by dieting, with occasional success, the longest interval without it being three days. She was very constipated.

On August 24 the vomiting became more severe and on August 29 diacetic acid appeared for the first time in the urine; on this day she was transferred to the Maternity Block for suspected pernicious vomiting of pregnancy.

On August 29 the note on admission was as follows: The patient looks unhealthy. She has severe acne rosacea with the usual distribution: the conjunctivæ are slightly yellow. She is fairly well nourished, but has not been weighed recently. She lies motionless on her back, moving only her head. She feels weak and ill and wants to vomit continually. Temperature 97·4°F., pulse 84. Sleep the first night was fairly good. The lips were dry, the tongue furred and greenish-yellow and vomiting was frequent after food and at other times. The bowels were very constipated. There had been amenorrhœa since May 25. On examination of the abdomen there was slight tenderness at the epigastrium and the colon could be felt loaded with fæces. The liver dullness was diminished, but it was doubtful whether this was only apparent through distension of the colon.

Examination *per vaginam*: The cervix and vaginal walls were soft. The uterus was enlarged, two and a half to three months pregnant, and was retroverted. Nothing abnormal was found in heart or lungs. *Urine*: Seven ounces of urine were passed in the first twenty-four hours. The colour was reddish brown, reaction strongly acid, specific gravity 1022. Diacetic acid was present and there was a faint trace of albumin. There was no blood, pus, sugar or bile salts.

For the first two days, August 29 and 30, the patient took 2 oz. of Benger's food three hourly without vomiting. She was still only passing 7 oz. of urine in twenty-four hours. The retroversion was corrected and a pessary inserted. On August 31 she vomited twice; the urine was diminished to 5 oz. the twenty-four hours. She was ordered a mag. sulph. and glycerine enema daily, which was effective. A rectal glucose saline of 1 pint was given six hourly and she took 3 oz. of Benger's and 3 oz. of coffee alternately by the mouth. The next day she vomited ten times. The urine was increased to 24 oz.; the ammonia coefficient was found to be 38·1.[1] She was now given only glucose saline *per rectum* with 1½ oz. of brandy in twenty-four hours.

On September 2 she still vomited frequently and had acute abdominal pain in the upper part of the abdomen. She was very thirsty. She was allowed to take 2 oz. of albumin water two hourly by the mouth.

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[1] All the estimations of the urine in this case before delivery of the child were made by Miss E. E. Hewer, M.Sc., London University, Lecturer in Histology and Demonstrator of Physiology (Royal Free Hospital), School of Medicine for Women.

Later in the day she felt greatly relieved and was allowed 2 oz. of tea and 2 oz. of albumin water alternately two hourly. The rectal salines were continued. On September 3 the quantity of urine passed was 16½ oz.; all food was retained. On September 4 the quantity of urine was 8¼ to 9 oz.; the ammonia coefficient had risen to the astonishing height of 78 per cent. She was allowed water and oranges, bovril, tea, pounded chicken, bread and butter and toast.

From that time on she vomited occasionally once or twice in the day, but within the next few days the diet was increased, chicken, baked apples, oranges and milk being allowed, and in addition 5 oz. of fluid of one kind and another was given three-hourly. The ammonia coefficient still remained as high as 50, but as the vomiting was diminished she was allowed a still more nitrogenous diet, fish, chicken, mutton broth and one egg daily, with the idea that a more generous intake of nitrogen might result in a more normal type of excretion. This proved to be the case. From that time the vomiting practically ceased, the ammonia coefficient gradually, as will be seen from the chart, declined and by September 11 all food was retained. She was then passing 20 oz. of urine daily. On the 17th of the month, however, vomiting again returned. The quantity of urine passed was then 45 oz. The ammonia coefficient began again to rise; the diet was somewhat reduced and the condition soon improved. She was now taking sulphate of magnesia and cascara by the mouth instead of by the enema, and on October 7 she was discharged.

The patient remained very well until the middle of December, when she began to lose her appetite and became very drowsy. Vomiting commenced again on December 25 and was fairly persistent until the 30th, when she was readmitted to hospital. She was then six and a half months pregnant; the child was living. The ammonia coefficient varied between 5 to 7 during her stay in Hospital and she was discharged on January 10.

She was readmitted on February 14 and the child was delivered by a perfectly normal labour on the 15th. The baby was a girl and weighed 5 lb. 9 oz. The baby was very jaundiced for a few days but regained its weight on the seventh day and was doing well when the patient was discharged on March 10.

During 1919 the patient remained well and her ammonia coefficient varied between 5, 6, 7 or 8. In December, 1919, she was found to be again pregnant and the condition of her urine has been watched during the four or five months of her present pregnancy. Up to the present time the ammonia coefficient has not been raised above 5 to 7, but with the exception of occasional vomiting once or twice during the day on isolated occasions, she has so far been perfectly well.

I considered the case one of pregnancy toxæmia in view of the diminished quantity of urine, the presence of diacetic acid and albumin from time to time, coupled with excessive vomiting and an extremely high ammonia coefficient. Since, however, every case of toxæmia is not fatal, the patient was carefully watched, induction of labour being postponed from day to day, and the case ended in the delivery of a healthy child. The fact that in the first instance this patient was suffering from what might be called a reflex cause of vomiting—the retroverted gravid uterus—appears to be negligible because its replacement had little or no effect upon her condition. When first admitted she was undoubtedly very ill. Her pulse however was only 84 and uniform throughout and the temperature was normal. Her average normal pulse-rate is 72.

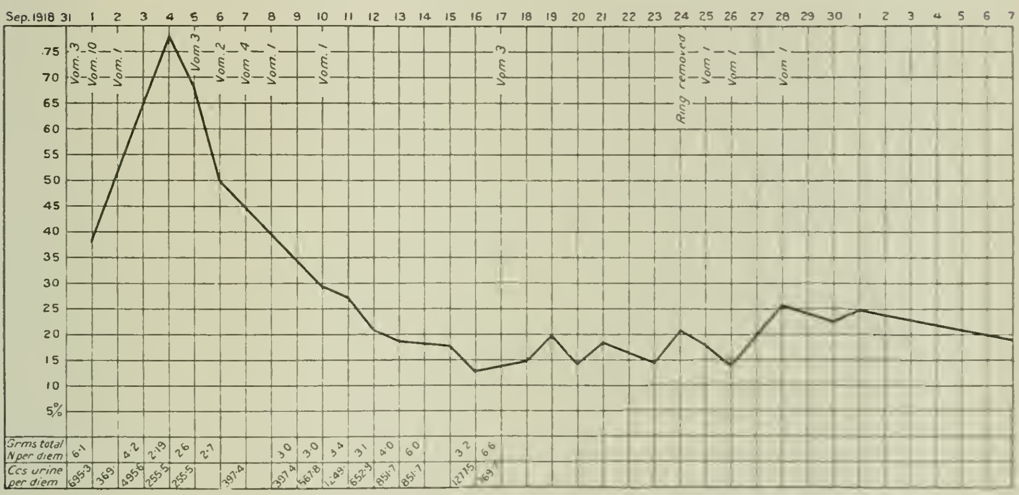

Chart recording the percentage of N—excreted as ammonia—the total excreted N being 100

The case seems to indicate that a consideration of the general condition, especially of the pulse, the quantity of urine passed and the presence or disappearance of diacetic acid in the urine is of more importance than the high ammonia coefficient in deciding the necessity for induction of labour.

I am indebted to Miss Hewer, M.Sc., for the observations made in the physiological laboratory during the patient's first illness and to Mrs. Pillman Williams for observations made during the period between the pregnancies and during the early months of the present pregnancy. Mrs. Williams has also made observations of the different samples of urine passed at various hours during the day, which show an interesting curve of change in the excretion of total nitrogen and the proportion excreted as ammonia during the twenty-four hours.

DISCUSSION.

Dr. MACKENZIE WALLIS: I have been interested in the details of the case submitted by Lady Barrett, more particularly as I have investigated a number of cases of pregnancy toxæmias myself. The point I would raise in discussing the ammonia coefficient is the method used in the determination of the ammonia in the urine, since the possibility of this excess being due to excessive protein breakdown may account for certain discrepancies, particularly if the formol titration method was used. In such a case excess of amino-acids in the urine would be estimated as ammonia. It is to be regretted that the other urinary changes were not determined in this case, particularly the amount of urea, and the diastase content. In pregnancy toxæmias the diastase is always distinctly increased, values of 200 to 500 units being found, in contrast to the normal 10 to 25 units. Further, there was no analysis of the blood for total non-protein nitrogen urea and other constituents which would have served as a control of the urinary changes observed. There does not seem to be any relation between the high ammonia coefficient in this case and the presence of aceto-acetic (diacetic) acid in the urine. In the wards of St. Bartholomew's Hospital I am at present investigating a case under the care of Dr. Williamson, which presents certain points of interest in connexion with this discussion. The patient was admitted with severe and persistent vomiting, and the urine on examination showed a large amount of aceto-acetic acid, an ammonia coefficient of 8 and a diastase content of 50 units. Her condition was so grave that it was decided to terminate the pregnancy, and this operation was followed by rapid improvement in the condition of the patient. The vomiting ceased, and the urine showed no aceto-acetic acid, a coefficient of 5, but the high diastase persisted for a few days. The urinary changes in this case are therefore of a reverse character to that usually found in pernicious vomiting of pregnancy, the aceto-acetic acid being out of all proportion to the ammonia coefficient. As a result of my work I have come to the conclusion that the ammonia coefficient is of no value either for prognostic or operative purposes. It is rarely increased in eclampsia unless vomiting is pronounced, but almost constantly raised in the pernicious vomiting of pregnancy. Again, it does not bear any definite relation to the severity of the toxæmia, or the amount of aceto-acetic acid in the urine. Neither the increased ammonia coefficient nor the aceto-acetic acid present are sufficient to account for the intoxication observed in such cases, but are simply associated phenomena. The effects are due to starvation, brought about by the vomiting, and it does not seem to be fully recognized that a pregnant woman is peculiarly susceptible to the slightest restriction of carbohydrates, and reacts accordingly. In the case described by Lady Barrett, the ammonia coefficient was so high that practically all the nitrogen in the urine might have been in the form of ammonia. I cannot see what value can be placed on such a figure unless we know the changes in the blood. There were only small amounts of aceto-acetic acid detected in the urine, and if the increased ammonia was due to starvation it should have run parallel with the amount of aceto-acetic acid in the urine. The fact that the ammonia coefficient fell directly the proteins in the diet were increased would support the view that the changes observed were due to starvation. Further, the body does not deviate from the normal path of metabolism except for very good reasons, and in this case there was the risk of considerable hæmolysis occurring if a quantity of neutral ammonium salts were circulating in the blood at any one time. That such hæmolysis did not occur in this case is obvious from the condition of the patient and the rapid recovery observed.

Dr. EDEN: I have recently had a case of pregnancy toxæmia with high ammonia coefficient in my ward at Charing Cross Hospital. The patient was in the fifth month of pregnancy and was sent to the hospital by a local practitioner as a case of eclampsia, she having been found in a "faint" by a neighbour. She was not seriously ill at any time during her stay in the hospital, but she had one attack which was observed by the staff nurse and the resident obstetric officer. It consisted of slight muscular spasm, beginning in the left side of the face, this becoming general and followed by some mental dullness and confusion. Her blood pressure was normal and there was no vomiting. The urine contained no albumin and no renal elements. The reaction to acetone bodies was strong, the diastase reaction was 20 per cent., and the ammonia coefficient on the first estimation was 17 per cent. One of my medical colleagues who saw her in consultation considered it a case of acidosis. The blood was not examined. The treatment adopted was a fluid, chiefly milk diet, saline purgation, and large doses of bicarbonate of soda by the mouth. The ammonia coefficient fell to normal within a week and the acetone bodies disappeared from the urine. The ammonia coefficient is a very uncertain guide to the clinical severity of cases of toxæmia. In Lady Barrett's case the severe vomiting may have been an important factor in raising the coefficient, apart altogether from the degree of toxæmia.

Dr. LAPTHORN SMITH: I am greatly interested in Lady Barrett's case report, because some twenty years ago I had within a few months three cases of excessive vomiting of pregnancy sent to me by young friends, or rather I should say I was called in consultation, because the women were unconscious or delirious from starvation and would evidently have died within a few days at most if I had not at once emptied the uterus. The vomiting stopped immediately, as if by magic, but it took weeks and months to nurse them back to health. These three cases led me to study the whole question very thoroughly, and I was surprised to find no less than ninety-three fully reported cases of death from hyperemesis gravidarum in the European literature alone. One of my friends, a professor of surgery, told me that his own wife had vomited steadily during the whole of her first pregnancy, beginning a few days after conception, and was confined to bed the rest of the nine months, vomiting continually until she was delivered. One of the above three cases was that of a widow who had made frantic efforts to get rid of her pregnancy, and her doctor told me she had succeeded, as he had seen the large amount of blood and what he was sure was the placenta. But I felt so sure that the vomiting would have stopped if this were so, that I insisted upon curetting her and brought away the ovum, and next day she was keeping down food. By accident I then stumbled on the bicarbonate of soda treatment of this vomiting for many other cases as they were sent to me during the last twenty years, and I can say that from that day to this I have never seen a case that was not soon relieved by this treatment. Sometimes the vomiting stopped in a few days and even in the worst cases in a few weeks. So that the question of emptying the uterus to stop the vomiting has never come up in any of the cases sent to me since I began it. I have generally prescribed about 80 gr. a day in the form of a mist. rhei et sodæ, with a few drops of chloroform and nux vomica. The moving of the bowels two or three times a day helps the peristalsis of the intestine to revert to the normal. I should be very pleased if Lady Barrett would tell us whether she has had much experience with the bicarbonate of soda treatment and whether her experience has been the same as mine. Twenty years ago we knew little or nothing of acetonuria or acidosis, but the recent researches of physiological chemists go to prove that both the condition under discussion and also the condition of eclampsia are largely due to acidosis and that consequently the alkali treatment is the best as far as drugs are concerned.

Lady BARRETT (in reply): During the patient's first illness when three months pregnant no differentiation was made between the amino-acids and ammonia, but in the later investigations made during her admission in December, 1918, and during the lying-in period in February, 1919, the amino-acids were separately estimated, and it was then found that the average difference between the ammonia and amino-acids combined by the Kjeldahl method and the separate estimation by Folin was approximately 2 per cent. I cannot state, however, whether the difference would have been greater when the coefficient was extremely high. Diacetic acid was only present in traces, and it appeared and disappeared with the presence of a small cloud of albumin. Diastase was not estimated, nor were estimations made from the blood. In Dr. Williamson's case of eclampsia with a high ammonia coefficient, the findings were in many respects different—e.g., diacetic acid was continually present. I agree with Dr. Eden that the excessive vomiting itself may have been a factor in the high excretion of nitrogen as ammonia, because the increase of nitrogen in the diet resulted in a more rapid form of the ammonia excretion than was obtained during the restricted diet. I have used bicarbonate of soda in salines given to toxæmic patients, but without any appreciable difference in the result from salines without carbonate. It may, however, be interesting to note that this patient has been on doses of bicarbonate of soda throughout her present pregnancy and no vomiting nor toxæmic symptoms have developed up to the present time (four and a half months).

A Note on the Value of Blood Transfusion before Operation in Severe Secondary Anæmias.

By HERBERT WILLIAMSON.

THE value of blood transfusion in cases of sudden and profuse hæmorrhage from the uterus has already been demonstrated to this Society by Dr. Russell Andrews and Dr. Gordon Luker. I wish to-night to direct your attention to its value in the more gradually produced anæmia of uterine fibromyomata.

M. M., aged 45, was admitted into St. Bartholomew's Hospital on May 11, 1919. She had been married for eighteen years but had never been pregnant. Menstruation commenced at the age of 13 and was regular and normal in amount until November, 1918. From November, 1918, to April, 1919, there was amenorrhœa. Early in April, vaginal hæmorrhage commenced and soon became profuse; she lost a considerable quantity of blood every day and passed large clots. Towards the end of April she commenced to suffer severe and constant pain over the left iliac fossa. The pain and bleeding continued until her admission into hospital on May 11.

On admission she was very anæmic, restless and breathless; the pulse was 130 and of poor volume, the temperature 101° F.; the tongue was dry and furred. There was incontinence of both urine and fæces and frequent vomiting. The urine contained a cloud of albumin but no renal elements. The blood count was: Red cells, 1,670,000 per cubic millimetre; white cells, 52,000 per cubic millimetre; hæmoglobin, 22·5 per cent.; colour index, 0·7. The patient was so breathless that great care was necessary in moving her and she fainted during examination. The breasts were inactive; a hard tumour was felt rising from the pelvis and reaching nearly to the costal margin; the greater part of the tumour was insensitive, but one portion, situated above the right Poupart's ligament, was tender. The vagina contained blood clot, the cervix was high up, near to the symphysis pubis and behind it was a rounded cystic tumour.

From the 11th to the 17th, the patient's condition gradually became worse, but I was afraid to remove the tumour. The temperature varied between 102° and 104° F., the pulse between 120 and 140; vomiting was frequent and the incontinence of urine and fæces continued. On the 17th, the red blood cells had fallen to 845,000 per cubic millimetre, and the white cells had risen to 65,000 per cubic millimetre. A differential count showed: Polymorphs, 85 per cent.; lymphocytes, 8½ per cent.; large mononuclears, 3½ per cent.; eosinophils, 1½ per cent.; basophils, 1½ per cent. On this day it was decided to transfuse the patient. The clerk of the case, Mr. Clayre, generously offered to give the blood, and both donor and recipient were found to belong to Group IV; 600 c.c. of citrated blood were transfused into the median basilic vein. The effect was almost magical. Three days later the temperature was normal, the vomiting had ceased, she had regained control over the bladder and rectum, the red cells had risen to 3,485,000 and the white cells had fallen to 29,000 per cubic millimetre.

I operated on June 5. The abdominal tumour proved to be the uterus enlarged by the presence of fibromyomata showing hyaline and cystic degeneration. The tumour felt in the pelvis was a pseudomucinous cyst-adenoma of the ovary. I performed subtotal hysterectomy with removal of the ovaries and tubes. Arrangements were made for again transfusing the patient during the course of the operation, but the amount of blood lost was so small that I did not consider it necessary. The patient made an uninterrupted recovery and left the hospital quite well at the end of three weeks. On the day of her discharge the red blood cells numbered 4,250,000 and the white cells 10,400.

I have recorded this case because it involves an important principle, and I believe the treatment adopted in this case will enable us to save lives which would otherwise be lost. It is my intention in future to transfuse all patients in whom the anæmia is severe before operating for fibromyomata. I believe by this means I shall diminish the risk of the operation, lessen the liability to thrombosis and shorten the period of convalescence. I propose also when the patient is very anæmic to adopt it in cases of carcinoma of the cervix before performing Wertheim's operation. I have already done this in one instance with a striking improvement in the condition of the patient.

I suggest, further, that blood transfusion should be performed before delivery in case of severe *antepartum* hæmorrhage, and during the last few months I had charge of the Lying-in Ward at St. Bartholomew's Hospital the blood of the students working in the ward was tested to ascertain their groups as possible donors.

DISCUSSION.

Dr. DONALDSON: I am very glad that Dr. Williamson has brought up the question of treatment of blood transfusion in obstetrical and gynæcological cases. The astonishing results obtained in the case of the wounded during the war have advanced this line of treatment enormously, but I fear it will never be generally used in emergencies until some method has been found for storing the blood without interfering with its action.

The PRESIDENT: The subject is of the utmost practical importance and much progress has been made with this treatment during the war. When successful the effects of transfusion of blood in suitable cases are constantly described as almost miraculous, but even with saline solution it is possible to obtain extraordinarily successful results. Blood transfusion is, of course, better. May we have details of the method employed?

Dr. WILLIAMSON (in reply to the President): The method of transfusion adopted is that described as the "citrated method," and the apparatus employed is that devised by the late Dr. Stansfeld.

Two Cases of Rupture of Vagina during Labour.

By A. C. PALMER, F.R.C.S.

THE cases were admitted to the Obstetric Department of the London Hospital. The first case had internal manipulations carried out before admission, the second had not. We think the cases are of interest in that in each instance it was the vagina which ruptured.

CASE I.

The patient, aged 35, was admitted on March 8, 1920, having been brought up by her doctor, who stated that the case was one of prolonged second stage with the head delayed in the pelvis. He had applied forceps without difficulty, and had extracted the child after a very hard pull. The child was stillborn and weighed between 13 and

14 lb. The perinæum was torn. There had been considerable delay with the placenta, but as the patient continued to bleed considerably in spite of good uterine contraction, he decided to do a vaginal examination and remove the placenta if necessary. On examination he found that by following the umbilical cord his hand passed into the abdominal cavity, where he found the placenta and removed it. The case was then brought up to hospital.

On admission the woman was very anæmic and collapsed. The pulse was 120, and could just be felt at the wrist. Temperature was 98° F. The abdomen was extremely tender. Dr. Luker examined the case under anæsthetic, and found that the "uterus was almost completely separated from vagina." Laparotomy was proceeded with at once. On examining the pelvis, the uterus was found free of all attachments to the vagina except for a narrow bridge in the region of the left uterine artery, and a small portion of the anterior wall of the cervix close to the bladder. The cervico-vaginal junction was torn through for more than three-quarters of its circumference. There was much free blood in the peritoneal cavity. The uterus was removed as rapidly as possible, bleeding points being ligatured. The right uterine artery was not seen, and the torn vaginal margin was oozing fairly freely. Owing to the gravity of the patient's condition no search was made for the right uterine artery. The vagina was packed to control hæmorrhage. Intravenous saline was given. The gauze was removed forty-eight hours later.

Apart from a rise of temperature on the fourth and fifth days after operation, the patient made an uneventful recovery, and is now convalescent.

The short vagina prevents the examining finger from reaching the promontory, although it passes 4½ in. Interspinous measurement, 10½ in.; intercristal measurement, 11¼ in.

This patient has had eight children previously, and there was no difficulty with any of the labours. We believe the difficulty in the present instance to have been due to the very large size of the child.

Case II.

This patient, aged 41, was admitted on March 12, having been attended by a midwife, who stated that the patient had been in labour since 11 a.m on the day before admission. There had been good pains all day, increasing in intensity during the evening, and becoming very severe at midnight. From 2 a.m. the pains were described as tempestuous and different in character from the preceding labour pains. No manipulation was carried out, the midwife made only one vaginal examination, and brought the patient up to hospital because she had become so collapsed.

On admission the patient was pale and collapsed, with pulse at 120, just palpable, and temperature at 96·5° F. The abdomen was tender, and the child unusually easy to feel through the abdominal wall. The head of the fœtus was impacted in the brim but not engaged. The fœtal heart was not heard. On the right of the fœtus was a hard tumour about the size of a fœtal head, and this was thought by the resident accoucheur possibly to be a fibroid. Forceps were applied easily, and as no advance of head was obtained with moderate pull, the child was delivered after perforation and crushing of the head by the resident accoucheur. Immediately after delivery it was observed by Mr. Lack, the resident accoucheur, that the tumour originally felt on the right side was the contracted uterus, and that the child had been lying in the abdominal cavity. The placenta was removed manually from the abdominal cavity.

At Dr. Luker's request I proceeded to do an exploratory laparotomy, and found a large T-shaped tear in the posterior vaginal wall, beginning at the cervico-vaginal junction and extending almost down to the vulva. The cross-piece of the T extended right across the posterior vaginal fornix. There was much free blood and clot in the peritoneal cavity. The uterus was removed as rapidly as possible. There was some difficulty in isolating the uterine arteries in the very œdematous and bruised cellular tissue at the base of the broad ligaments. The lower margin of vaginal tear was picked up deep in the pelvis, and the bruised edge sutured to control oozing. The pelvic peritoneum was partially sewn over to allow drainage by tube *per vaginam* for twenty-four hours. One and a half pints of intravenous saline were given.

The operation was followed by incontinence of urine and fæces for four days, and total incontinence of urine for ten days after operation. The patient is now convalescent. Pelvic measurements, interspinous, 9 in.; intercristal, 10 in. Diagonal conjugate, 3½ in., with promontory high.

The patient had five children previously. The first four labours had been without incident, the fifth had required forceps for delivery. Four easy labours had occurred in spite of well marked general contraction of the pelvis; the fifth required forceps; the sixth ruptured the vagina almost completely, and made laparotomy with hysterectomy necessary.

In the first case the pelvis was normal; the woman had eight previous easy labours, yet the ninth, with a large child, ruptured the vagina, almost completely separating it from the uterus, and rendering immediate hysterectomy necessary.

In each case the object of the hysterectomy was twofold: (*a*) As an essential step in the control of hæmorrhage; (*b*) in the removal of damaged and devitalized tissue, as a prophylactic against puerperal sepsis.

DISCUSSION.

Mr. EARDLEY HOLLAND: Rupture of the vagina in association with rupture of the lower uterine segment is much more common than is generally believed. It is not sufficiently realized how very greatly the vagina is stretched and drawn up by the uterus during obstructed labour. The slides I demonstrate on the epidiascope of sections through the lower uterine segment from the case of a patient who died undelivered during obstructed labour afford an example of this. In this case the retraction ring lay at a level of 11·5 cm. above the symphysis pubis, and below the ring the fœtal head was enclosed by what was at first thought to be an enormously distended and elongated lower uterine segment. The sections show, however, that only the upper 4·5 cm. consist of the extended cervix, all below that consist of the elongated and stretched vagina. The sections further demonstrate that in this case the lower uterine segment consists of cervix only. In this case, if rupture had occurred, it would almost certainly have been in the vagina.

Dr. HERBERT SPENCER: It is sometimes difficult to see the line of demarcation between the cervix and vagina in the second stage of labour, but it is absurd to speak of the vagina as taking part in the formation of the lower segment of the uterus.

Case of Sacculation of a Gravid Bicornute Uterus.

By T. G. STEVENS, F.R.C.S.

A. M., aged 32, admitted to St. Mary's Hospital on February 21, 1920, with the history that labour started on February 19. Although the patient was in great pain, especially in the back, the uterine contractions did not appear to be very powerful. On examination the fœtal head was found to be at the upper part of the uterus, which appeared to be of the size of very nearly a full-time pregnancy.

Per vaginam the upper half of the pelvis was filled up by a fluctuating swelling which depressed the posterior fornix, whilst the os uteri was completely out of reach in front of this mass. The pulse on admission was 101 and the temperature 97°F. This patient had been seen by myself some three months previously, when a similar but smaller swelling was felt *per vaginam* behind the cervix, which even at that time was raised so as to be only just within reach of the examining finger. The swelling was then diagnosed as an ovarian cyst, and although it was immovable it was thought advisable to wait until labour started before doing any operation, in the hope that the tumour could be removed and then delivery completed *per vias naturales*. At the time of admission, which was a fortnight before the time of expected delivery, the swelling was still thought to be an ovarian cyst obstructing delivery, although it was very unusual in that it completely filled the pelvic brim and upper half of the pelvic cavity. As the cervix was above the pubes and could not be reached at all, and the patient was in great distress, it was determined to perform Cæsarean section and then deal with the tumour. On opening the abdomen it was noticed that the bladder was much higher than usual, but the uterus itself presented no striking abnormality. The usual median incision was made in the uterus and the fœtus extracted, the breech being low down in the brim of the pelvis. On delivering the uterus through the abdominal incision it was found that the pelvic swelling was really a sacculation of the uterus itself formed by the greatly distended anterior wall, the actual fundus being held down in the pelvis by a firm adhesion $1\frac{1}{4}$ in. broad by $\frac{1}{8}$ in. thick. The expansion of the uterus had therefore taken place almost entirely at the expense of the anterior wall. Further, a more solid projecting mass was pulled up out of the pelvis attached to the left side of the expanded uterus. This at first was thought to be a fibroid, but it proved to be the enlarged, thickened but not expanded half of a bicornute uterus. The adhesion was divided and when the fundus of the uterus was brought up, it presented a typical picture of a bicornute uterus one half of which was about twice as large as the other. The cervix at first could not be found, but it was located as a flattened-out structure in front and below, making an acute angle with the impregnated half of the uterus; it only just admitted one finger and had hardly commenced to dilate. The uterus contracted well and there was very little hæmorrhage—unusually little. The uterine incision was found to be almost vertically below the angle between the two cornua, so that it opened the unimpregnated half as well as the cornu which had contained the fœtus. The child was in a condition of white asphyxia, but recovered after a hot bath and artificial respiration. The mother made an uninterrupted recovery and went out with the child on March 11.

Cases of sacculation of the gravid uterus are very rare events and have usually been believed to be the result of retroflexion in the early months of pregnancy. The cause in this case was without doubt the adhesion of the impregnated cornu at the bottom of Douglas's pouch, but how this adhesion occurred there was nothing to show. The tubes and ovaries were quite normal and there were no other adhesions. This is the second occasion on which I have performed Cæsarean section on a double uterus; the first case, reported before this Section, was one of labour obstructed by the retroflexed (but not adherent) half of a uterus didelphys.[1] No part of the uterus was removed in that case and the patient has since had a child without difficulty.

Case of Ruptured Interstitial Ectopic Gestation.

By V. Z. Cope, F.R.C.S.

In view of the fact that the number of cases of interstitial ectopic gestation has not yet reached three figures it is perhaps worth while recording the following case which was admitted under my care as a case of acute abdomen at St. Mary's Hospital on June 28, 1919.

The patient, E. T., was aged 40, and had had three miscarriages at intervals of six years, the abortions taking place at the seventh, fifth, and second month respectively. The last miscarriage was six years before her admission. The history was that she had been taken ill with sharp pains in the left iliac region at 2 a.m. on the day before admission. Throughout that day the pains continued and spread over the whole abdomen, whilst vomiting and retching distressed her. She was admitted to hospital about thirty-six hours after the onset of pain.

On admission it was elicited that the last period was in February, four months previously. She believed herself pregnant, for she had morning sickness and enlargement of the breasts. The temperature was 99° F., pulse 80, and respiration 20. The abdomen was rather tumid and there was general tenderness, especially in the hypogastrium.

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[1] *Proceedings*, 1913-14, vii (Sect. Obst.), p. 186.

Vaginal examination revealed a depression of the left fornix and a swelling on the left side of the pelvis. In view of the history, the signs obtained, and the fact that the patient looked decidedly anæmic, there was not much hesitation in diagnosing rupture of an extra-uterine gestation. Operation was advised and carried out about thirty-eight hours after the commencement of the patient's symptoms. The abdomen, opened by a paramedian hypogastric incision, was found to contain a large quantity of free blood, and there was a great deal of clot in the pelvis. Situate upright in an almost median position in the pelvis was a swelling about the size of a uterus three months' pregnant; it was smooth, and large vessels coursed over the summit. There was a rupture on the left postero-superior aspect of the swelling. The left Fallopian tube and the left round ligament were in a lateral position to the swelling and were widely separated the one from the other. This fact, together with a certain rotation of the swelling, at first made orientation difficult. But when the right uterine cornu and Fallopian tube and the right round ligament were found low down on the right of the swelling, and not much enlarged, the diagnosis of a ruptured left-sided interstitial ectopic gestation was made with certainty. It would have been impracticable to resect the sac, so a supravaginal amputation of the uterus was performed. The patient left hospital quite well in three weeks' time.

DESCRIPTION OF SPECIMEN.

The specimen removed consists of the body of the uterus, which is very slightly if at all enlarged, and a large gestation sac which measures about 4 in. by 2½ in. by 2¼ in. The cavity of the uterus has no connexion with the sac. The left Fallopian tube and round ligament are attached to the lateral aspect of the sac and are separated a greater distance than usual from each other. The placenta is well formed and attached to the left superior aspect of the sac; the rupture from which the hæmorrhage had proceeded is to be seen on the postero-superior aspect. The attachments of the right tube and round ligament are to be seen low down on the right of the swelling. The fœtus, which is still attached to the placenta by a tortuous cord, is of a development corresponding to about a three months' pregnancy. Some large vessels are to be seen coursing over the exterior of the sac at a site corresponding to the placental insertion.

COMMENTS.

Upon comparing this case with the series of cases discussed by Wynne in the *Johns Hopkins Bulletin* for February, 1918, one remarks that the patient was older than usual, and that the symptoms came on at a slightly later time of pregnancy (fourth month) than is customary. Moreover, surgical advice was not at once sought, so that operation was undertaken at a somewhat later time than usual after the onset of symptoms.

The operation of supravaginal hysterectomy was the only operation admissible, for in view of the previous loss of blood any half-measure which did not guarantee full and permanent control of the vascular organ would have been unjustifiable. Resection of the sac may be permissible or advisable at an earlier period, but at the end of the fourth month one could not consider it. The ovaries were conserved, since their conservation did not make the operation any more difficult.

The situation of the rupture in relation to the placental insertion suggests that the eroding action of the syncytial layer of the villi was an important factor in leading to the rupture.

REPORTS OF THE PATHOLOGY COMMITTEE ON MR. V. Z. COPE'S SPECIMEN OF INTERSTITIAL ECTOPIC PREGNANCY.

After consideration of Mr. Cope's specimen of interstitial pregnancy the following report was made: "This specimen requires further investigation as to the nature of the smooth-walled lining of the fœtal sac. If this sac proves to be lined with endometrium, showing glands and decidual changes, this will prove that the pregnancy has occurred in a rudimentary uterine horn."

Final Report.—"We have examined this specimen together with further sections cut through the wall of the gestation cavity. There are no endometrial glands in the sac wall and we cannot decide whether this is a cornual or an interstitial pregnancy."

Section of Obstetrics and Gynæcology.

President—Mr. J. D. Malcolm, F.R.C.S.Ed.

Rupture of the Bladder associated with a Retroverted Gravid Uterus.[1]

By S. Gordon Luker, M.D.

Mrs. S. O., aged 38, was admitted to the London Hospital, on June 27, 1919. She had had four children, of whom the oldest was aged 10 and the youngest 4; since then she had had two abortions, one in 1916 at three to four months, the other in 1918 at about seven weeks.

History of present illness: Her last monthly period had commenced on March 15, and she estimated that she was three months pregnant. On June 23, four days before admission, she had been seized with acute abdominal pain, and had walked about the room the whole night. She did not vomit at the time but had vomited twice since. She had had some difficulty in micturition that night and since, but there had been no retention of urine. There had been no show. On June 27, at 3.30 p.m., she felt as if something burst in her stomach causing her acute pain and vomiting. She was brought up to hospital and admitted.

Condition on admission: On admission at 8 p.m. her pulse was 94, her temperature 97° F., and the tongue was moist and furred. The abdomen was flat above the pubes and resonant in front; there was dullness in the flanks. *Per vaginam* a large tumour was found in Douglas's pouch which felt like the retroverted gravid uterus with a soft fluid swelling in addition. A catheter had been passed in the receiving room and 8 oz. of blood-stained urine had been withdrawn.

It was decided to perform laparotomy at once.

Operation: Under anæsthesia a catheter was passed and 2 oz. of turbid fluid were withdrawn. On opening the abdomen 6 to 8 pints of fluid resembling urine were found free in the abdominal cavity. The uterus, three months pregnant, was in the normal position, this having apparently been spontaneously corrected under the anæsthetic. On examining the bladder a linear tear of the superior wall was found about 1 in. long running antero-posteriorly; the wall and mucous membrane of the bladder appeared fairly healthy; there was no active bleeding from the tear. It was repaired by suturing with two layers of fine chromicized catgut. The peritoneal cavity was mopped out and the abdomen then closed with drainage to Douglas's pouch. The patient aborted two days later and afterwards made an uninterrupted recovery. She was discharged from hospital on July 25, 1919.

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[1] At a meeting of the Section, held March 4, 1920.

Examination of the fluid obtained from the abdomen showed that it was alkaline and contained 5 per cent. urea, red blood cells and occasional patches of pus. Cultures were sterile. As regards the cause of the ruptured bladder, I think it most likely that the patient had been suffering from retention of urine with overflow for some days, caused by the retroverted gravid uterus, and that eventually, on the day of admission, the bladder gave way during some violent movement. I can find evidence of only one other case of this rare complication of pregnancy in the literature; this was reported by Kelly, in the *British Medical Journal*, 1883 (X), i, p. 8. In this case a woman, three months pregnant, died suddenly and was found at autopsy to have a ruptured bladder.

DISCUSSION.

Mr. GORDON LEY: I remember seeing a somewhat similar case in the London Hospital in 1917. I was then called to a patient dying of general peritonitis. On examination the uterus was lying very low in the pelvis, which it completely filled. It contained a sixteen weeks' fœtus, the foot of which was presenting through a two-finger cervix. The catheter drew off foul smelling purulent fluid. At post-mortem the bladder showed an acute hæmorrhagic cystitis with a perforation of the fundus of the bladder. The patient had had a catheter passed once seven days before for retention, this being followed by incontinence from retention.

Dr. DRUMMOND ROBINSON: I have recently heard of a case—operated on for me by a colleague—of perforation of the bladder occurring a few hours after a full term confinement. The first stage was long and forceps were used. There was a very small perforation of the bladder difficult to find, with escape of fluid into the peritoneal cavity. The patient died.

Case of Recurrent Hæmorrhage from Laceration of the Cervix, treated by Blood Transfusion and Hysterectomy.

By S. Gordon Luker, M.D.

Mrs. H., aged 30, a 4-para, was seen by me on the eleventh day of the puerperium, July 2. Labour had been prolonged; delivery had been effected with forceps thirty-six hours after rupture of the membranes; the doctor stated that at that time the os was not fully dilated. On the ninth day a severe hæmorrhage occurred, which was arrested by a hot vaginal douche and temporary plugging of the vagina.

On July 2, I examined the patient. She looked very pale but seemed to be recovering from the hæmorrhage. The pulse was 110. The temperature was normal. *Per vaginam*: Involution of the uterus was normal; there was a tear in the cervix on the left side; the lochia were normal. Absolute rest and expectant treatment were advised, and the doctor was warned that if the bleeding recurred, immediate operation would be necessary.

On the night of July 3, the patient had another hæmorrhage and was sent up to the London Hospital after vaginal plugging by the doctor.

On admission the patient was very ill and white. The pulse was 130, the temperature 100° F. On the morning of July 4, I examined her under an anæsthetic. There was a deep tear of the cervix on the left side about 4 cm. long; the surface of the tear was dirty and in parts covered by a slough; it was not bleeding at the time; the uterus was explored and a few small fragments of débris found; the cavity was swabbed out with tr. iodi and a hot intra-uterine douche given. As the cervical tear was dirty, suture was not performed. Thirty hours later the vaginal plug was removed and another hæmorrhage occurred in the night; a deep suture was inserted just above the upper angle of the cervical tear by the resident accoucheur, Mr. A. E. Herman. Twelve hours later the patient had another hæmorrhage and became pulseless. I therefore decided to transfuse with blood and do a panhysterectomy. One pint of citrated blood was obtained from a universal donor and infusion was started as soon as the uterine arteries were clamped. The patient stood the operation well, and at the end her pulse was 140.

On examining the specimen it was found that the uterine cavity was septic and foul, and there was a deep laceration of the cervix on the left side.

After the first few days progress was good, and the patient was discharged in the fourth week.

I have decided to report this case as an example of the importance of blood transfusion in certain obstetric and gynæcological conditions, such as *ante-partum* hæmorrhage, *post-partum* hæmorrhage, ruptured ectopic pregnancy, &c., where life is endangered immediately or later, if some sepsis or other complication should supervene, or even if very prolonged convalescence be anticipated.

Two independent opinions were obtained before the absolute necessity of blood transfusion was decided upon; both agreed that without it the patient would not live.

DISCUSSION.

Dr. EDEN: The important feature of Mr. Luker's case is the deep laceration of the cervix, the direct cause of the hæmorrhage being ulceration of the walls of large vessels exposed by the tear, and this ulceration is the result of sepsis. There was no bleeding for three days, which shows that no important vessels were torn by the injury. I am glad that Mr. Luker had the courage to remove the uterus, for this brought the treatment into line with that of severe rupture of the uterus during labour.

Dr. ANDREWS: Mr. Luker's treatment in this case has been exceedingly successful. I had a somewhat similar case under my care three years ago. The patient was admitted to the hospital, septic and having lost a lot of blood, a few days after rapid removal of a four months' ovum without an anæsthetic after dilatation of the cervix with Hegar's dilators. I explored under an anæsthetic and found that there was a tear in the right side of the cervix about the level of the internal os, through which the finger went into a suppurating cavity in the right broad ligament. From this cavity I removed almost the whole of the fœtal skull. Two days later there was a very severe hæmorrhage which was stopped by tight plugging. Two days later there was again a very severe hæmorrhage which almost killed the patient. I decided that if the bleeding recurred I should have to open the abdomen and try to find the source of the bleeding, but in view of the fact that there was a suppurating cavity in the right broad ligament I was unwilling to do this. Fortunately, after the second severe bleeding there was no recurrence, and the patient eventually did well.

Dr. LAPTHORN SMITH: With reference to Mr. Luker's case of *post-partum* hæmorrhage due to deep lacerations of the cervix, I can recollect at least four such cases. In one instance I was called in consultation by younger friends who had been all night with the patient and who had in the morning delivered her instrumentally. They told me that notwithstanding the firm contraction of the uterus the hæmorrhage had continued in spite of copious douching with very hot water. In such cases I always suspected a tear involving the cervical branch of the uterine artery; and I always carried in my emergency bag two pairs of volsellum or bullet forceps and an Emmet's cervix needle and catgut. Immediately after delivery the uterus is very movable, and it is very easy to grasp the anterior and posterior lips of the cervix and draw them down a few inches out of the vulva, when we shall at once see the little artery pumping a weak jet with each beat of the heart. If we see it when the tear first occurs the jet would be fine but powerful, but as the blood-pressure falls with the loss of blood it in time becomes just a perceptible oozing. While the nurse holds the uterus just out of the vulva it is very easy to pass the non-cutting curved needle well above the angle of the tear, and the moment the loop of catgut is tied all bleeding instantly stops. Knowing as I do the evil effect of an unrepaired lacerated cervix on the future of a woman's health, it is my custom after stopping the hæmorrhage to continue with the same piece of catgut a running suture which brings the torn surfaces accurately together, not only thereby lessening the immediate danger of septic absorption from a large open wound, but also, what is equally important, saving the woman from cancer when she reaches the age of 40, which is quite likely to happen if a large tear, deep enough to go through the cervical artery, is left to heal by cicatricial tissue. It should be more generally known that cancer of the cervix never occurs unless there has been an unrepaired tear which means, of course, cicatricial tissue. Several of my friends who are professors of obstetrics at the head of great lying-in hospitals have felt this responsibility so greatly that they teach their students to examine the cervix in the above manner in all cases in which they have reason to suspect that the cervix has been torn, and if so found to put in a few stitches as I have above described.

Utero-placental (Accidental) Hæmorrhage: A Clinical Report of Fifty Consecutive Cases.

By Gordon Ley, F.R.C.S.

Since the commencement of July, 1915, I have had the good fortune to have seen, treated, or seen treated, and followed up, fifty cases of so-called accidental hæmorrhage and retroplacental hæmatoma. It was my original intention in writing this paper to deal with both the clinical and pathological sides of this condition. The paper however, particularly its pathological side, has necessitated the reading and abstracting of a great amount of literature and short details of many of the abstracts must be given a place in the paper. Further, the pathological side necessitates the introduction of extensive summaries of morbid material taken from three cases. This means that the paper would be one of very considerable length. It has therefore appeared to me best to divide the paper into two parts, a clinical paper, which I am reading to-night, and a pathological paper which I hope to read at no very distant date.

The ætiology of this condition is of considerable interest and will be dealt with first. The average age-incidence in the series of fifty cases was 31·6 years, the youngest case being 19 and the eldest 43 years.

The parity incidence was as follows: Nine (18 per cent.) were primigravidæ; 41 (82 per cent.) multiparæ. The average parity of the multiparæ was 5·7 pregnancies with a maximum parity of 15 pregnancies. The abortion incidence was 1 abortion to 9 labours at term, as against a normal abortion incidence of 1 to 4 or 5 labours at term.

With regard to the causative factors. It will, I think, be best to take up separately the causes given by authors and eliminate them, where possible, singly. *Traumatism* on careful questioning played no part in any one of the cases. Neither did it in any of the 17 cases recorded by Whitridge Williams. It was thought to be the cause in a small percentage of Essen Möller's 29 cases and in 5 of Gaston's 70 cases. Frequently the patient attributed the hæmorrhage to an accident, but the nature of the accident and the interval between the accident and the onset of symptoms always excluded the alleged accident as the causative factor.

Rupture of the sinus circularis of the placenta is thought to be an occasional causative factor by Essen Möller.

Fright, morbus cordis, fibroid tumours and diseases of the blood, played no part in any of the cases.

Shortness of the umbilical cord was not the causative factor in any one of the 50 cases. The average length of the umbilical cord in 32 cases in which it was measured was 21·3 in.; the longest being 34 in.; the shortest 13 in. In one case the cord is mentioned as having been wound three times round the child but there was no tension on the cord, this child being born alive. Shortness of the cord was given as the cause in 12 of the 70 cases recorded by Gaston. It appears to me impossible for shortness of the cord to cause separation of the placenta unless the fœtus is being pulled on. Obviously the resistance by soft parts is such that the fœtus not only ceases to travel forwards at the time the uterine contraction ceases, but is actually forced backwards by the recoil of the muscular pelvic floor. During the pain the uterus with the placenta is travelling in the same direction and at the same pace as the fœtus is travelling. Therefore there can be no pull on the cord either during or at the termination of a pain, while the child remains within the genital tract.

Endometritis is, of course, an impossible ætiological factor to exclude, but if, as is generally stated, endometritis is a cause of abortion, the low abortion incidence would appear to negative this theory.

Syphilis.—The Wassermann reaction was positive in only one of 15 cases in which it was applied. It will therefore be seen that syphilis played no part at any rate in these cases. It is mentioned as playing a part in one of Gaston's 70 cases.

One condition, and one condition only, was pre-eminently associated with accidental hæmorrhage, i.e., *albuminuria*. This was present in 42 (84 per cent.) of the 50 cases. The amount varied from a fine cloud to 15 parts per 1,000 (Esbach). It was usually, however, in considerable quantity. Hyaline and granular casts were present in 13 (26 per cent.) of the cases. Chronic nephritis was clinically present in only 3 (6 per cent.) of the cases. In the others, the albuminuria was apparently of toxæmic origin, i.e., it cleared up after delivery, the average duration of albuminuria, excluding the chronic renal cases, being 11·3 days. Toxæmic symptoms, i.e., œdema, headache, vomiting, &c., were associated in seventeen (34 per cent.) of the cases, eclampsia with one. This association of albuminuria cannot be looked on as an accidental occurrence, and in the absence of other causative factors, one naturally looks to the cause of the albuminuria as the cause of the hæmorrhage. The association of a toxæmic condition in a higher percentage of multiparæ than primigravidæ, is, I know, without precedent; and I have no satisfactory explanation of this phenomenon. Winter, in 1885, first directed attention to the presence of albuminuria in these cases, since which time nephritis or albuminuria has been regarded as the most constantly associated factor. It was associated with the condition in 11 out of 17 cases (nephritis once) recorded by Whitridge Williams; in 37 per cent. of 29 cases, Essen Möller; in 34·25 per cent. of cases recorded by Aschkanazy; in 50 per cent. of cases, Maas; in 30 of 70 cases, Gaston; in 33 of 58, Bar and Kervily; in 82 of 158, Dorman; in 34 of 67, Hartmann.

The frequency of the condition in association with eclampsia has also been noticed, cases being recorded by Bar and Kervily, Couvelaire, Essen Möller, Zarate, Seitz and Young amongst others. These figures, although in none of the records is the percentage as high as in my series.

still further emphasize the association between albuminuria of toxæmic origin and utero-placental hæmorrhage.

The examination of the pathological material which I have obtained shows areas of necrosis with hæmorrhage in the uterine wall, and I am able here to formulate the following theory for the hæmorrhage: (1) Focal necrosis, with hæmorrhage, in the musculature and decidua of the uterine wall. (2) Resulting separation of an area of placenta opening the mouths of uterine sinuses. (3) Extensive retroplacental hæmorrhage.

The onset of the hæmorrhage is usually towards the end of pregnancy. In this series there were between the twenty-third and twenty-eighth week, 3 cases; between the twenty-ninth and thirty-second week, 8 cases; between the thirty-third and thirty-sixth week, 11 cases; between the thirty-seventh and fortieth week, 24 cases.

The cases can be divided clinically into three groups: (*a*) Those in which the blood entirely escapes—*external cases.* (*b*) Those in which the blood is entirely retained *in utero*—*internal cases.* (*c*) Those in which there is free escape of blood with retention of a considerable amount *in utero*—*combined cases.*

Group (*a*): *External Cases.*—These cases are infrequent. Among the series of fifty cases there were only seven belonging to this group. The hæmorrhage in these cases is never of great severity and is usually slight. The only symptom is bleeding, which comes on without cause. Repeated losses, as met with in placenta prævia, do not occur. In the absence of labour pains, there is no pain, but the bleeding is likely to bring on labour. On abdominal examination, nothing abnormal is as a rule revealed, i.e., the abdominal findings of placenta prævia are absent. The fœtal heart cannot be heard in about half the cases; its absence will assist the diagnosis, since in placenta prævia the child is usually alive at this stage. On vaginal examination nothing abnormal is revealed. If the cervix will admit the finger, the placenta will not be felt. The presence of albumin in the urine will go far to confirm the diagnosis, but in the slight cases it is often absent. It was present in only two of the cases in this series. The diagnosis between this type of case and a lateral placenta prævia can often only be made after the expulsion of the membranes and placenta, and then only when the bag of membranes is complete.

The Treatment.—If the loss has been slight and has ceased and if the child is alive, rest with a hypodermic injection of morphia will often result in the pregnancy continuing to term. The patient should be dieted and the bowels kept open three times daily for the remainder of her pregnancy with a view to elimination of toxin. If the loss is considerable, labour pains will always be present. The membranes should be ruptured, a tight binder applied and a hypodermic injection of 1 c.c. of pituitrin administered. If it is essential that no further loss should ensue, that is, if the condition of the patient is really serious, the membranes should be ruptured, the vagina plugged with gauze, a tight binder and a perineal pad applied and fastened together, and a hypodermic injection of 1 c.c. of pituitrin administered. With regard to plugging, I have not had any case in which puerperal sepsis has followed its use. It has always been a routine that the vagina should be dried and swabbed out with iodine, before plugging with dry sterile gauze. I feel, however, after my examination of pathological material that with plugging there is a risk of spontaneous rupture of the uterus through a necrosed area. This, rupture, actually happened in one case of combined hæmorrhage (*vide* Case 27) in which plugging was used.

Results.—If treatment is carried out along these lines, bleeding will cease, labour will progress rapidly, and delivery will be spontaneous, the placenta and membranes as a rule, following the baby at once. The resuits to the mothers have been, in this series of cases, uniformly good; the progress of labour having been satisfactory from the onset of treatment. Half the children were stillborn, but the life of the child is not under the control of the medical man, being dependent entirely on the initial separation of the placenta.

(*b*) *Internal Cases.*—These cases have been sub-divided into: (1) Clinical cases of internal hæmorrhage; (2) retro-placenta hæmatomata.

(1) Cases of this class are also infrequent. Among the series of fifty cases, there were only eleven. The hæmorrhage in these cases is always considerable and this is undoubtedly the reason why the blood does not appear externally. The explanations for its non-appearance hitherto given are: (i) that the cervix is not dilated; (ii) that the membranes are adherent around the internal os; (iii) that the presenting part obstructs the canal; (iv) that the blood ruptures into the amniotic sac. These are all unsatisfactory. With regard to the first of these explanations, it is well known that in threatened abortion there is bleeding in the absence of dilatation. Why not in this condition? To give as a definition of threatened abortion, bleeding without dilatation of the cervix, and to give as a reason for internal hæmorrhage the absence of dilatation of the cervix, is a paradox which should

have no place in obstetrical literature. As a point of fact, dilatation up to two fingers is often present in internal cases.

(2) With regard to the second explanation—the adhesion of membranes—one has only to watch the delivery of a case of internal hæmorrhage to see that this plays no part. The placenta and membranes follow directly on the baby and I have seen no case of combined or internal hæmorrhage in which this sequence of events did not occur. With regard to the third explanation: In the absence of rupture of the membranes it is obviously impossible for the presenting part to block the cervical canal. With regard to the fourth explanation, it appears to me to be mechanically impossible for the blood to rupture into the amniotic sac. For it to do so a sharp salient of membranes must project into the amniotic sac. The tension in the amniotic sac, normally not inconsiderable, will be raised at once by retroplacental hæmorrhage, and as fluid under pressure resists pressure equally in all directions, the formation of this salient will be rendered impossible.

The reason why the blood is retained is that the uterus is paralysed. One assumes a sudden considerable hæmorrhage which forcibly distends the uterus which, thus taken unawares, becomes over-distended and paralysed. The degree of paralysis varies from one in which, although the power of contraction is lost, the power of resisting further distension is retained—i.e., muscular tone is present; to one in which all power is lost the uterus becoming distended to the maximum stretching point of muscle; even this occasionally being overcome, the case terminating in uterine rupture (*vide* Cases 27 and 33).

Symptoms.—The onset is sudden with a severe, or intensely severe, *pain* in the abdomen. This is referred to by the patient as "indescribably bad," as "a tight band round the waist," or as a "bursting pain." The pain persists, and there is no alleviation. The patient's mental state is often so affected by its severity that she is incapable of giving a coherent history, but rolls about the bed complaining constantly of the agonizing pain. Pain may be completely or almost completely absent. It was a marked symptom in 54 per cent. of the cases. *Vomiting* is frequently present. It was present in 45 per cent. of the cases. It may be slight or persistent. The recognition of *pallor* by the patient or by her friends is a common symptom, and is helpful to the clinician. *Fainting attacks*, sometimes described as fits, are not infrequent. They were present in 45 per cent. of the cases.

An examination of the patient reveals anæmia, usually extreme. The face is blanched, the face and hands cold and clammy. The pulse varies from a normal good pulse to a running feeble pulse of 120 or more. The temperature is as a rule subnormal, the respiration rate is usually raised. The abdominal examination is characteristic, so much so that once palpated it is, I believe, difficult to fail to recognize a future case. The uterus is large; it may be definitely larger than it should be for the date of pregnancy. It is tense; this hardness is characteristic and resembles no other uterine condition. It is frequently, although not by any means always, tender; the tenderness may be localized to a definite area of the uterus. The fœtal parts cannot be recognized on palpation as a result of the hardness of the uterus. The fœtal heart cannot be heard. There are no uterine contractions. On vaginal examination the cervix may admit one or more fingers or may be undilated. The bag of membranes, if felt, is tense all the time but not bulging. The urine will be found to contain albumin, which was present in every case in this series. The amount was never less than a cloud, and in only four cases less than four parts per 1,000 (Esbach). Granular and hyaline casts were frequently present.

Treatment.—Attempts to stimulate the uterus are useless, tend to increase shock and should not be encouraged. Attempts at delivery *per vaginam* will as a rule be fatal, and should play no part in the treatment of this condition. Rupture of the membrane is usually fatal, as it relieves the one thing that is keeping the bleeding in check—i.e., the intra-uterine pressure, which has become greater than the patient's blood pressure. The treatment that I have adopted is to employ rest, and to watch carefully (1) the size of the uterus, which should be outlined with pencil; (2) the pulse; (3) the patient's general condition. The patient is put in bed and kept warm. If her pain is intensely severe she is given a hypodermic injection of morphia, $\frac{1}{4}$ grain. The fact that morphia masks symptoms must of course be taken into consideration.

If her condition improves she should be left, the pulse being taken and charted hourly, and the height of the fundus carefully noted. After a few hours' rest, the patient will recover from her shock, the uterus will recover from its paralysis, and contractions will start (*vide* Case 43). The onset of contractions produces external loss, which should be looked on as a favourable sign indicative of expulsive pains. The blood at first expelled is often watery—i.e., serum stained with blood. This, I believe, is the origin of the theory that the blood has ruptured into the amniotic sac; the nurse in attendance has on more than one occasion informed me that the patient's membranes have ruptured, and that "bloody liquor" was coming away; examination has shown, however, an intact bag of membranes. When contractions are definitely present and there is external loss, the case can be treated as one of severe external hæmorrhage—i.e., the membrane can be ruptured, the vagina plugged, a binder and perineal pad applied, and 1 c.c. of pituitrin administered hypodermically, or the case can still be left to nature. The former was done in one case with good result, the latter in five cases with excellent results.[1] The child will be expelled, stillborn, and will be at once followed by the flattened placenta, membranes and one or more pounds of clot.

If the patient's condition becomes worse—i.e., if the pulse rises, the uterus gets larger, or her general condition becomes worse—the case can only be treated by laparotomy. Cæsarean section should be performed, and after removal of the fœtus, placenta, membranes and clot, vigorous attempts should be made to make the uterus contract (*vide* Case 33). This is usually possible with powerful hand and hot towel stimulation. If satisfactory contractions are produced, the uterus should be sutured. If it remains limp and flaccid, in spite of stimulation, it must be removed (*vide* Cases 37 and 41), preferably by subtotal hysterectomy. Good judgment is required in defining just when stimulation should be asserted to have failed. In Case 37 it was carried out for too long a period, the patient being allowed to lose more blood than she could stand the loss of. After Cæsarean section and suture the uterus should be carefully examined for external partial ruptures (*vide* Case 33). The presence of these will be shown by free intraperitoneal blood. These ruptures are due to the extreme tension, possibly coupled with necrosis, rupturing the peritoneum and the outer muscle layers, which contain more fibrous tissue than do the inner layers, and are also of course more stretched than are the inner layers. These tears if present should be sutured. The uterus should then be examined as a matter of interest for subperitoneal hæmorrhages, which are usually present, as are also intramuscular hæmorrhages, which may be noted at the time of Cæsarean section. The case in which the uterus was sutured after Cæsarean section (Case 33) recovered, and Cases 3 and 4 have had a live baby at term since, without untoward symptoms. The cases in which hysterectomy has been necessary (Cases 37 and 41) have died shortly after operation from the combined shock and loss of blood.

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[1] Since writing this paper I have seen three other cases treated by the latter method with equally good results.

Retroplacental Hæmatoma.—By this term I mean hæmorrhages which are entirely behind the placenta, and are, as a rule, associated either with no external loss or only with a show. There were seven cases of this type in the series of fifty cases. They are clinically unrecognizable, but should always be suspected when in a case of toxæmic albuminuria or chronic nephritis intra-uterine death of the fœtus has occurred. They appear to be of particularly frequent occurrence in cases with chronic nephritis, the three chronic nephritis cases in this series all being in this class. There may be a history of a sudden short attack of pain, but more often there is a complete absence of symptoms. The condition is not of necessity associated with death of the fœtus, which will be dependent on the area of placenta separated. Albuminuria was present in all the cases in this series.

Combined Cases.—These cases are by far the most numerous. In the series of fifty there were twenty-five cases which came under this heading. Albuminuria was present in twenty-two of the cases, and was definitely absent in only two. Hyaline and granular casts were frequently present. The cases can be divided into two sub-groups: (*a*) Cases of concealed hæmorrhage in which recovery of the uterus has occurred almost at once, or at any rate before the patient is seen, so that when first seen she is losing blood; (*b*) Cases of revealed hæmorrhage, in which a sudden severe hæmorrhage has caused paralysis of the uterus, with consequent cessation of loss.

(*a*) These cases are by far the most frequent. They show a combination both in symptoms and signs of concealed and revealed hæmorrhage. There are, as a rule, abdominal pains, vomiting, anæmia, and often fainting attacks, followed either at once or after a short interval by bleeding. In some cases these symptoms, with the exception of bleeding, are not present, and only the characteristic uterus indicates the presence of internal hæmorrhage. The treatment is on the lines of a concealed hæmorrhage after external bleeding has started—i.e., either the case is left to nature or the membranes are ruptured, a tight binder is applied or a hypodermic injection of 1 c.c. of pituitrin administered, or the above are associated with vaginal plugging. The results to the mothers have been with one exception excellent; one case dying of uterine rupture (*vide* Case 27). The children have been with three exceptions stillborn.

(*b*) These cases are rare. It is obviously improbable that the uterus will become distended after labour has once started. They are recognized by the characteristic signs of internal hæmorrhage following on external hæmorrhage The treatment is that of internal hæmorrhage, that is, the treatment of the state present. The results should be satisfactory to the mothers.

Briefly summarized the cases have been as follows:—

	Cases	Incidence of albuminuria	Maternal mortality	Fœtal mortality
External bleeding	7 (14 per cent.)	2 (28 per cent.)	0	4*(56 per cent.)
Internal bleeding	11 (22 per cent.)	11 (100 per cent.)	2 (18 per cent.)	11 (100 per cent.)
Combined bleeding	25 (50 per cent.)	22 (88 per cent.)	1 (4 per cent.)	22
Retro - placental hæmatoma	7 (14 per cent.)	7 (100 per cent.)	0	3 (42 per cent.)
Total ...	50	42 (84 per cent.)	3 (6 per cent.)	40 (80 per cent.)

* Two died after delivery.

To Dr. Comyns Berkeley and Dr. Eardley Holland my best thanks are due for permission to publish the cases at the City of London Maternity Hospital. To Dr. Russell Andrews my best thanks are also due for permission to make use of, and publish, certain cases occurring at the London Hospital. Dr. Russell Andrews was extremely kind in allowing me to see and treat cases of utero-placental apoplexy in the London Hospital under his care, and for this and other kindnesses I am greatly indebted to him.

APPENDIX.

The following are notes of a few of the most characteristic cases in the series:—

(6) M. W., aged 33, 5-para; four children, last 1912; one miscarriage at six and a half months. Admitted to the City of London Maternity Hospital on March 18, 1916, with the following history. Thirty-four weeks pregnant. She was quite well until 9 o'clock a.m. on the day of admission; she then had a sharp pain in the vulva similar to the pain that she always had at her menstrual periods. From that time she had a slight continual pain and slight labour pains. At 10 o'clock a.m. she noticed that she was very pale. She was admitted to hospital at 11.30 a.m. The cervix admitted two fingers. There was a slight show. There were no pains. I saw her at 6.30 p.m. She was extremely anæmic. She had been losing blood for ten minutes. The temperature was 97° F., and the pulse varied from 72 to 92. She had vomited frequently since admission. The uterus was the size of a pregnancy at term;

RESULTS OF TREATMENT.

	(1) None				(2) Membranes ruptured ; tight binder ; pituitrin			
	Mother		Child		Mother		Child	
External bleeding ...	4	G (4)	G (3)	SB (1)	—	—	—	—
Internal bleeding ...	5	G (5)	—	SB (5)	1	G (1)	—	SB (1)
Combined bleeding ...	10	G (10)	G (3)	SB (7)	5	G (5)	—	SB (5)
Retro placental hæmatoma	6	G (6)	G (3)	SB (3)	—	—	—	—

	(3) Membranes ruptured ; tight binder ; pituitrin ; plugging of vagina				(4) Forceps delivery.			
	Mother		Child		Mother		Child	
External bleeding ...	1	G (1)	—	SB (1)	—	—	—	—
Internal bleeding ...	—	—	—	—	—	—	—	—
Combined bleeding ...	8	G(7) D(1)	—	SB (8)	1	D (1)	—	SB (1)
Retro-placental hæmatoma	—	—	—	—	—	—	—	—

	(5) Leg brought down				(6) Cæsarean section				(7) Cæsarean hysterectomy			
	Mother		Child		Mother		Child		Mother		Child	
External bleeding	2	G(2)	—	D(2)	—	—	—	—	—	—	—	—
Internal bleeding	—	—	—	—	3	G(3)	—	SB(3)	2	D(2)	—	SB(2)
Combined bleeding	1	G(1)	—	SB(1)	—	—	—	—	—	—	—	—
Retro - peritoneal hæmatoma	—	—	—	—	1	G(1)	G(1)	—	—	—	—	—

it was tense hard, and was tender over an area on the left side at the level of the anterior superior spine. Fœtal parts were not felt and the fœtal heart was not heard. Contractions were present every ten minutes. The cervix admitted two fingers with ease. The membranes were tense and bulging, the vertex was presenting. The placenta could not be felt. The urine contained a very large quantity of albumin. There had apparently been about 10 oz. of blood lost externally. At 6.40 p.m. chloroform was administered. The cervix was dilated to four fingers. The membranes were ruptured; the vagina was plugged with gauze; a perineal pad and a tight binder were applied, and one ampulle of pituitrin injected hypodermically. The external loss had been about 15 oz. at this time. At 7.40 p.m. the pad was bulging and it was removed. The plugging was removed and was followed by the baby, the placenta, and 1 lb. 2 oz. of blood clot with some fluid blood. The baby was stillborn. The uterus contracted well and there was no further loss. The point of rupture of the membranes was 4 in. from the edge of the placenta. The appearance of the placenta was normal. On the 19th the urine contained a faint cloud of albumin. On the 26th the urine was clear. The puerperium was uneventful and there was no pyrexia. On the 29th the uterus was anteverted and involution normal. The patient was discharged on the 30th. This patient has since had a baby at term. The pregnancy and labour were normal. There was no albuminuria.

(12) A. S., aged 25, 1-para; one child, January 4, 1915. Admitted to the City of London Maternity Hospital on August 24, 1916, with the following history: Forty weeks pregnant. The pregnancy was perfectly normal until some time in May; she then had very severe pains in the left side of the abdomen and back, which lasted for one hour. After that she went on well for another month, when she had a worse attack of pain in the same area of the abdomen. During this attack she was very sick; there was no loss; the attack lasted three hours. Subsequent to this attack she had a thick yellow discharge. She had complained of swelling of the legs for three weeks, of hands for two weeks. She was admitted in labour. Labour was normal, the baby was alive, apparently full time but weighed only 5 lb. 9 oz. The placenta and membranes were expelled intact. The placenta showed an extensive central triangular pond-like depression, which occupied about one-fifth of the whole placental surface. It was filled by yellowy-red soft clot and its floor was composed of flattened placental tissue. There were two considerable infarcts in other areas of the placenta. The urine contained a very large quantity of albumin. On the 26th the urine contained a fine cloud of albumin as it also did on the 30th and on September 3. I have seen this patient since and there is still a cloud of albumin in the urine, arterial tension is raised, and she has a persistent high blood-pressure, that is, she has chronic nephritis. She was discharged on September 6, the puerperium was uneventful, the uterus was anteverted and involution normal. The child progressed satisfactorily.

(23) P. R., aged 26, 1-para; one child five years ago, normal labour. Admitted to the City of London Maternity Hospital on August 11, 1917,

with the following history. Thirty-eight weeks pregnant. She had been quite well until 10 o'clock a.m. on the day of admission. She then started losing blood without cause, and lost about 1 pint between 10 and 11 o'clock. She was admitted to hospital at 11 o'clock. She was not then extremely anæmic. There was a blood-stained show. The cervix would not admit the tip of a finger. This child was lying as a left occipito-anterior. The fœtal heart was heard and fœtal parts were felt. At 2 o'clock p.m. severe pain suddenly started in the back and later in the back and abdomen. The pain was continuous, and was not like labour pain. She vomited once. I saw her at 9 o'clock p.m. She was then extremely anæmic and was retching frequently. She complained of cramp in the right thigh and was obviously in severe abdominal pain. She looked extremely ill. The temperature was 98° F., the pulse varied from 104 to 130. The uterus was considerably bigger than a full time uterus, just reaching to the ensiform cartilage. It was tense all the time, contractions could not be recognized, but were, I think, present. There was extreme tenderness over the right lower segment. No fœtal parts could be recognized, except the head in the lower segment. The fœtal heart could not be heard. On vaginal examination the cervix admitted two fingers. There was a blood-stained show. The membranes and head were presenting, the membranes being tense the whole time. The urine contained a fine cloud of albumin. There were no casts in the deposit. A diagnosis was made of concealed accidental hæmorrhage since 2 o'clock p.m., previously revealed hæmorrhage. At 9.15 p.m. pains being definitely present chloroform was administered. The cervix was dilated to four fingers, the membranes were ruptured, the vagina was plugged, a perineal pad and a tight binder were applied, and one ampulle of pituitrin was administered hypodermically. At 11 o'clock p.m. the plugging was forced out, it was followed by a stillborn baby, the placenta and membranes, and 2 lb. 7 oz. of clot. There was no further loss. The point of rupture of the membranes was 4 in. from the edge of the placenta. The placenta appeared to have been entirely separated as its whole surface was flattened. In its centre was an old yellow-floored, irregular pond. It contained no infarcts. On August 13 the urine was clear. The puerperium was entirely uneventful, and the patient was discharged on August 22, the uterus being anteverted and the state of involution normal.

(27) L. J., aged 41, 9-para: nine children, all normal labours. The last day of last period was doubtful, but she believed herself to be full time. Admitted to the City of London Maternity Hospital on October 9, 1917, with the following history: The pregnancy had been uneventful and she had been quite well until the early hours of the day of admission. She then got out of bed and sat in a chair because she felt sick. At 9 o'clock a.m. she felt extremely faint and her neighbours on being called noticed that she looked extremely pale. She vomited at 10.30 a.m. Pains started at 11 o'clock a.m. They were severe and had become steadily worse; they resembled labour pains. She started to bleed at 11 o'clock a.m., and lost continually until the time of admission, at first a lot, latterly only a show. She was admitted at

8.30 p.m.; the temperature was 97·2° F., the pulse 100. The degree of anæmia was extreme. The uterus was larger than a full-time uterus; it was tense the whole time, but there were frequent firmer contractions every few minutes. The uterus was tender all over. The fœtus could be made out distinctly lying as a right position of the vertex. The fœtal heart could not be heard. The urine contained a very thick cloud of albumin, and there was a deposit consisting of granular casts. On vaginal examination the cervix admitted one finger, the canal being ¼ in. long. The membranes were presenting, being tense the whole time. The vertex was above the membranes. She was oozing watery blood. At 9.15 p.m. chloroform was administered. The cervix was dilated with ease to rather more than three fingers. On dilatation the placenta could not be felt. The membranes were ruptured, the vagina was plugged, one ampulle of pituitrin was injected hypodermically and a tight binder and perineal pad were applied. She had a few pains from midnight until 2 o'clock a.m. on October 10. She then slept until 6.30 a.m., and on waking had slight pains. The pulse was then 84 and her condition was very satisfactory. At 6.45 a.m. anæmia increased slightly and the pulse became irregular and rapid. I saw her at 7.45 a.m. The pulse was 100 to 110, the respiration 40. She was certainly more anæmic than on the previous evening. The uterus showed a great increase in size since the previous day and was extremely tender. This tenderness made its outline impossible to define. I felt sure that a further hæmorrhage had taken place into the uterus and decided that Cæsarean section was the best treatment. At 9.30 a.m. chloroform was administered. The plugging was removed, the cervix was found to be fully dilated and the head half way down the vagina. Axis-traction forceps were therefore applied and a still-born macerated, full-time child was delivered. The placenta and clots did not come away although the uterus was hard and empty. A hand was inserted into the vagina and an extensive anterior intraperitoneal rupture of the uterus was found. The placenta was removed from the peritoneal cavity by traction on the cord. It was a normal looking placenta showing no infarcts. The whole of its surface, save for one edge, was compressed and flattened. Laparotomy was immediately performed. A transverse rupture of the lower uterine segment, extending from the right uterine artery region, across the front of the uterus to the mid-line was found. At this point the tear extended obliquely downwards and to the left and involved the vaginal cervix. It then turned vertically upwards and, at the level of the transverse tear already described, it extended again transversely to the region of the left uterine artery. Posteriorly the uterus was intact, There was extensive hæmorrhage in the tissues of the right broad ligament. There was a very large quantity of blood in the abdominal cavity including 1 lb. 5 oz. of clot. This was obviously the intra-uterine concealed clot. Hysterectomy was performed at the level of the transverse tear and the posterior flap was sutured to the anterior thinned flap. The patient died half an hour after the termination of the operation. A post-mortem was done through the operation wound a few hours later. Nothing of note was found except anæmia of all organs. There was no hypertrophy of the heart. The liver and kidneys macroscopically showed only anæmia. The uterus and portions of the liver and kidneys were taken to the London Hospital and a description of them will be given in a later paper.

(32) G. C., aged 21, primigravida. Admitted to the City of London Maternity Hospital on January 5, 1918, with the following history: Thirty-four weeks pregnant. She had been quite well throughout her pregnancy until a fortnight before admission, and since then she had vomited all she had taken. She had had no headache and there had been no visual symptoms. Labour started at 3 o'clock a.m. on the day of admission. The urine then contained a fine cloud of albumin and a few granular casts. She was admitted to hospital at 4 o'clock p.m. At 8 o'clock p.m. she suddenly started losing blood. When seen she was losing considerably, and had lost about 1 pint in ten minutes. The uterus was the size of a thirty-four weeks' pregnancy. The child was lying as a right occipito-anterior; the fœtal heart was heard. The head was deep in the pelvis. Chloroform was administered. The cervix was found to be four-fifths dilated. The bag of membranes was presenting; no placenta was felt. Loss was considerable. The membranes were ruptured and an ampulle of pituitrin administered hypodermically. There was no further loss. At 11.45 p.m. she was delivered of a thirty-four weeks still-born male fœtus. The placenta and membranes followed, the point of rupture of the membranes being directly opposite the centre of the placenta, i.e., the placenta was fundal. One-eighth of the surface of the placenta was flattened. There were no infarcts. On January 8 there was a very considerable degree of jaundice. The stools were somewhat clay-coloured. There were no physical signs. On the 9th the jaundice had practically disappeared. The urine was clear. On the 17th the uterus was anteverted and involution had progressed normally. The puerperium was uneventful. She was discharged on her sixteenth day.

(33) R. P., aged 32, 6-para; five children, first instrumental stillborn, the others normal labours, last two years ago; one miscarriage at four and a half months. Admitted to the City of London Maternity Hospital on May 23, 1918, with the following history: Forty weeks pregnant. She had been quite well until May 21. She then noticed swelling of legs and had frequent fainting attacks as also on the 22nd. At 9 o'clock p.m. on the 22nd, she began to have pains in the back. These were not severe until 2 o'clock a.m. on the day of admission. They then became very severe indeed and got gradually worse until the time of admission. The pain was terribly severe and was quite indescribable. Retching started at 2 o'clock p.m. on the 22nd and had continued up to the time of admission. There had been no vomiting. Her friends noticed that she had become extremely pale on the morning of the 23rd. There had been no loss up to the time of admission. She was seen by the District Sister at 12 o'clock noon. She was anæmic, the temperature was 97·6° F., the pulse 80. She was in great pain. The urine contained a very large quantity of albumin, 6/1,000 (Esbach). She was admitted at 3.30 p.m., and was

seen by me very shortly afterwards. The temperature was 96·4° F., the pulse 80. She was extremely ill and was intensely anæmic. She was rolling about and groaning, obviously in very great continuous pain. The tongue was clean. There was slight œdema of the feet and legs. The uterus was considerably bigger than a full-time pregnancy. It was extremely tender as was also the whole abdomen. It was tense and hard. No contractions were present. No fœtal parts could be felt and the fœtal heart could not be heard. On vaginal examination the cervix admitted one finger with ease. The membranes were tense but not bulging. There was, and had been, no loss. A diagnosis of concealed accidental hæmorrhage was made and as her condition was obviously worse than it had been at 12 o'clock noon, Cæsarean section was decided on. She was given ¼ gr. of morphia previous to the operation. At 5 o'clock p.m. her membranes ruptured and meconium-stained liquor amnii was discharged. At 5.30 p.m. she was very much worse. The pulse had risen to 120. She was still in very severe pain. The uterus had become if anything larger. At 5.50 p.m. laparotomy was performed. The peritoneal cavity contained 1½ pints of blood. Cæsarean section was performed. A stillborn baby, placenta, membranes, blood and blood-clot were all evacuated together. There was much fluid blood and little clot. The uterus collapsed over the hands; the broad ligaments were grasped and the uterus massaged with a hot cloth. It contracted well within two minutes. There was hæmorrhage into the wall of the uterus at the site of incision, but this was not very extensive. The uterus was sutured with buried interrupted silk and superficially with catgut. There was considerable subperitoneal hæmorrhage in the regions of the cornua and streaks of subperitoneal hæmorrhage were scattered over the surface of the uterus. On the posterior wall of the uterus, at the junction of the upper and lower segment, there was a transverse tear 3½ in. in length, extending through the peritoneum and ⅓ in. into the muscle wall in its deepest parts. Just above this there was a very short transverse peritoneal tear, about 1 in. in length. The large tear was sutured in the same way as the Cæsarean section incision. The pelvis was freed from blood and the abdomen was closed. The placenta was completely flattened but contained no infarcts. The point of rupture of the membranes was anteplacental. On May 24 she was extremely well. The urine contained a faint trace of albumin. There were no casts. On the 26th the urine was clear. The puerperium was entirely uneventful and the patient was discharged on her twenty-fourth day, the uterus being anteverted and the degree of involution normal. The Wassermann reaction was negative.

(37) J. F., aged 40, 13-para; twelve children, all normal labours, the last, two years ago; one miscarriage, twins, ten years ago. Admitted to the City of London Maternity Hospital on November 26, 1918, with the following history: Thirty-two weeks pregnant. She had been quite well until 6 o'clock a.m. on the day of admission. She was then suddenly seized with violent abdominal pain, which persisted and became steadily worse. The pain was continuous and very severe. There was frequent vomiting at the onset, and there was a continual feeling of faintness. She was seen by the District Sister at 12.15 p.m.

The pulse was 80, the temperature 98° F. The uterus was nearly the size of a full-time pregnancy, and was slightly tense. Fœtal movements were felt, and the fœtal heart was heard. At 12.45 p.m. she was again seen by the District Sister. She was much worse. The uterus was very tense and distinctly bigger. The pulse was 120. She was admitted to hospital at 1.25 p.m., and her condition was reported to me on the telephone at 2.30 p.m. A waiting inactivity was ordered, and the condition of the pulse, uterus and patient, was requested to be reported at 3 o'clock p.m. At 3 o'clock p.m. I was informed that her condition had not improved, but that she was no worse. I saw her at 4 o'clock p.m. She had begun to lose blood at 3.40 p.m., and the membranes had ruptured at the same time. Previous to this there had been no loss. She was extremely anæmic, and looked very ill. She was rolling about the bed in pain. The pulse was 120, the temperature 99° F. The pulse had been 100 at 1.45 p.m., 98 at 2.15 p.m., 108 at 2.45 p.m., 116 at 3.10 p.m., and 116 at 3.40 p.m. The fundus of the uterus has risen 1 in. since the time of admission. The uterus was bigger than a full-time pregnancy. It was tense the whole time, and there were no contractions. No fœtal heart could be heard and no fœtal parts felt. The urine contained a very large quantity of albumin 4/1,000 (Esbach), and there was a deposit consisting of granular and hyaline casts. On vaginal examination the cervix admitted two fingers. The membranes were ruptured, and there was a breech presenting. As she was bleeding externally, it was decided to leave her for a quarter of an hour to see if labour had started. During this quarter of an hour she became slightly worse, and the uterus became slightly bigger. Laparotomy was decided on, and she was given $\frac{1}{4}$ gr. of morphia hypodermically. At 5.45 p.m. laparotomy was performed. There was a little blood-stained fluid in the peritoneal cavity. Cæsarean section was performed and a stillborn fœtus and $3\frac{1}{2}$ lb. of clot removed with the placenta and membranes. The placenta and membranes were everywhere separated from the uterine wall, and fell out with the baby. The placenta was completely flattened and showed no infarcts. The point of rupture of the membranes was anteplacental. One ampulle of pituitrin was injected hypodermically at the time the uterus was incised. The uterus was pulled out of the abdomen ; it was quite soft and lax, and made no attempt to contract. It was grasped with hot towels and massaged. It contracted slightly, and it was therefore sewn up, silk sutures being embedded, and a running catgut stitch being inserted superficially. After suture the uterus relaxed again, and a further stimulation produced no contractions. It was obvious from her condition that she was bleeding into the cavity of the uterus. Hysterectomy appeared to be the only possible treatment, although I felt sure that the patient would not survive. It was performed, and the abdomen was closed. She died, apparently from shock and loss of blood, at 9 o'clock p.m. A post-mortem revealed death from anæmia due to hæmorrhage. There was extreme anæmia of all organs. Pieces of the liver and kidneys were removed for section. The uterus showed extensive hæmorrhage beneath the peritoneum, over the left cornua, and into the tissues of the left upper broad ligament. There were also extensive hæmorrhages into the musculature throughout the uterine wall. There were no peritoneal lacerations, but internally there were numerous decidual lacerations extending deeply into the wall. I am of opinion that if I had removed this uterus at once when it was found that no satisfactory contractions occurred, she might have recovered.

(41) C. W., aged 37, 7-para; seven children, last two years ago; no miscarriages. Admitted to the London Hospital on February 21, 1919, with the following history: Forty weeks pregnant. She had been quite well, except for continuous frontal headache, since the day before, until 10 o'clock a.m. on the day of admission, when she suddenly fainted in the street. On coming round she felt a very severe continuous pain in the lower abdomen, more to the left side than the right. The pain persisted and became gradually worse. She fainted once or twice after the first attack. She had vomited two or three times since the onset. On examination there was a profound degree of anæmia, the temperature was 99° F. and the pulse 80. She was obviously in continuous severe pain, which was located below and to the left of the umbilicus, The uterus was considerably larger than a full-time pregnancy. It was hard. tense, and very tender all over, especially below and to the left of the umbilicus. No fœtal parts were felt. and the fœtal heart could not be heard. There were no contractions. On vaginal examination there was, and had been, no loss. The cervix admitted one finger. The membranes were felt tense and bulging. The urine contained a very large quantity of albumin, 12/1,000 (Esbach). Hyaline and granular casts were present in the deposit. Laparotomy was done by Dr. Eardley Holland. There was no free blood in the peritoneal cavity. The uterus was incised, and a stillborn baby, 2½ pints of blood, the placenta and membranes were at once expelled. The uterus made no effort to contract, and numerous hæmorrhages were observed beneath the peritoneum in the region of the cornua. It was accordingly removed by subtotal hysterectomy. The patient never rallied after the operation, and died at 12 o'clock midnight on the 21st.

(43) E. H., aged 35, 9-para; eight children, all normal labours, last two years ago; one miscarriage, thirteen years ago, at four and a half months. Admitted to the London Hospital on February 28, 1919, thirty-eight weeks pregnant, with the following history: She had been quite well throughout her pregnancy until the end of January, since then she had not felt well complaining chiefly of a weak and heavy feeling. She had complained of headache now and again for one month. At 6 o'clock p.m. on February 27 she had a sudden attack of heavy pain in the lower abdomen which persisted. It was never a very severe pain. She vomited four times after the onset of pain. She fainted twice, at 1 o'clock a.m. and 2 o'clock a.m. on the day of admission. Her neighbours noticed that she had become extremely pale at 1 p.m. There had been no loss, she had not felt the baby since the onset of the pain. On examination at 4 o'clock p.m. there was a considerable degree of anæmia. The temperature was 97·4° F., the pulse 88. The uterus was larger than a full time pregnancy. It was tense, but not tender. It was

contracting very feebly at intervals. No fœtal parts could be felt and the fœtal heart could not be heard. On vaginal examination the cervix admitted one finger. The membranes were presenting. There was no loss. The urine contained a cloud of albumin, hyaline and granular casts were present. The test for diacetic acid and acetone was negative. It is interesting that the urine had been clear on February 12. The uterus was mapped out with blue pencil, the pulse was taken hourly and she was left in order that she and the uterus might recover from their shock. There was no further loss and there were no definite uterine contractions. At 10 o'clock p.m. her condition was exactly the same. She was therefore examined vaginally. The cervix was three-fifths dilated, labour was therefore progressing favourably. The membranes were ruptured and a tight binder was applied and one ampulle of pituitrin was administered hypodermically. Strong pains started at once. At 10.45 p.m. she was delivered of a stillborn child, which was followed by the placenta, membranes and 1 lb. 8 oz. of clot. The uterus contracted well and there was no further loss. The placenta showed two small infarcts and a fringe along one edge which had remained attached. The rest of the surface was compressed and flattened. The patient was discharged on the twelfth day. The puerperium was normal as was also the degree of involution. The urine was clear on discharge.

BIBLIOGRAPHY.

Aschkanazy. Inaugural Dissertation, Königsberg, 1902.
Bar. *Arch. mens. d'Obst. et de Gyn.*, 1912, p. 162.
Bar et Kervily. *Bull. Soc. d'Obst. et de Gyn. de Par.*, 1906, ix, p. 118.
Couvelaire. *Ann. de Gyn. et d'Obst.*, 1911, viii, p. 591; *ibid.*, 1912, ix, p. 539.
Dorman. *Sloane Hosp. Reports.* 1913, i, p. 57.
Essen Moller. *Trans. XVIIth Internat. Congr. Med.*, 1914, Sect. VIII, p. 25.
Gaston. *Ann. de Gyn. et d'Obst.*, 1916, xii, p. 667.
Hartmann. Coepenhamn, 1907.
Muus. *Ugeskrift f. Lager*, 1903, p. 638.
Seitz. *Arch. f. Gyn.*, 1903, p. 71.
Williams, W. *Surg., Gyn., and Obst.*, 1915, xxi, p. 541.
Winter. *Zeitschr. f. Geb. u. Gyn.*, 1885, xi, p. 398.
Young. *Proc. Roy. Soc. Med.*, 1914, vii, (Sect. Obst. and Gyn.), p. 307.
Zarate. *Ann. de. Gyn. et d'Obst.*, 1915, xi, p. 235.

DISCUSSION.

Mr. T. G. Stevens: I cannot entirely agree with the operation of Cæsarean section as the best method of treatment in the worst cases of concealed hæmorrhage, in which the pulse is gradually rising and the general condition of the patient is not improving. I hold that no one can guarantee the performance of the conservative Cæsarean operation without some loss of blood, and often without a serious loss. This extra loss may very easily turn the scale against the patient and lead to a fatal result. On the other hand hysterectomy, without previously opening the uterus, can be done with a minimum blood loss, perhaps 2 or 3 oz. at the outside, and this is the operation I advise. If performed with gas and oxygen anæsthesia plus novocain infiltration of the abdominal wall, it is practically a shockless operation and can be very quickly performed.

Dr. EARDLEY HOLLAND: This paper is a nice record of a nice set of cases, most of which were under the care of Mr. Comyns Berkeley or myself at the City of London Maternity Hospital. I became very interested in the subject whilst I was doing my work on fœtal death, as I found retroplacental hæmorrhage was a surprisingly common cause. Out of 300 cases I found thirty-six were due to this cause: including five of isolated retroplacental clot. In the latter the fœtuses were all macerated: in the "accidental" hæmorrhage cases only five were macerated. There was albuminuria with casts in 60 per cent. There are two sections for discussion—ætiology and treatment. As to treatment I am in complete agreement with Mr. Ley in all essential details; it is the standard treatment that we adopt at the City of London Maternity Hospital. But I am especially interested in two points of treatment—one is plugging, and the other is the propriety of hysterectomy following Cæsarean section for certain severe cases of concealed hæmorrhage. I know plugging in conjunction with a tight binder is used successfully, and has the favour of experienced men, and that most cases in which plugging has been used recover. But I am not convinced that the plugging has anything to do with their recovery, for the reason that I cannot see what is the mechanical effect of the plugging in controlling hæmorrhage. It is quite likely that they recover in spite of, just as much as because of, the plugging. It is said that plugging controls hæmorrhage because it makes direct pressure on the uterine arteries: even if this is true there is still left a considerable blood supply from the ovarian arteries. It is also said that plugging and a tight binder stop the hæmorrhage by increasing the intra-uterine pressure. To do this they would have to raise the pressure upon the entire surface of the uterus to the blood-pressure, in order to prevent further uterine expansion in any direction from continued internal bleeding. I especially emphasize pressure upon the *entire* surface of the uterus because the uterus is a plastic hollow organ and if its means of expansion is limited in one direction, i.e., antero-posteriorly, by compression between a binder and the posterior abdominal wall, it could still expand in other directions, i.e., upwards towards the diaphragm, and laterally towards the kidney-pouches. The efficiency of the binder would have to be tested by noting cessation of pulsation in the femoral artery. I have definitely made up my own mind about Cæsarean hysterectomy for concealed hæmorrhage. I do not believe it is ever necessary. I have performed Cæsarean section four times for concealed hæmorrhage. Three of the cases were very severe, in which the uterus remained paralysed and flabby and thin, like a wet wash-leather bag. The only one in which I removed the uterus was the last of the series, and the patient died, from shock I believe. From my experience in the other two severe cases I am convinced that a uterus will always contract if time be given it: I should not mind waiting fifteen or twenty minutes for this to occur, controlling hæmorrhage in the meantime by compression of the uterus between hot towels without and hot gauze pads within. Another reason, or excuse, sometimes given for hysterectomy is that, if the uterine wall is infiltrated with blood it is better removed, as it is a dangerous possession. I know that is not true, at all events of all cases, and believe it is not true of any. One of my cases of Cæsarean section was as bad a case as any of interstitial hæmorrhage. The peritoneal coat of the uterus was split in several places, there were subperitoneal hæmatoma, a fair amount of blood in the peritoneal cavity, and the cut surfaces of the uterus showed much interstitial hæmorrhage. I sutured the uterus. That was in 1916: at the end of last year the patient had a normal labour at term, and delivered herself of a 7¼ lb. child. I have notes of another case at the City of London Maternity Hospital, who had Cæsarean section performed by Dr. Barris, in 1915, and delivered herself unaided of a full term 9½ lb. child in 1917. Both of these cases are included in Mr. Ley's list.

As I understand Mr. Ley is going to give another paper on the pathology of the uterus in cases of concealed accidental hæmorrhage I shall not refer to this aspect, but, all the same, I should like to state that, in my opinion, the interstitial hæmorrhage in the uterine wall, as well as the peritoneal splits and the subperitoneal hæmorrhages are all due to a tearing of the uterine tissues from the great bursting-stress to which it is subjected, and not to an intense necrosis of the fibromuscular and vascular tissues.

This brings me to the nomenclature of the condition. To begin with, I view the very name apoplexy, as applied to organs or tissues infiltrated with blood, with disfavour. Apoplexy literally means a knock-down blow, and was originally applied to the knock-down effect of cerebral hæmorrhage. It seems an extraordinary confusion of thought to speak of an "apoplectic" kidney or lung or ovarian cyst. The term is used in Goodell's famous paper on concealed accidental hæmorrhage in 1869—he speaks of "apoplexy of the placenta." It was given great prominence by Couvelaire, when he published[1] his classical description of cases of concealed accidental hæmorrhage with interstitial and subperitoneal uterine hæmorrhage: he is responsible for coining the term "uteroplacental apoplexy"—the one used by Mr. Ley as the title of his paper. Now the point comes, are we justified in calling all cases of accidental hæmorrhage cases of uteroplacental apoplexy? Mr. Ley uses the words synonymously, as I understand him: we are not justified in doing that, for we have no evidence to support it. Utero-placental apoplexy is the name given to certain severe cases of concealed accidental hæmorrhage which were treated by Cæsarean section, and in which the peculiar bloody state of the uterus was visible to the eye: the uterus, externally and on its incised surfaces, was visibly in a so-called state of apoplexy. But there is, so far as I know, no evidence at all that a similar "apoplectic" state of the uterus is found in any case of accidental hæmorrhage other than a severe case of the concealed variety. If this is true—if interstitial uterine hæmorrhages are found associated only with concealed hæmorrhage, and not with external, it is strongly circumstantial evidence for the view that the interstitial uterine hæmorrhage comes from the tearing of tissues due to a great bursting-stress.

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[1] *Amer. Journ. Obstet.*, 1869, ii, pp. 281-346.

When we come to consider the mechanics of so complicated a structure as the pregnant uterus we must tread carefully. But Mr. Ley has made a mistake in elementary mechanics when he states that the outer layers of the uterus are more stretched than the inner layers. All the layers are, of course, stretched relatively equally: not absolutely equally, of course, since the outer layers have a greater circumference than the inner, but the expansion of each is relatively equal in proportion to its original circumference, which is the same thing as saying that all layers are stretched equally. Peritoneum is a very slightly extensile tissue, whereas muscle is one of the most extensile of all tissues; it is natural for the peritoneal coat to split before the muscle when the uterus is suddenly distended. That view is supported by Matthews Duncan's experiments on the behaviour of the uterus under the influence of bursting-stress; he found the peritoneum yielded before the muscle. Mr. Ley seems unnecessarily anxious to tilt at commonly accepted theories and explanations, and when he disposes of them *seriatim* in such a light and easy manner, he is not always correct. As for an undilated state of the cervix causing retention of blood in the uterus, such an obviously erroneous explanation is hardly worth bringing forward.

Again, Mr. Ley says that in the absence of rupture of the membranes it is obviously impossible for the presenting part to block the cervical canal. I am sure it can. The breech lies in fairly close contact with the fundus, whilst the vertex rests on the lower uterine segment: if the placenta lies over the breech in the fundus, a projection forwards of the placenta by a retroplacental hæmorrhage will, by direct fœtal pressure, jam the vertex into the lower uterine segment. He also says that a projection forwards, or salient formation of the membranes is impossible. That such a projection or salient of the membranes is possible is well shown by P. Bar's frozen sections. Salient formation is obviously possible, by the fact that a plastic cavity like the uterus has the faculty of gaining compensation for contraction in one direction by expansion in another. I quite agree that rupture of the blood into the amniotic sac is, mechanically, very unlikely; but it is described, by good observers, and one must take into account the suddenness with which the blood is poured out behind the membranes; the pressure of this must be, at the moment of its escape, greater than the intra-amniotic pressure. The impulse, theoretically, may be sudden enough to rupture the membrane before its pressure-raising effect has been distributed throughout the amniotic fluid. This is the well-known principle of the inertia of bodies. I merely mention these points because the mechanics are difficult, and I do not like to see them dismissed in such an easy-going way as Mr. Ley dismisses them. These few criticisms are only on minor points, and I am, at all events, grateful to Mr. Ley for his facts.

Dr. ANDREWS: Mr. Gordon Ley has made an excellent use of his extraordinary collection of cases of accidental hæmorrhage. I was very glad to add a few cases which occurred at the London Hospital to his very large number of cases which occurred at the City Road Lying-in Hospital. I cannot agree with Mr. Gordon Ley that shortness of the cord could never cause too early separation of the placenta. There is a specimen in the London Hospital Museum from a case of fatal inversion of the uterus due to shortness of the cord in a spontaneous labour. If inversion can occur thus, separation of the placenta also might occur. Mr. Gordon Ley's paper rather suggests that the explanation of concealed accidental hæmorrhage as due to paralysis of the uterus is new, but this view has been generally held for a long time.

Dr. K. M. ROSS: Will the author explain why in cases of severe accidental hæmorrhage, and sometimes in cases of *post-partum* hæmorrhage, the pulse-rate is often not more than 90 per minute, rising later to 120-140, as it is usual to state that with severe hæmorrhage the pulse is rapid? This is a fact which I have noted in cases of concealed accidental hæmorrhage and also lately in two cases of severe *post-partum* hæmorrhage.

Dr. GORDON LEY (in reply): First, I wish to thank Mr. T. G. Stevens, Dr. Russell Andrews, and Dr. Eardley Holland for their kind remarks. I am in a quandary with regard to Mr. Stevens's advocacy of hysterectomy and Dr. Holland's advocacy of Cæsarean section, and I feel this point must be left *sub judice*. On the one hand three recoveries with two subsequent pregnancies favour Cæsarean section. On the other hand two deaths from ineffective uterine contraction favour hysterectomy. In one of these the degree of necrosis of the uterine wall made the possibility of efficient contraction quite impossible. If one could only recognize extensive necrosis before opening the uterus, I should certainly advocate hysterectomy in such cases. I thank Dr. Kenneth Ross for his comment on the pulse-rate. I have often noted that even in severe cases the pulse-rate is slow. Is albuminuria the sequence of the hæmorrhage or the result of the toxæmia? Dr. Young dealt with this subject some years ago. I am not in agreement with Dr. Young, and think I shall be able to show that the albuminuria is the sequence of toxæmia, and that the toxæmia is not the sequence of placental separation. I cannot agree with Dr. Eardley Holland when he says that the intramuscular hæmorrhages are due to stretching. Recently I had a case of placenta prævia in which the uterus became forcibly distended as the result of considerable intra-uterine hæmorrhage. This uterus showed no areas of intramuscular hæmorrhage.

Section of Obstetrics and Gynæcology.

President—Mr. J. D. MALCOLM, F.R.C.S.Ed.

Accidental Rupture of Pedicle of Calcified Fibroid.[1]

By PHILIP D. TURNER, M.D.

ON January 15, 1920, Miss P., aged 44, was bicycling along the sea wall from Ryde to Sea View when she accidentally rode over the edge and pitched on her left side on to the sand, about 5 ft. below. She bruised her left hip, which seems to have borne the brunt of the fall, and had to be taken to her home in a bath chair, when she sent for Mr. Carter of Sea View. He found no lesion except a bruised hip, but thought it advisable to see her again the following morning. She was then complaining of pain in the left side of the abdomen, which was tumid. Her pulse which was 74 the previous evening was now 96.

I saw her with Mr. Carter at 12.30 that day (January 16), and found her lying on her back breathing rather quickly with a sort of diaphragmatic catch in the breath. The abdomen was decidedly tumid, but the muscles rather rigid, especially on the left side. Dullness in the left flank extended to the level of the anterior superior spine. Pulse 100. A very hard tumour, about the size of a fœtal head, evidently a calcified fibroid, was felt rising out of the pelvis. I expressed the opinion that she had ruptured something in her fall, probably connected with the fibroid, and was bleeding into her peritoneal cavity.

I had her removed at once to hospital, and opened the abdomen at 4 p.m., just under twenty-four hours after the accident. The peritoneal cavity contained a quantity of fluid blood and clots. The tumour proved to be a pedunculated calcified fibroid attached to the fundus by a pedicle about $1\frac{1}{2}$ in. long and $1\frac{1}{4}$ in. thick. A tear was visible on the left hand side of the pedicle, running in its axis and almost throughout its length—blood was issuing from this. The tear, which was longitudinal in the capsule of the pedicle, was transverse and almost complete through its substance. The tumour was cut away by dividing the remainder of the capsule. Bleeding arose from two small arteries, which would not hold a Spencer-Wells forceps. Silk ligatures were passed underneath them with a curved needle and tied. The wound on the fundus was then sewn up by deep antero-posterior silk sutures and the abdomen closed. Recovery was uneventful.

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[1] At a meeting of the Section, held June 3, 1920.

The fibroid was roughly cubical in form, about the size of a new-born fœtal head, with sharp spicular angles, which might easily have inflicted wounds on other parts when projected with violence, as it must have been by the fall. The linear tear in the capsule probably acted as some restraint on the quantity of blood lost, as it was not gaping.

The accident appears to be a rare one, as I have not been able to hear of any similar case. The unusual nature of the injury probably appertains to the unusual character of the fall. It is not often that a woman with her bicycle falls 6 ft., and in such a case if the fall were on hard ground the injuries would probably be many and serious. In this case, the fall being on soft sand, no serious injury to limb or trunk occurred, the yielding impact of the sand, while saving the hard parts from injury, allowed the heavy tumour to plunge out of the pelvis to the length of its tether and to tear its pedicle.

DISCUSSION.

THE PRESIDENT: I have never seen a case of this kind, but it is not very uncommon to find that in dragging away a tumour from the uterus to get at a short pedicle the latter tears, so that it is easy to understand how violence would produce the conditions described.

Dr. LAPTHORN SMITH: The result of the treatment of this patient fully justified the doctor's prompt action. The case is a very rare one: I have never even heard of anything like it. I can recall several, however, where the patient had fallen off a chair or a step-ladder and was taken with serious abdominal symptoms. In one case the woman had all the symptoms of a severe abdominal hæmorrhage and within a few hours I opened the abdomen and found an ovarian cyst which was partly strangulated by twisting of the pedicle and the fall had caused a rupture of a very large vein into the cyst. In another a fibroid tumour with a long pedicle had completely shut off its blood supply by twisting and thus becoming a foreign body, a local peritonitis was set up so as to form adhesions and thereby nourish it through the omentum and intestines. But in neither had there been any free hæmorrhage into the abdominal cavity as in the case reported which, in this respect, was quite unique.

Rupture of the Uterus; Treatment by Gauze Packing; Recovery.

By J. Ford Anderson, M.D.

Mrs. E., aged 33, a multipara, with a history of three normal labours and one miscarriage, was admitted to a maternity institution in the borough of St. Marylebone in the night of January 26-27, 1918. The patient was losing blood freely and was greatly collapsed, and the midwife in charge said she appeared moribund. A note was brought with her from a medical practitioner in a northern suburb to the following effect, and this is the only history of the case which I obtained. He was called to the patient at 9 p.m. on January 26, and found her in labour with a fœtus of approximately four months, the feet presenting and partially born. There was no contraction of the uterus, but expression and traction were used, and the fœtus, he said, was easily removed. After removal of the fœtus he discovered a tear of the cervix uteri extending outwards on the left side. He removed the placenta "with the fingers," and in doing this found coils of intestine lying in the uterus. I saw the patient for the first time on January 27 at 11 a.m., that is to say, fourteen hours after delivery. The midwife had meanwhile tried to restore vitality, but the patient was still in a state of extreme collapse, blood was oozing from her vagina, the uterus was relaxed, and the cervix was contracted. There was no evidence of violence externally, but when I examined her internally under an anæsthetic, I distinctly felt a coil of intestine lying in the uterus, and from its sacculated character I considered it to be large intestine. On exploring further I found that the rupture or laceration which admitted the prolapsed bowel was situated at the fundus of the uterus—transverse in direction—and, so far as I could judge, between 3 in. and 4 in. in length. At the same time I noted the deep tear in the cervix already referred to, and I ascertained positively that the tear at the fundus and the tear of the cervix were not continuous, they were quite independent of each other.

The prognosis of such a case could scarcely be optimistic. The patient continued collapsed with a feeble pulse, and there was already evidence of sepsis and incipient inflammation in the left iliac region. My colleague and I agreed that operation was inadmissible, and I therefore proceeded to try other means.

The patient was lightly anæsthetized and placed on her left side, with the pelvis raised, and I replaced the prolapsed bowel and proceeded as follows: I stimulated contraction of the very lax uterus by pressure of my left hand on the fundus, and at the same time I packed the uterus with sterilized gauze, with my right hand, while an assistant retracted the posterior vaginal wall with a duck-bill speculum. I used an ovum forceps and kept control of the uterus throughout. I also asked the midwife to administer a hypodermic injection of 1 c.c. pituitrin immediately, and repeat this injection in ½ c.c. doses every four hours. The first packing was removed and replaced with similar precautions on January 28, after being thirty hours *in situ*. The uterus was then fairly contracted and the bowel had receded, and on January 27, sixty hours from the first introduction, the gauze was finally removed. There was swelling and tenderness in the left iliac region almost from the first, the temperature varied for several days from 103° to 104° F., and the lochia were fœtid. The patient was kept in the recumbent position and, besides pituitrin, morphine was administered hypodermically, continuous saline injection into the rectum was arranged, and occasional warm douches of lysol solution (1 in 320) were administered. Antiphlogistin was the dressing for the pelvic inflammation, and oxygen inhaled frequently proved a great boon in combating the condition of collapse. Catheterism was required for a period.

On February 13 the tears in the cervix and in the uterus had apparently healed, and there was trifling discharge, but the pelvic cellulitis, which was clearly due to sepsis from the cervical tear, with pain and tenderness and swelling in the left iliac region and fluctuating temperature, were still present on February 20, when for some family reason the patient insisted on taking her discharge at her own risk. A few days after her return home she was admitted into a poor law hospital in her own neighbourhood, where the pelvic abscess was opened, and I was informed by the surgeon that she was discharged on April 19, having made an uneventful recovery. The illness lasted eight weeks from the labour.

That is the case. It really presents two problems: A laceration of the body of the uterus, and a tear of the cervix leading to pelvic cellulitis. As to the fundal tear, it was isolated, and it would probably have given very little trouble after a week; but the cervical tear with its septic pelvic abscess retarded progress, and became a longer matter, and I may add that this was a condition which we who practised in pre-Listerian days had too many opportunities of observing.

My object has been to put on record a case which adds to our experience in the treatment of rupture of the uterus. The question of operation would be generally, I think, the first thought which would occur to any practitioner who finds himself in the presence of such a case. It was certainly my first thought until I was compelled to discard it, and several medical friends to whom I spoke thought as I did, their comment being: "Why was no operation done?"—and one gentleman, the surgeon at the poor law hospital at which the abscess was opened, a gentleman of long surgical experience, told me that he had never before met with a case which recovered without operation. I conclude, therefore, that the professional man in the street thinks operation the best course as a rule. If this is wrong he should be instructed. When I packed with gauze in the present case, I only selected that method of treatment after rejecting operation as inadmissible and fearing the worst, but I am inclined now to modify my general views on the subject after my experience in this case and reading Dr. Herbert Spencer's four successful cases and his comments thereon in the Obstetrical Society's *Transactions* for 1900 (vol. xlii). He there states that he packed his four cases with iodoform gauze, and they all recovered, and he goes on to say that the only successful cases which he had treated were those in which he packed the uterus. It would be most interesting and instructive if we could ascertain Dr. Spencer's present views on the subject, and as to whether he has seen reason to alter his conclusions in the years that have elapsed since he recorded his cases.

DISCUSSION.

The PRESIDENT: Dr. Ford Anderson and the patient are both to be congratulated upon the result of his treatment. If the case could be divided into parts, I think a complete rupture of the uterus into the peritoneal cavity would be best dealt with by opening the abdomen and suturing the uterine wall. Every case must, however, be treated on its merits, and in Dr. Anderson's patient the complications made the method I have just indicated impracticable. In Dr. Spencer's cases referred to, two of the ruptures were not into the peritoneal cavity, which made the danger less, but his other two and the one now related prove the efficacy of treatment by packing.

Dr. AMAND ROUTH: I think packing the uterine laceration should be reserved for cases rupturing into the broad ligament or for those in which the laceration has not included the peritoneum. Whilst congratulating Dr. Anderson on the result, I cannot understand how the patient escaped peritoneal infection after the replacement of the intestines in an evidently septic case, for the difficulty of disinfecting the prolapsed coils must have been great. How does Dr. Ford Anderson explain the fundal laceration?

Dr. DRUMMOND ROBINSON: Some twenty years ago I had a somewhat similar case. A young woman was delivered of her first child by forceps. The case was one of persistent occipito-posterior presentation. Profound collapse followed and a mass which proved to be intestine, omentum, &c., protruded from the vulva. A laceration through the cervix extended into the body of the uterus. The intestine was pushed back and the laceration plugged with gauze. This patient recovered and subsequently gave birth to two children.

Dr. FORD ANDERSON (in reply to Dr. Routh): I would point out that I took every possible aseptic precaution in replacing the prolapsed bowel and in packing the uterus and that the bowel was never prolapsed beyond the cervix. As to the causation of the accident: it is a matter of conjecture as I have no history beyond that which I have given. I regret Dr. Spencer's absence, but I have received a note from him which I will now quote. He says: "I am glad you are reading notes of a case of rupture of the uterus treated by packing. . . . There is no doubt that it is the best treatment for many cases, as shown in my paper. These patients are usually too ill to bear hysterectomy, and though now many cases have been successfully treated by hysterectomy the cases have often been cases which should not have been ruptured at all but cases badly treated by version, &c., and then the operator has proceeded to remove the uterus. One Russian obstetrician had a long list of such cases. They are no guide to the treatment of the ordinary cases of rupture seen in a condition of profound shock."

Wertheim's Hysterectomy for Advanced Carcinoma of the Cervix made possible by the Use of Radium.

By WILLIAM FLETCHER SHAW, M.D.

CLINICAL CONSIDERATIONS.

THE greatest advance in the treatment of carcinoma of the cervix was the general adoption of the radical or Wertheim's operation in place of the old-fashioned vaginal or pan-hysterectomy. By this method it is now possible to obtain a definite cure of this dread disease, though the percentage of cures is still very low. This is partly due to the failing common to all operations for malignant disease, which in reality are very rough and ready methods of treatment, and will probably be superseded when the true ætiology of the disease is discovered. But the chief cause of failure is the late stage at which the gynæcologist is consulted, usually from fear or disinclination on the part of the patient. I have not found it possible to calculate the total number of cases of carcinoma of the cervix I have seen in the last few years, but the number in which I have been able to do a complete radical operation must be a very small percentage of the whole, and this I think is the experience of all gynæcologists.

The next great advance in the treatment of carcinoma of the cervix was the discovery that radium acted beneficially on the growth. Although on some types of growth and in some situations radium has little effect, in carcinoma of the cervix its action is wonderful. The first time I had the opportunity of observing this was in 1914. I had seen a case of advanced carcinoma of the cervix with a hard pelvic mass the size of an orange on the left side in a patient, aged 53. Operation was quite out of the question so I was very anxious to try the effect of radium, of which we were then having most optimistic reports. Dr. Burrows, who had just arrived in Manchester, kindly treated this patient for me. The result was little short of marvellous. Altogether she had three applications in the course of twelve months, and both the primary growth and the secondary mass disappeared, so that at the end of twelve months there was no growth to be felt, just a normal-sized uterus partly fixed to the left side of the pelvis; at the same time, her general condition had greatly improved and she was free from pain. Unfortunately, this good effect did not last long. Soon the pelvic mass re-formed and continued to grow in spite of further application of radium. The bladder and rectum became involved and the patient died at the end of the second year from my first seeing her. The net benefit was probably one year's extra life and I do not think the final stage was so distressing as is often the case.

Since that time Dr. Burrows has treated a large number of my cases of carcinoma of the cervix with radium. A large percentage of these cases showed a marked improvement with this treatment, in many the disease had apparently disappeared locally when they were examined two months after the application, but in no case of mine has the improvement lasted more than one year, even with repeated applications of the radium. This is very disappointing. The initial improvement was, however, so marked that I still hope further experiments with different methods of application may lead to complete cures.

Case I.—In November, 1916, I was asked to see a patient in consultation with Dr. Christie, of Longsight. She was aged 45, had not passed the menopause, and had suffered from excessive hæmorrhage for six months. Examination revealed a large friable mass on the cervix extending on to the vaginal walls. At that time I was advising the radical operation in all cases of carcinoma of the cervix in which there was the slightest chance of removing the growth, there being no other treatment which gave any hope of cure, and it seemed better to risk an abandoned operation than to neglect even the faintest chance of cure, but this case was too far advanced for me to attempt a radical operation, so I advised the application of radium as a palliative. Dr. Burrows applied this in December, 1916, and I saw the patient again in February, 1917. The result of the radium was excellent; the cervix had contracted to little above normal size, had lost all friability, and so far as I could tell was not fixed. Apparently the uterus could now be removed quite easily, and after consultation with Dr. Burrows I decided to attempt a radical operation. If the growth was entirely local and had been so reduced in size and activity by the radium, there seemed a possibility of removing the whole trouble; if the growth had become disseminated, and this seemed likely with so advanced a growth, then the patient would die of recurrence elsewhere, but this would be no worse, probably not so bad, as dying of local recurrence, which from previous experience we felt sure would occur. I took the patient into St. Mary's Hospital and opened the abdomen on March 12, 1917. After ligaturing the infundibulo-pelvic and round ligaments on each side and cutting the utero-vesical fold of peritoneum, I found the ureters on the posterior fold of the broad ligament. After tying the uterine arteries far out, I proceeded to lift them out of their bed, but I now found the real difficulty in this operation—a difficulty which has since caused me to abandon six similar operations. What had been growth originally had contracted down under the influence of radium into a smaller mass, but this was as hard as cartilage. Fortunately in this case the ureters were only slightly involved and I was eventually able to separate them from the adjacent and partly surrounding hard cartilage-like tissue. We all know the difficulties of an ordinary Wertheim's operation, but after the application of radium the difficulties are increased enormously, owing to this hard tissue which replaces the growth.

I saw this patient at intervals, the last time on May 18, 1920, three years and two months after the operation, and three and a half years from the first examination, and she was then very fit and well, doing all her housework and putting on weight. The previous week she had walked seven miles on four consecutive days without any fatigue. From the condition she was in when I first saw her I do not think she could have lived one year without the treatment and her condition during that time would have been very miserable. Whether eventually there be any recurrence or no, she has had over three years of excellent health. This seems a great advance.

Since that date I have operated upon thirteen other patients with advanced carcinoma of the cervix, too advanced for operation when first seen, but apparently operable two months after the application of radium. In six of the cases I had to abandon the operation owing to the impossibility of separating the ureters or bladder from the hard tissue which had replaced the growth, but in the remaining cases, eight altogether, I was able to do a complete radical operation.

Case II.—Mrs. H., aged 59, passed the menopause ten years previously and had vaginal hæmorrhage for three years before I saw her on July 2, 1917. She complained of pain in the back and left side, but had not lost flesh. She had a large friable cervix, but when I examined her under an anæsthetic, preparatory to performing Wertheim's operation, I found the growth had extended too far to allow of removal. Dr. Burrows treated her with radium and on August 21 I found the cervix to be normal in size and apparently free. On August 30, 1917, I performed Wertheim's operation and found very little hard tissue around the cervix, but had considerable difficulty in separating the ureters. Unfortunately two days after the operation she had signs of intestinal obstruction, but was too collapsed to allow of further operative interference, and she died that day. Post mortem we found a piece of intestine adherent to the wound and kinked, but with no signs of sepsis.

Case III.—Mrs. F., aged 61, passed the menopause ten years and had vaginal hæmorrhage for about one year. I first saw her on August 3, 1917, and then found a large friable foul mass springing from the cervix and extending on to the vaginal vault on the left side and the cervix was fixed. I considered this growth quite inoperable and asked Dr. Burrows to treat her with radium. On September 1 the cervix was quite small and showed no signs of growth. Dr. Burrows applied radium for a second time on September 1 and on September 23 I performed Wertheim's operation. In this case I did not find any extensive mass of hard tissue, but the ureters were difficult to separate. There were no glands and no adhesions to the bladder. This patient was in very good health when I last heard from her on March 1, 1920, two and a half years after the operation.

Case IV.—Mrs. B., aged 59; passed the menopause twenty years, and had vaginal hæmorrhage for six weeks when she came to see me on October 1, 1917. I found a large friable growth on the cervix and took her into hospital with the intention of performing Wertheim's operation, but found this impossible, so I cauterized the growth and four days later Dr. Burrows applied radium. On November 21 I again saw her and found the cervix quite small and not friable, so I performed Wertheim's operation on December 3, 1917. This was not a particularly difficult operation compared with some of them, but she had a large mass of hard cartilaginous tissue at the base of the left broad ligament which I was able to excise completely. On July 19, 1918, seven months after the operation, Dr. Burrows found she had definite recurrence between the bladder and the vagina and she died one month later.

Case V.—Mrs. A., aged 38. Constant vaginal discharge and hæmorrhage for two years. No urinary nor rectal symptoms. She had a large fungating growth of the cervix extending with a hard edge on to the vaginal wall at the back, not adherent to the bladder or rectum. Radium was administered by Dr. Burrows on February 22, 1918, and I performed the radical operation on May 27, 1918. Both her doctor and I have lost sight of her, but her doctor is sure she had signs of recurrence about one year after the operation.

Case VI.—Mrs. A., aged 55; passed the menopause some years. History of slight offensive discharge and later severe hæmorrhage for five months. She had a large fungating growth of the cervix spreading extensively anteriorly on to the vaginal wall. Radium was administered on April 23, 1918, and I performed the radical operation on July 11, 1918. I last heard from her on February 18, 1920, when she was very well.

Case VII.—Mrs. B., aged 46; not passed the menopause. For six months menstruation every fortnight and hæmorrhage after coitus. Friable mass on posterior lip of cervix, extending to the vaginal wall. Radium was applied on November 4, 1918. I performed Wertheim's hysterectomy on January 7, 1919, and had much difficulty in separating the ureters. One large gland adjacent to the right iliac vessels was removed and this proved to be carcinomatous when examined microscopically. On March 3, 1920, fourteen months after the operation, she was again seen by me; she had put on much flesh and looked very well and had no sign of recurrence.

Case VIII.—Mrs. S., aged 57; had passed the menopause seven years and had vaginal hæmorrhage for one year. She had a large friable cervical growth which extended on to the vagina on the right side. Radium was administered in May, 1919, and I did a radical operation on August 25, 1919, but was doubtful if I completely removed the growth on the right side. On May 18, 1920, I heard she was well but not feeling very strong.

Results.—One patient (Case II) died from the effects of the operation. Two patients (Cases IV and V) had recurrence respectively a few months and during the second year after the operation. One patient (Case VIII) is well but it is less than a year since the operation was performed, and one (Case VII) is very well and putting on weight fourteen months after the operation. Cases VI, III and I are very well respectively nineteen months, two and three-quarter years and three years after their operations.

Difficulties of the Operation.—The only difference between these operations and those done without the application of radium is the presence of hard cartilaginous material in the place of the original growth. In the cases in which this had extended completely round the ureters or on to the bladder I found it impossible to complete the operation (six cases altogether). In the other eight cases the growth had not extended so far laterally or in front and I was able to complete the operation, although the technical difficulties were enormously increased in most of the cases by this material, which made the separation of the tissues very difficult.

Diagnosis.—Unfortunately several of the specimens were lost. All these cases were done during the war when the usual routine methods of examination were entirely suspended. This is not a matter of great importance in determining whether the cases were definitely malignant or not, as the radium reduces the malignant growth to scar tissue, often with no trace of malignant cells. In one (Case VII) no trace of malignant tissue could be found in the cervix, but one of the glands removed showed new growth. Advanced carcinoma of the cervix presents no difficulties in the diagnosis and all these cases presented friable deep growths extending on to the vaginal surface of the cervix and in some cases on to the vaginal wall. Each case was diagnosed as "inoperable carcinoma" by myself, and tubes were inserted into the soft tissue by Dr. Burrows, who also carefully examined each case before inserting the tubes.

Increased Operability.—As all these cases were considered to be "inoperable" when I first examined them, the application of radium does definitely increase the operability, but I am not able to give any precise figures. Dr. Burrows has treated many advanced cases for me, but many do not come to see us again as they are instructed to do, and in all probability these include a large proportion of cases in which the symptoms are temporarily cured. Several patients whom I considered operable refused operation, and as I regarded this treatment rather in the nature of an experiment at first, I did not press it, though I should do so now.

METHODS OF ADMINISTRATION OF RADIUM.

BY ARTHUR BURROWS, M.D.

This part of the paper is concerned with the methods employed at Manchester in radium treatment and in giving some indication from the radium point of view of the proportion of inoperable cases of carcinoma of the cervix of the uterus that is likely to become operable.

In the treatment of carcinoma of the cervix by radium, a great deal depends upon the technique and the quantities of radium employed. Occasionally cases are rejected because they are too advanced, and little or nothing, or even harm, is to be expected from radium treatment, but their number is relatively small. Briefly, the main points in treatment are these:—

(1) Many tubes must be buried in the cervix and surrounding tissues.

(2) The largest possible dose must be employed.

The large number of tubes (five or more) allows a big dose to be given with a minimum of local necrosis. The less the necrosis, taken as a whole, the better is the power of healthy regeneration of the cervical and surrounding tissues. A single central tube placed in the cervical canal may give sometimes a good result, but it is not nearly so certain as the many-tube method, nor does it permit of the fairly even distribution of the radium throughout the tumour which is so desirable. Radium emanation tubes are small in bulk, and when screened by pointed tubes of platinum, the walls of which are $\frac{3}{10}$ mm. thick, they are easily pushed into the tumour.

Other metals may be used, when of the required thickness, but they are not so convenient. I usually place a larger central silver screened tube up the cervical canal towards the upper end of the growth.

It will usually be found that no uniformity of good results can be obtained by using less than 120 mg. of radium metal or millicuries of emanation for twenty-four hours. Larger doses may be employed and if necessary the time correspondingly reduced.

The growth must be cleaned as much as possible before the treatment, but cauterization and curettage are not necessary. Douching should be insisted upon daily for six weeks after the treatment, to prevent the formation of vaginal adhesions. Recently I have treated some cases by Viol's method,[1] that is, by burying and leaving in the cervix small unscreened emanation tubes, but I am not yet disposed to give a definite opinion upon the method, although it seems to me to promise even better things than the older ones.

When Dr. Shaw and I were first considering the question of operation following radium treatment, I was not using the present adequate dosage, but I will give a few facts which indicate in some degree the possibilities of increasing the number of operable cases of carcinoma of the cervix of the uterus by radium.

Taking my first 150 cases treated in Manchester, the last application of radium to which was given early in 1917, we have nearly three years in which to observe results, and I find that without operation:—

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[1] *See* Janeway's paper, "The Treatment of Uterine Cancer by Radium," *Surg., Gynæcol. and Obstet.*, December, 1919.

Group A.—One has been well over four years; four have been well between two and three years.

Group B.—Eight cases were observed for periods of one to two years, during which they remained well, but have not recently reappeared for examination; four similar cases to the last kept well from six to ten months.

Group C.—Thirty-four did not return for examination after one treatment. This commonly happens because the patients are free of all symptoms and think that they are cured.

Group D.—Forty-four had signs of disease, but no symptoms for six months or more; five were operated upon by Dr. Shaw and two by other surgeons.

This gives a total of 102 cases accounted for. The remaining forty-eight cases were benefited imperfectly or for less periods than six months or not at all. Nearly half the cases reported well at the end of the year in my Annual Reports recurred subsequently.

I want to bring home the fact that all these cases before radium treatment were regarded as hopeless and inoperable. I think we may safely assume that Groups *A* and *B* became operable; that, say, 10 per cent. of Group *D* became so also; and we know that seven were actually operated upon, making up a total of twenty-eight or nearly 20 per cent.

To look at the matter from another point of view. For the last two-and-a-half years with improved radium methods I have been able to report 20 per cent. of cases as being well at the end of the year. These had nearly all clearly become operable in the hands of an experienced surgeon and if for any special reason a few were not, the number would be more than made up by the improved cases in which the growth had sufficiently receded for operation to be performed. I think that Dr. Shaw has made this clear in his section. In fact, when I analyse my figures again in two years' time I am sure that I shall find that 20 per cent. is much too low.

Thus it would seem that 20 per cent. of inoperable cases of carcinoma of the cervix of the uterus come within the range of surgery after radium treatment.

DISCUSSION.

Mr. Comyns Berkeley: For many years now I have been using radium for inoperable carcinoma of the cervix. I use 250 mg. of radium bromide, the quantity being contained in five separate tubes. I insert the radium in different parts of the growth by means of a trocar and leave it in for eight hours. As the result of my experience I am disappointed as to any lasting improvement. The hæmorrhage and discharge are relieved and in many cases the pain. The treatment destroys the growth projecting into the vagina, and in some cases the cervix disappeared leaving only a small mass of hard tissue at the roof of the vagina. In all the cases, however, that I have traced, except one, the patient eventually died of the disease, which spread internally. The exception was a case in which I had to abandon the radical operation, after opening the abdomen, because the growth was too extensive to remove. I treated this patient afterwards on two occasions with radium. Two years later she returned to hospital with an incisional hernia, and as there was no evidence of growth on bimanual examination I operated upon the patient for the hernia. When the abdomen was opened no trace of the growth could be found. The body of the uterus appeared normal, there was no fixation and no evidence of extension. The hernia was operated upon, but nothing further was done, and after the patient left the hospital she could not again be traced. I would point out how difficult it is, in London at any rate, to trace hospital patients. In collecting statistics I have tried many ways to find where the patient was living. After my letters were returned with the notice that the patient had left no address, I have written to the police, local house agents, postal authorities, and the present tenant of the house formerly occupied by the patient, but in many cases I have not been successful in tracing the patient. With regard to the use of radium, so as to make inoperable cases operable, I have had some experience, but I found that the difficulties of the operation were far greater than those of a radical hysterectomy for advanced disease. The carcinomatous tissue seemed to be replaced by a hard cartilaginous substance, and in some cases it was almost impossible to get the ureters free. I have had no experience of cure in this class of case, the patients dying of a recurrence. I think it should be remembered that experience shows that if the radical operation can be done, apart from cure, the average extension of life, is, as I have shown, about three years. And therefore, the fact that Dr. Shaw and Dr. Burrows have reported that their cases were living one, two and nearly three years after an operation following the treatment by radium, does not carry us much further, except that these cases were inoperable before the radium was used. It would be interesting to hear, later, what proportion of these cases has passed the three years' limit. With regard to the insertion of radium after the radical operation I have not had any experience. I always hesitated to do this because of the serious results I have heard of when this procedure was followed. To insert tubes of radium into the space between the rectum and bladder seems to invite the complication of very serious recto-vaginal fistulæ, and as a fact such have occurred. If the tubes were inserted at the sides of the uterus away from the rectum and bladder, does Dr. Burrows think there would be any danger to the great vessels and ureters? My experience both of carcinoma of the cervix and of treatment by radium is that the effect of the radium does not extend far enough. I have considered the feasibility in these cases of opening the abdomen and inserting additional tubes in the uterus and broad ligaments, leaving them there for eight hours and then reopening the abdomen and removing them. If this could be done without any great risk to the patient some lasting good might result, as many observers are satisfied that the vaginal growth can be made to disappear. Perhaps by this method, that part of the growth which extends beyond the action of the radium when inserted into the vaginal cervix might also be destroyed. The subject is a most important one, and I am sure that the members of the Section are much indebted to Drs. Shaw and Burrows for reporting such an interesting series of cases, and I hope at some future date they will make a further report.

Dr. DRUMMOND ROBINSON: In 1915 I saw a lady, aged 54, who had suffered from more or less continuous vaginal hæmorrhage for about a year. There was carcinoma of the cervix—demonstrated by microscopic examination of a portion of the cervix. Operation was refused. A year later the vaginal walls were invaded and the bases of the broad ligaments were indurated. The patient was treated on several occasions at the Radium Institute. To-day no trace of the carcinoma can be found, the cervix and upper part of the vagina being occupied by dense non-friable tissue. This patient first presented symptoms of the disease six years ago.

Dr. AMAND ROUTH: I have seen symptomatic cures follow treatment by radium, and consider that team work by the gynæcologist and the radiologist as shown in these cases is a hopeful sign, for it is a step towards the time when uterine cancer and fibromyomata may be prevented or cured apart from radical operations.

Dr. R. A. GIBBONS: Although I have not had much personal experience of this treatment of carcinoma of the cervix, it may be of interest to the authors of the papers to know that when the late Dr. Pozzi, the eminent gynæcologist in Paris, was over here just before the war, he discussed this treatment by radium with me. Dr. Pozzi said that he had been working on the subject for some considerable time, and that he had operated on many cases which before the application of radium had been deemed to be inoperable. I gathered that he was collecting these cases with a view to publishing them, in order to show that there was a distinct advantage in treatment by radium. He also stated, in the same way as Mr. Comyns Berkeley has done, that he had considerable difficulty in following up the subsequent histories of the patients. I mention this to show that work has been done on this subject although up to the present, as far as I know, comparatively little has been published. In my own limited experience this treatment has not saved life.

Mr. GILLIATT: When assisting Mr. Comyns Berkeley I have met with the extreme hardness of the tissues described by Dr. Shaw. I feel that the length of time which elapses between the application of radium and the radical operation may have some bearing on the degree of hardness of the tissues. In the cases I have seen the interval was about fourteen days, and I feel that the radical operation may be easier in so far as this induration is concerned if the interval is longer, say six weeks to two months. I consider this is a point on which further experience is necessary before any definite opinion can be expressed.

Dr. HEDLEY: I have operated upon two cases of carcinoma of the cervix which before radium treatment I considered to be quite inoperable. In both the growth on the vaginal walls had disappeared and the cervix on vaginal examination seemed to be normal, but the growth higher and deeper in the cervix was still recognizable after removal of the uterus. The scarring produced by the radium seemed to increase the difficulties of the operations. For the last seven years I have been in the habit of leaving a tube of radium above the cut end of the vagina after removing the uterus in ordinary operable cases. The effect of radium when brought into direct contact with a carcinoma of the cervix is so striking in some cases that I felt in this way it might be possible to destroy any cancer cells left behind in the cut cellular tissue. The results of this method of treatment seem to be promising. I tie a 100 mg. tube of radium sulphate or bromide, surrounded by a 2 mm. filter, into one end of a piece of rubber drainage tube about 6 in. long. After doing the hysterectomy I push the empty end of the rubber tube down the vagina and have it drawn down from below until the radium lies just above the cut vagina. I then close the pelvic peritoneum over it. The radium is removed after twenty-four hours by pulling the lower end of the tubing. Only in two cases have I been suspicious of radium doing any harm. In one case I put 100 mg. into the vagina on five occasions after the first application during the operation. This patient developed small recto-vaginal and vesico-vaginal fistulæ after the last treatment two years after the hysterectomy. In the other case the left ureter gave way three weeks after operation, but I think it possible this was the result of kinking, although the patient had no symptoms suggesting this until the urine began to escape.

Dr. WILLIAMSON: My experience in the use of radium in carcinoma of the cervix has been very similar to that of Mr. Comyns Berkeley. In many cases I have seen the vaginal growth shrink and disappear, the hæmorrhage and discharge cease, the pain is lessened or entirely lost and the general condition improved. Life has been prolonged and from being an almost intolerable burden has become again desirable. If this were all that radium could do it would merit our careful study, but nearly all who have spoken to-night have recorded apparent cures. I can add two to the number. The first, a case of columnar-celled carcinoma of the body, in which all traces of the growth have disappeared and the patient is in good health seven years after the last application of the radium; the second, a case of recurrence of the growth after Wertheim's operation for carcinoma of the cervix; this patient remains free from symptoms or physical signs of growth five years after the last application. Both these patients were treated by the equivalent of 210 mg. of radium bromide for periods of twenty-four hours; the applications were repeated at the end of a month and then at increased intervals. In a series of cases treated these are the only two patients, as far as I am aware, who lived for more than three years. The usual course of events, as I have seen it, is the sudden recrudescence of growth widely distributed in the pelvis after a period of apparent quiescence. I have attempted operations after treatment by radium in five cases. In two I abandoned the procedure on account of the density and fixation of the tissues around the ureters; one patient died forty-eight hours after the operation, and two are still alive and apparently in good health eighteen months and six months respectively from the date of the operation. I have on several occasions introduced radium through the vagina into the pelvis twenty-four hours after Wertheim's hysterectomy; one of these patients developed a vesical fistula, which I have not been able to close. I have not seen harm result in any other case. From what I have seen in my own cases, and from what I have read and heard of the experiences of other gynæcologists, I believe that radio-active substances will eventually replace the knife in the treatment of carcinoma of the uterus. At present the science of radio-therapy is in its infancy, and much has yet to be learnt both with regard to dosage and to methods of application. The effects of sublethal doses require further investigation. It has been shown in mice that cancer-cells which have been subject to radiation of sufficient intensity to cause loss of reproductive function, and to produce certain changes in the nuclei but not to kill the cell, have the power of conferring immunity against the same strain of tumour (Morson). These experiments suggest that possibly we are wrong in using large doses, and that smaller doses applied in several parts of the growth may be more effectual in bringing about a permanent cure. The vast importance of the subject calls for a collective investigation in which the surgeon, the radiologist, the physicist and the cytologist must combine. At present individual investigators are working empirically with no established units of dosage and employing a nomenclature which is confusing and often misleading: it is time these things were corrected.

The PRESIDENT: Dr. Burrows mentioned the possibility of doing harm by applying radium. I have seen two cases, one of advanced inoperable cancer of the uterus and one of recurrent cancer after excision of the rectum, in which treatment by radium was not only useless but it appeared to make the growth extend with greatly increased activity. It would be instructive if Dr. Burrows could throw light upon the kind of case in which this is likely to happen. In the first of the cases I have mentioned the patient was under 30 years of age, and in the second she was under 40, and this comparative youth may have influenced the results. These experiences made me rather avoid the method, but recently I have had a much more satisfactory case. It is almost a repetition of one mentioned by Dr. Williamson. In 1912 Wertheim's hysterectomy was performed for an early carcinoma of the cervix, and in 1915 the patient returned with a small recurrence at the top of the vagina, which was verified as a squamous-celled carcinoma by excision of a piece and by microscopic examination. This was treated, in association with Dr. Finzi, by an application of 150 mg. of radium bromide in two 2-mm. tubes of platinum for twenty-four hours, followed by several smaller doses of shorter duration at extending intervals. There appears to be a complete cure. About two years ago the vagina was to a great extent obliterated, but there was no hardness nor other sign of cancer. The lady is at present in France, but I had a letter from her yesterday saying that so far as symptoms go she is quite well.

Dr. W. FLETCHER SHAW (in reply): I thank the members for the interest they have shown in the paper by so many taking part in the discussion. This interest indicates dissatisfaction with the present position of our treatment of carcinoma of the cervix, but it also shows that advance has been made, and all are stimulated to improve the position still further. When I was a resident in St. Mary's the only operative treatment performed for the early cases was vaginal hysterectomy, which was proved to be worse than useless. Later came the general adoption of Wertheim's operation, then radium, and now I and Dr. Burrows advocate the combination of these two methods. One point raised in the discussion is the time which should elapse between the application of radium and the Wertheim operation. In the advanced cases reported this evening it was necessary to wait two months to allow of contraction of the growth by the action of the radium, as it was impossible to remove the growth when first seen; but in earlier cases, which in future I intend to have treated by radium previous to the performance of Wertheim's operation, I intend to attempt the operation the day after the radium has been applied, and so to avoid the sclerosing action of the radium on the tissues outside the growth. This method was adopted in one case by Dr. Donald with happy results. I think this method preferable to administering the radium after the operation, as there should not be the same liability to sloughing of the tissues. Mr. Comyns Berkeley stated that the duration of life after a Wertheim's operation is about three years: this is probably correct, but during a large portion of this time the patient is suffering from the effects of the recurrent growth. In my own experience, when recurrence has occurred, there is usually some sign of it before the end of the first year, the patient ceases to improve in health, and then gradually falls back until definite symptoms of recurrence can be observed. In the cases reported in this paper four of the patients are well over the first year, one three years and two months, and all are putting on weight, have regained their strength, have a good colour, and are feeling very well, quite different to the type of case which drags out an existence with recurrence until the third year. The method of applying the radium and the method of application I do not pretend to know anything about, and leave this entirely to Dr. Burrows. If radium is to be the benefit I hope it ultimately will prove to be, it is necessary to experiment with various doses and methods of application, and this is such a big subject that I consider it should be left in the hands of an expert who gives his whole mind to this subject.

Dr. BURROWS (in reply): It is difficult to comment advantageously on the considerable number of cases that have been quoted during the discussion.

and the varying opinions that have been expressed as to the value of radium treatment, for the reason that there appears to be no uniformity of method in the treatment of the cases quoted. The quantities vary, the screens vary, and their nature and thickness is often vaguely described. The time and frequency of application are quite different from one another, and confusion arises from the use of radium bromide as the standard of measurement. I do not think that any uniformity of results can be obtained when using screens of three-tenths of a mm. of platinum, if the equivalent of a less quantity than 120 mg. of radium element is used to treat each case. Moreover, I am certain that the quantity should be divided among five or more tubes buried in and round the cervix of the uterus. Dr. Drummond Robinson raises the question as to whether radium treatment should be entirely in the hands of experts or if it should be applied by the general gynæcologist or others. It is difficult for an expert to say that he is essential, but I do think that some special knowledge of radium, its physics and action, leads to more scientific methods of application, and, as a rule, more reasonable grounds are found for varying and changing the method of treatment. In any case I think he helps on the general progress of the subject. I should here like to point out that we have been mostly talking of the treatment of carcinoma of the cervix of the uterus by heavily screened tubes. The new method of burying unscreened emanation tubes into the cervix, devised by Dr. Viol of New York, is a treatment which has great promise. Reverting to the subject of the standard of measurement for radium, I think it would be much better if the old commercial standard of hydrated radium bromide were dropped, and the more scientific method of estimating strength in terms of radium element were adopted, as is general in the United States of America. In addition there is the advantage that when radium emanation is used, the unit is the curie from millicurie, and the unit of radium emanation corresponds to measurements in terms of radium element.

With reference to the more immediate subject of this discussion—viz., operation after radium treatment—it is obvious that there are considerable difficulties in performing hysterectomy a considerable time after an application. I certainly think that the question of operating immediately after an application should be seriously considered. The action of the radium on the tissues left would not be interfered with. The operation would be much more easily performed, but the possibility of sloughing following the treatment would have to be taken into consideration, and only further experience can let us know if there is a real danger of it occurring. Prophylactic treatment following an ordinary hysterectomy for carcinoma of the cervix of the uterus has been successfully employed, and I think should be investigated as likely to lead to profitable results.

With regard to the effects on the malignant tumours produced by radium, there is no doubt that local sloughing may occur round the inserted tube, but in carcinoma of the cervix this is never a matter of much importance, and I have never found it necessary to cover my tubes with rubber or any substance to cut off the secondary radiation if they are buried. There is more likelihood of well-marked sloughing occurring round a single large tube than there is round a number of smaller, separate tubes when they are inserted into a tumour. The radius of the therapeutic action of radium in the tubes used in the treatment of carcinoma of the cervix of the uterus is 1 cm. to 2 cm., varying somewhat with the strength of the tubes. In this respect the use of multiple tubes is of great advantage in extending the area of action of the radium and at the same time diminishing local necrosis.

In treating a large number of cases of carcinoma of the cervix it is found there is a certain proportion in which it is better not to apply the radium, owing to the fact that no benefit is likely to result from the treatment and in some cases possible harm. Thus, if the growth is extremely large and adherent to the bone of the pelvis, an ulcerated cavity may be the only result of radium treatment. If there is a large excavating, thin-walled ulcer, as a rule radium is of no value. Extensive involvement of the vagina is another contra-indication, and tumours ulcerating through into the bladder, ureter, or rectum should be avoided, as there is in these cases, after radium treatment, always some loss of tissue, which in such situations cannot as a rule be repaired. The work that has been done on the irradiation in tumours in mice and rats has been very suggestive of the possibility of giving a dose of radium sufficient not to destroy the tissues, but only to set up a process of automatic repair and regeneration. Although this method is one of scientific promise, so far as I know at present nobody has been able to devise a method by which it can be satisfactorily applied to human beings.

Lastly, I should like to draw attention to the fact that results from the treatment of inoperable carcinoma of the cervix by radium are almost always compared with those obtained from operative treatment of early carcinoma of the cervix, and although radium does not come out badly from the test, it is obviously a very stringent and, I think I might say, unfair comparison. What really is the fact is that in treating these hopeless inoperable cases either by radium alone or by radium followed by operation, every single case cured is a great step forward, as no other treatment has so far been able to do so much. There is nothing to compare it with but previous utter failure, and the results from radium treatment are such that in the future it is possible that it may become the sole treatment for carcinoma of the cervix of the uterus.

Case of Spontaneous Rupture of the Uterus following an Administration of Pituitary Extract.

By ALECK W. BOURNE, F.R.C.S.

THE patient was a 12-para who began labour at 4 a.m. on May 13. The last child was born two years ago. The pelvic measurements were normal, and previous labours had given rise to no difficulty. Pains were sluggish during the day, and in the afternoon a sedative was given on account of primary inertia. By 9 p.m. the os had become fully dilated, and the head was bulging the perinæum, but as pains seemed ineffectual, a $\frac{1}{2}$ c.c. of pituitary extract was given hypodermically. Very little change occurred for an hour, and no progress was made, when at 10 p.m. the pains suddenly became extremely violent, being described as almost tetanic. The patient was not crying out, and the head was in the act of being born when she suddenly gave a sharp cry and collapsed. The head receded, pains ceased, and the pulse was not perceptible.

I saw her on admission to St. Mary's Hospital at 11 p.m. There was profound shock, no pulse at the wrist, and very slight hæmorrhage from the vagina. The abdomen was soft, rendering the palpation of the fœtal parts very easy, and fluid dullness rose high in the flanks. As the patient was too deeply shocked to be operated upon immediately she was given an intravenous injection of gum, $\frac{1}{4}$ gr. of morphia and other restorative measures. The pulse under this treatment soon returned to the wrist, and, with considerable general improvement, the head was removed by perforation and cutting through the neck. It was found to be impossible to extract the whole child by a preliminary gentle traction. The head was in the persistent posterior position, but small, and came away with ease. The simple manipulation of delivering the head, however, caused a further sudden collapse with loss of pulse and colour. One and a half pints of the husband's blood, which had proved suitable, was injected by Mr. Bryan, but the patient did not rally, and died at 1 a.m.

The post mortem showed the child's trunk and placenta free in the abdomen, with the uterus retracted behind it, and a large quantity of free blood. There was a tear of 7 in. in the peritoneum of the anterior leaflet of the broad ligament, opening up the utero-vesical pouch anteriorly. The rent in the uterine wall was 5 in. long, beginning at the left lateral wall of the lower segment, and extending downwards and inwards to involve the cervix. The bladder was uninjured. The lower segment was extremely thin. Sections at the site of the lesion showed an extreme degree of fibrosis, with very few muscle fibres to be seen. Also, there were a large number of capillary vessels, with some extravasated blood. Sections stained by Sudan III showed practically no intracellular nor other fat, no more than in the case of a normal full term uterus used as a control.

The case is noteworthy on account of the rupture occurring in the absence of any obstruction whatever, after a sluggish and short labour, and when the head was in the act of being born.

DISCUSSION.

Dr. WILLIAMSON: I have seen a case of spontaneous rupture of the uterus follow the use of 1 c.c. of pituitary extract. The patient was a multipara and the extract was given when the cervix was two-thirds dilated: after its administration the pains were violent and the child was born rapidly. Two hours later the patient suffered severe pain over the left iliac fossa, she became breathless, and her pulse increased in rapidity. Next day a swelling was found over the left iliac fossa and on vaginal examination a rent was discovered involving the cervix and lower uterine segment on the left side. The swelling was evidently a hæmatoma in the broad ligament due to rupture of branches of the uterine artery. I have seen another evil effect follow the use of pituitary extract; in this case I found a ring of firm contraction round the neck of the child whilst the rest of the uterus above and below the ring was in a state of relaxation. Injections of morphia and atropine failed to relax the ring and the child died before it could be extracted. These accidents are doubtless both rare, but they constitute a real danger and I believe, and I teach, that with the possible exception of cases of accidental hæmorrhage pituitary extract should not be administered until after the birth of the child.

Dr. FLETCHER SHAW: Mr. Bourne should be thanked for recording this case. A large number of cases of rupture occur through the administration of pituitary extract and I think these should be recorded as a warning. Unfortunately pituitary extract is very largely used to save time, but I teach my students that it must never be given until after the termination of the second stage, except for accidental hæmorrhage. I have seen two cases of rupture, both multigravidæ, who had no trouble with their previous confinements. In both these cases pituitary extract was given before the cervix was dilated, was followed by very severe pain and quick expulsion of the child and in each case after delivery there was found to be a large tear of the cervix extending into the broad ligament and in one case through the peritoneum: this case terminated fatally.

Mr. EARDLEY HOLLAND: In my opinion pituitary extract may be a source of great danger both to the mother and to the fœtus. I have noticed its harmful effects in three different classes of cases. In the first place I have met a case of rupture of the uterus in which the accident was almost certainly due to the violent action of pituitary extract; as the clinical history of this case is the same as all those related by the preceding speakers I will not recount it. In the second place I have seen the dangerous effects produced by the so-called sensitization of the uterus by the administration of pituitary extract preliminary to the induction of bougies. In two cases of this I have seen tonic contraction of the uterus: in one the uterus was ruptured by version and in the other the uterus gripped the fœtus so firmly that delivery of a dead fœtus was extremely difficult; in another, tonic contraction of the uterus was impending, but was not given time to develop. Thirdly, I am convinced that pituitary extract is often responsible for the death of fœtuses; I have performed post-mortem examinations on at least four fœtuses with tears of the tentorium cerebelli and cerebral hæmorrhage consequent on a precipitate ending of the second stage of labour brought about by the violent action of pituitary extract. Such cases as I have related are no doubt rare, but they are sufficiently common to warrant us, as teachers of students and post-graduates, in urging caution in the use of so powerful a drug.

Dr. FORD ANDERSON: Was Bandl's retraction ring present in this case? Speaking generally, I would suggest that pituitary extract, if used at all before the birth of the child, should be used with all the care and all the precautions which have always been enjoined when ergot of rye has been administered at such a time.

Mr. BOURNE (in reply): Many cases of rupture of the uterus have occurred when pituitary extract has been administered before full cervical dilatation, but the present case is almost unique from the fact that the head was on the perinæum when the accident happened. Further, also, I think that the condition of extensive fibrosis of the lower segment was an important predisposing factor.

Section of Obstetrics and Gynaecology.

President—Mr. J. D. Malcolm, F.R.C.S.Ed.

Primary Carcinoma of the Fallopian Tube.[1]

By J. D. Barris, F.R.C.S.

The patient was a married woman, aged 55, who was admitted to the Royal Waterloo Hospital on May 13, 1920, complaining of bleeding from the vagina. She had been pregnant three times, and had had three children, the last thirty years ago. The menopause occurred at the age of 52½. Since then she had been in her usual health until November, 1919, when she first noticed bleeding. The bleeding recurred at such frequent intervals that it was almost continuous, and she began to lose weight.

On examination the uterus was not palpably enlarged, but was displaced forwards and to the left by a cystic swelling which almost filled the pelvic cavity. The cervix appeared healthy. Laparotomy was performed on May 18, 1920. The right Fallopian tube in the recent state was distended, shiny, smooth, and measured 10 in. in length and 12 in. at its greatest circumference. It was kinked upon itself and lay mainly above and behind the uterus; the abdominal ostium was closed. The uterus was not enlarged; scattered over its anterior surface were several minute firm nodules. Two similar nodules lay immediately below the insertion of the left tube to the uterus and another nodule at the fimbriated end of the left tube. The right ovary was closely attached to the right tube and was about twice the average size; the left ovary appeared normal. The uterus and its appendages were taken out *en masse*, care being taken that the dilated right tube was not ruptured, as it was recognized that the case was malignant, from the presence of the nodules. No secondary deposits were visible on the abdominal peritoneum, and no enlarged glands were palpated. There was no ascites. On laying open the uterus and cervix no evidence of malignant disease was found. The right tube was then opened longitudinally. The wall was thin and translucent with scattered opaque areas. The cavity contained a large quantity of altered blood-stained fluid and numerous masses of papillomatous growth adherent to the inner wall. The right ovary when laid open showed several secondary deposits in its upper part; the lower part contained two cysts the size of a cherry.

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[1] At a meeting of the Section, held July 8, 1920.

Microscopic section shows the growth to be a columnar-celled papillomatous carcinoma. The cystic portion of the right ovary showed no evidence of malignancy.

I thought the specimen a sufficiently rare and beautiful one to merit bringing before your notice, and would put forward the following comments: First, that the classical symptom of watery discharge was not present; secondly, that the tube had at the operation the appearance of an ordinary large hydrosalpinx; and, thirdly, that the case shows the advisability of hardening specimens intact before subjecting them to examination. This specimen has unfortunately shrunk considerably in the processes of hardening since it was first opened.

Dr. HERBERT SPENCER: In addition to the four cases already published, I have recently met with a fifth case of primary cancer of the Fallopian tube at a post-mortem examination of a patient who died of multiple secondary growths in the peritoneum. It is a pity that Mr. Barris's specimen was not hardened before opening it. It seems probable that the carcinoma developed from a papilloma occurring in a hydrosalpinx.

Specimen of a Fibroma of Ovary.

Shown by ALFRED RICHARDSON.

THIS is a specimen of a solid pedunculated tumour arising from one pole of an ovary. It was removed from the abdomen of a young girl, aged 16, who had no symptoms beyond a few dyspeptic pains after meals, but was otherwise perfectly, in fact exceptionally, well formed and healthy. The tumour may be said to have been accidentally discovered during the course of a routine abdominal examination for the above mentioned dyspeptic symptoms. The menstrual history was normal.

Clinically the mass was hard, nodular, centrally placed and rising out of the pelvis to a point 2½ in. above the symphysis in the mid line. It was movable from side to side, but in a vertical direction movement was not noted. On vaginal examination under an anæsthetic it was recognized that the tumour was separate from the uterus, and it was therefore labelled ovarian and possibly sarcomatous in nature.

The only feature of note about the operation was the presence of a small quantity of free fluid in the peritoneal cavity. Otherwise being pedunculated and free from adhesions, the mass was very easily removed. On examination it turned out to be a roughly ovoid solid growth (5 in. by 4 in.), with a very well-marked warty surface. The warty processes were coarse and in parts separated by fissures of 1 in. to 1½ in. in depth, though over the greater part of the tube they were sessile in character. Microscopically the tumour was a "fibrifying fibroma" with a certain amount of myxomatous degeneration.

I am showing the tumour not so much on account of its being a fibroma of the ovary, but on account of its curiously papillomatous surface, and I shall be very interested to know if any surgeon present can give details from his experience of a growth of similar nature.

I am much indebted to Dr. J. S. Fairbairn for permission to show the specimen.

Dr. HERBERT SPENCER: I have not seen such an extensive mass of "fibrotic warts" as are shown in this specimen of fibroma. Nor have I observed an ovarian fibroma from such a young patient. In one case I saw, twelve years after a total hysterectomy for myoma, a lobulated tumour as large as a double fist develop apparently in the left ovary: the lobulations were larger and smoother than in the specimen shown to-night. It was adherent to the sigmoid mesocolon, but was easily removed. It looked on section like a fibroid, but microscopic examination showed it to be a spindle-cell sarcoma. I would suggest, in view of the youth of the patient, that sections should be cut in several places in order to exclude sarcoma. The sections under the microscope certainly appear to be those of a fibroma.

ADDRESS BY THE PRESIDENT.

Some Points concerning Tumour Growth.

Many diseases are caused by micro-organisms and there can be no doubt that others arise in the same way although the life-history of their particular germs may not be known. Following a line of thought suggested by these considerations it has been argued that the discovery of a specific organism which is the cause of cancer must be only a matter of time. This reasoning assumes much that is not obvious.

In diseases of parasitic origin no symptoms except those from pressure and exhaustion are caused directly by the parasite. A guinea-worm may be felt under the skin, but symptoms do not develop until an inflammatory pustule forms. A hydatid cyst may be obvious to sight and touch without attracting attention in any other way. A symptom of disease caused by a micro-organism always manifests itself as a response of the tissues to an irritation created by the microbe or as an effect of destruction of tissue. In scarlet fever the cause of the disease enters the body of its victim and remains there from one to eight days before the fever begins. Then an inflammation of the throat occurs followed by congestion of the skin. So in diphtheria there is an inflammation of the throat and in typhoid fever Peyer's patches are affected. Why the reaction occurs in different parts of the body is not known. It seems likely that in diseases which have an incubation period all parts are exposed to irritation although only some react. Possibly aërobic conditions influence the result. The nature of the irritant also has its effect, for a scarlet-fever throat differs from a diphtheritic throat. But just as a guinea-worm lives under the skin and a hydatid cyst in the internal organs, so various microbes may find particular localities easy of access or favourable for the development of their irritating qualities. The tubercle bacillus frequently affects the lungs, but no part of the body seems exempt from its evil influence. Tubercles are collections of cells brought together for the purpose of destroying and removing the bacilli. They may exist without symptoms. In actinomycosis a swelling forms by a development of actinomyces and of cells of the body which collect in response to the irritation of the mycelium. The large mass of inflamed tissue which sometimes arises in the pelvis in puerperal cases or around an inflamed appendix appears to be a response to a mixed infection.

An inflammatory development of cells may be brought about without the action of micro-organisms. Sterile turpentine introduced into the tissues causes pus formation. The capsule around a hydatid cyst or around a piece of sterile metal is caused by a mechanical irritation and is associated with a development of cells into fibroid tissue, but no micro-organisms are concerned in the process and Lister taught that some degree of inflammation and development of cells is essential to all healing of wounds. In tetanus evidence of inflammation is wanting, but it is obvious that selected cells of the nervous system are irritated by a poison just as when an overdose of strychnia is taken.

Thus, when a swelling is caused by the action of parasitic micro-organisms, it is of inflammatory origin and associated with an excessive growth of cells which are derived from the normal cells of the body.

There are several possible causes of an excessive development of cells. In the lowest forms of animal life reproduction is by fission and by gemmation. In the higher types, it is effected by the union of a germ cell with a sperm cell. In a few cases it is by parthenogenesis.

There are no other known methods of reproduction, but the bodies of all the higher animals are formed, at first, in miniature. Then they grow. Every muscle is complete at an early stage of intra-uterine life, and its enlargement must be by a multiplication or creation of muscle cells. So the uterus is developed; it increases to its full size at puberty and if conception takes place its enlargement is proportional to that of the fœtus. Again an obvious increase of adipose tissue is common and sometimes excessive after middle life. Emaciation may also take place and the uterus diminishes in size after delivery and after the menopause.

These things happen and multiplication of living cells of any kind is therefore easily understood. How it is controlled so that the various parts retain their shape, and how growth is normally arrested at the proper times, are questions almost as obscure as the origin of life. We do know that a good supply of nourishment, as opposed to starvation, is important and that a proper supply of internal secretions influences the rate of growth, the size attained, and the physiological efficiency of various structures.

When an excessive formation of white cells takes place as a consequence of irritation of tissue the explanation cannot be found in any sexual activity. The increased growth seems closely to resemble reproduction by fission, but its object is not reproduction. The excessive development depends upon an inherent power of the cells of the body to multiply for its growth and nourishment, and for the protection and healing of injured parts. This protective and reparative power is a normal attribute of inflammatory cell growth. It comes into action when the umbilical cord separates, and it is necessary for the healing of wounds which are unavoidable accompaniments of animal life.

Many believe that cancer is caused by a specific micro-organism, but there is no rational basis for the reasoning that because an irritation of tissue by parasites often leads to an inflammatory excessive growth of white cells therefore the development of a neoplasm must be caused by parasitic microbes. The things compared are essentially different.

Like inflammatory cells, the cells of a cancer, of a sarcoma, and of every innocent new growth are developed from normal cells of the body. Cancers and adenomata arise from epithelium, and they differ from each other in the fact that cancer cells penetrate the surface from which they grow, passing into the tissues, and through the lymphatics to neighbouring glands and to distant parts. They retain the power to increase and multiply and form tumours wherever they are. On the other hand an adenoma is an encapsuled growth without any power to pass into the tissues or to invade distant parts. It is harmful only by causing pressure and by appropriating the nourishment intended for the normal body. A sarcoma and a fibroma do not grow from epithelial cells, and they have a similar relationship to each other that a cancer and an adenoma have. Difficulty sometimes arises in deciding whether a swelling is innocent or malignant. Certain tumours of the kidney in children invariably cause death if not removed. Their excision is fatal in about half the cases operated upon, and Bland-Sutton[1] asserts that of fifty patients who recover forty-five die within twelve months. He found only two really satisfactory cases which survived more than four and a half years. One of these, operated upon by Dr. Robert Abbe of New York, was described[2] by him as a unique case in 1912, the patient being alive and well twenty years after the operation. The other case was under my care. A tumour measuring 6 in. by 4 in. was attached to the anterior surface of the right kidney of a child just under 2 years of age. It consisted of tubular and solid columns of epithelial cells, and Mr. Targett described it as a malignant adenoma. But the structure differed from that usual in these growths by showing no sarcomatous and no unstriped muscle cells, and when publishing[3] the case I suggested that the healthy condition of the patient might be accounted for by the peculiar microscopic characters of the tumour. Mr. Shattock recently re-examined the growth, which is preserved in the Museum of the Royal College of Surgeons, and he came to the conclusion that it is not a malignant but an innocent adenoma. This revised opinion and the fact that the patient is in good health nearly twenty-eight years after the operation emphasize the importance attached to the microscopic structure of the tumour. The specimen illustrates the difficulty which may arise concerning the nature of a neoplasm.

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[1] Bland-Sutton, "Tumours, Innocent and Malignant," 6th ed., 1917, p. 110.

[2] *Annals of Surg.*, 1912, lvi, p. 469.

[3] *Trans. Clin. Soc. Lond.*, 1894, xxvii, p. 94; 1895, xxviii, p. 287; 1903, xxxvi, p. 233.

This difficulty was also found in a tumour which I brought before our Section as one of papilliferous adenoma complicating a fibromyoma of the uterus.[1] The specimen showed a diffuse growth of the mucous membrane of the corpus uteri, and microscopically adenomatous tissue separated to a very slight depth the muscle fibres of the uterus. The question arose as to whether a cancer existed or whether non-malignant glandular tissue penetrated the muscular wall where the mucous membrane normally does so. The Pathology Committee of the Section concluded its report with the statement that "the opinion of the members present (to-day) was divided on the question of malignancy of Mr. Malcolm's specimen."

A spindle-celled sarcoma and a tumour consisting of fibrous tissue may resemble each other so closely that no decision as to their nature is possible from a naked-eye inspection, and even microscopic sections may give rise to differences of opinion.

Although the dividing line between innocent and malignant growths is sometimes ill-defined, yet all neoplasms resemble inflammatory swellings in the fact that their cells are developed from normal cells; but there is this great difference that in an inflammatory swelling the new cells are concerned with the protection of the body, the destruction of invading parasites, and the healing of injured tissues, whereas these statements are not applicable to the cells of any kind of new growth.

Sir Henry Butlin described cancer as a parasite. He said cancer cells behave exactly as the parasitic protozoa do, possessing, like them, only two attributes, to live and to reproduce. Cancer cells can be transplanted from one animal to another of the same species, and from the second to others, so that the tumour may live indefinitely, and cancer cells have been grown in artificial media. But Butlin expressly declared that cancer cells arise from the normal cells of the animal body, and this differentiates cancers from all parasites. A parasite is a living organism which preys upon *another* living organism. This meaning is well established, and although cancer cells act as parasitic cells, and the resemblance of their life history to that of the parasitic protozoa is exceedingly close, a cancer cannot be described as a parasite unless a new definition of the word is created and accepted. To use the word parasite in this sense can only lead to confusion.

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[1] *Proceedings*, 1915, viii, p. 95.

In the same way, neither a sarcoma nor any innocent tumour can properly be called a parasite. But they all behave as parasites, living on the nourishment which ought to be used for the sustenance of the body. If they grow sufficiently quickly and sufficiently large they cause emaciation and death.

Some so-called innocent tumours are clinically malignant. They may grow indefinitely, and some of them invariably prove fatal. Many fibromyomata of the uterus when they attain a certain size cease to grow, or enlarge very slowly, and a really big tumour of this kind is hardly ever seen in these days of active surgery, but formerly such tumours sometimes attained to enormous dimensions. When they came well out of the pelvis they might hang forwards against the abdominal wall so as to make little pressure on important parts, and then they caused death by exhaustion. Ovarian tumours often grow chiefly by an increase in the fluid secreted within them. The following case shows the importance of this. In 1899 John Langton attempted to perform ovariotomy, and failed. In 1906 I tried again, and opened a multilocular cyst, obviously of ovarian origin, but failed to find any part of the peritoneal cavity. This patient has been relieved by tapping from 1893 onwards. Since 1904 Dr. G. A. Gibb has kept an accurate record. In that year there were four tappings, and these have gradually increased until now they are necessary about every ten days. In all there have been 308 tappings, and the amount withdrawn since 1904 is 423 gallons. It is estimated that since 1893 the total fluid removed is over 450 gallons, which would weigh about two tons. The patient, now 67 years old, eats enormously, and has no bowel troubles except just before the tappings. Dr. Gibb has sent me the following inscription from a tombstone in Bunhill Cemetery, City Road, E.C.:—

> "Here lies Dame Mary Page, relict of Sir Gregory Page, Bart. She departed this life March 11th 1728 in the 56th year of her age. In 67 months she was tapped 66 times: Had taken away 240 gallons of water without ever repining at her case or ever fearing the operation."

A non-malignant ovarian tumour is a fatal disease unless the surgeon intervenes, and these tumours occasionally show another characteristic of malignancy. "Should rupture of the cyst occur, detached portions of epithelium may become implanted upon a peritoneal surface or upon the raw tissues of a wound, and these grow—reproducing in their growth the characters of the parent cyst. These secondary growths, however, are not malignant, they do not invade and destroy the tissue upon which they have become implanted, they persist merely as surface growths."[1]

In ovarian tumours the line of division beween those of innocent and those of malignant nature seems to be imperceptible, and when the whole subject of new growth is considered the most reasonable view seems to be that both innocent and malignant neoplasms arise because of an inherent power of the cells to develop in the various ways which eventuate in the formation of the different kinds of tumours.

That tumour growth may be stimulated by circumstances is probable, but that it can be created by a specific micro-organism is incredible. The initiation of a growth of body cells into tumour cells, whether innocent or malignant, by the action of a parasite, presupposes a power of fertilization exercised upon the normal cell by the parasite, and, if we consider that such near species as the horse and the ass, the canary and other finches, when they breed together, produce sterile offspring, the idea that a creative influence can be exercised by any living micro-organism on the cells of an animal body seems to be irreconcilable with all facts at present known.

That many stimulations of tissue favour a development of cancer is obvious. Examples are seen in chimney sweep's cancer, smoker's cancer, and some effects of X-rays. An association of cancer with irritated scar tissue is also recognized. It would appear, however, that just as a development of inflammatory cells could not take place in response to irritation without an inherent power of increase and multiplication within the body cells, so the growth of cells in a neoplasm must depend upon an inherent power in these cells to develop into tumours. If this be true, it is obvious that, although various irritations may stimulate tumour growth, nevertheless tumours may develop without any outside stimulating influence.

Dr. J. R. Goodall, at the meeting of this Section held on February 5 of this year,[2] suggested that dermoid ovarian tumours arise by a process of parthenogenesis. I take it this means that there is an attempt to reproduce the species. But a dermoid ovarian tumour consists of a higgledy-piggledy conglomeration of more or less normal body structures having no resemblance to the animal from which it grows. There are few complete parts, and there may be enough teeth for a family. Parthenogenesis is a recognized mode of development of certain creatures without the intervention of sexes, and it may be argued that, apart from inflammation, every abnormal development of cells is an attempt at reproduction without sexual influence. A new and partially independent living structure is created. But, in attributing any new growth to parthenogenesis, we must alter the meaning of the word, for parthenogenesis is a complete and perfect process. Every new growth may perhaps be correctly described as an abortive attempt at parthenogenesis arising from an inherent power of the tissues to undergo erratic development.

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[1] Williamson and Barris, "On the Occurrence of Carcinoma in Cystic Teratoma of the Ovary," *Journ. Obstet. and Gynæcol. Brit. Emp.*, 1911, xx, pp. 211-229.

[2] See *Proceedings*, 1920, xiii, p. 85.

This view is irreconcilable with the belief that cancer is caused by a parasite. There have been many attempts to find a specific cancer-producing organism, but, like a will o' the wisp, it has never been caught. Fibogen,[1] of Copenhagen, has recorded that rats, when fed on cockroaches containing a nematode named *Spiroptera neoplastica*, develop cancer in a large proportion of the cases so treated. The eggs of the nematode were seen lying in the upper layers of the epithelium of the stomach wall of the rats. But some of the animals developed benign epithelial proliferations, and evidences of inflammation were almost invariably found. That it is possible to stimulate cancer formation in rats is a most important fact, but there is no evidence that the *Spiroptera neoplastica* acts in any way except as an irritant, stimulating a growth of cancer which seems to be readily developed in rats. That there is a specific action of the *Spiroptera neoplastica*, or of its eggs, upon epithelial cells, which changes them into cancer cells, has not been shown.

If we accept the view that a specific creative effect produced by a micro-organism upon a cell of the body of one of the higher animals is a biological impossibility, and that tumours arise from an inherent power of erratic growth in the body cells, there seems to be some hope that increasing knowledge of the internal secretions may offer an explanation and perhaps provide a mode of influencing tumour formation. It is known that some of these secretions directly control growth.

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[1] *Brit. Med. Journ.*, 1920, i, p. 680.

The development of uterine fibromyomata during the child-bearing period and the common occurrence of mammary and uterine cancer when menstruation ceases may well be due to some influence which exists or undergoes a change at these times. On the other hand, a study of the cancer of old age suggests a search for that mythical dream—perpetual life, and the sarcomata of children seem equally incapable of control. Nevertheless, so many surgical and scientific advances have been made within the memory of many here present that there is no occasion for despair about the eventual discovery of the cause of cancer. But in these remarks I have stated fundamental reasons why the quest for a specific cancer-producing micro-organism must be futile.

I

PROCEEDINGS

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SESSION 1919-20

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Section of Odontology.

President—Mr. J. F. Colyer, F.R.C.S., L.D.S.

The Debt of the Dental Profession to John Hunter.[1]

By J. F. Colyer, F.R.C.S., L.D.S.

In the Annals of the Barber-Surgeons it is recorded that "John Bryckett toothe drawer," was admitted "for a brother into the house." This record is dated September 10, 1551, and it would appear that Bryckett died a few years later, as, from a further record, dated August 26, 1557, we learn that Mrs. Dawson, the widow of one Bryckett, "a Toothe drawer, shall paye no quartryge to the hawle nor hange oute any signs or clothe with teethe as she heartofore hath don." This record suggests that Bryckett's widow had been guilty of the offence of "covering," incidentally it discloses to us the outward and visible signs which at that time announced the whereabouts of a dental practitioner.

The Annals also show that on November 23, 1557, a "licence" was granted to one William Thomlyn "to drawe teethe and to make cleane teethe and no more." As the Barber-Surgeons exercised considerable control over the practice of surgery and required of their members a certain standard of professional knowledge, we may safely assume that a "licence" to perform dental operations was granted only to a person who already possessed some degree of skill.

In those days a Londoner who needed the aid of a dentist was probably more fortunately placed than a Provincial, if we may judge of the country practice from a signboard which was unearthed at Ottery St. Mary, Devon, and which is now in the possession of the Royal Dental Hospital. This signboard announced that, "The Smith Glazier Let Blood & Draw Teethe, Tea Kittels & Potts, Buckills Lantren Cups To be Handeld Heare."

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[1] President's Address delivered at a meeting of the Section, held October 27, 1919.

Coming down to the days of John Hunter, we gather that the practice of dental surgery was not altogether neglected by men of education such as Berdmore, who was dentist to King George the Third, and was also a member of the Surgeons' Company. In 1770 Berdmore published a "Treatise on the Teeth," from which we learn that dental operations were then performed mainly by barber-dentists, and also that, for the cure of the toothache, the virtues of the loadstone were extolled and charms and incantations were practised by vagrant mountebanks and impostors. In those days the practice of dentistry, even by the more educated practitioners, was empirical, and it could hardly be otherwise in view of the scanty knowledge then possessed of the anatomy and physiology of the teeth. One has only to turn to Berdmore's writings to see what quaint views were held. The enamel was supposed to thicken, harden and polish until the age of 20 or 25, and after that time it "repaired no longer." The developing permanent teeth, he tells us, "are often furrowed and indented by the pressure of the milk-teeth, stunted in their growth, thrown out of their proper direction and sometimes turned in their sockets." In Berdmore's work there is no clear description of any of the diseases of the teeth, and the only useful observations he makes refer to the harmful effects on the enamel of the use of tooth powders.

These few remarks will convey some idea of the position of dentistry and dental science in England when, in 1771, Hunter published his classical work on the "Natural History of the Human Teeth," which revolutionized the practice of dentistry and provided a sure groundwork for later researches.

That work, great as it is, records, however, only a portion of his labours, and if we appraise him by that work alone we shall fail to appreciate the remarkable extent of the knowledge which he acquired by original research and may unwittingly rob him of the full measure of his deserts.

Hunter was the first to realize (1) that disease cannot be treated rationally until Nature's own methods of curing disease are understood, and (2) that the working of those methods cannot be understood without an intimate knowledge of the organs themselves and their functions in health throughout the whole animal and vegetable kingdoms. The demonstration of these principles was the aim of his life and his specimens were collected and prepared with this great object in view.

His attention was drawn to the teeth at quite an early period of his career, for we know that the statements recorded in the first part of the "Natural History" were based on observations made while he was working with his brother William. It is somewhat remarkable that in later years, when pursuing his scheme of research, Hunter continued to take a keen interest in the practice of dentistry. The explanation is probably to be found in Jesse Foot's book on Hunter. According to that writer, there lived in Gray's Inn Lane one named Spence who combined the practice of dentistry with barbery. Spence seems to have been a practitioner who understood the limitations of his own powers, and, learning of Hunter's reputation, frequently called him to consultations on the teeth. Hunter at this time was a struggling young surgeon and no doubt the fees he received were a welcome addition to a slender income. It is more than probable that the advice Hunter gave helped to increase Spence's reputation as a dentist, and Spence, with the laudable ambition of improving his position, moved to the then fashionable district of Soho Square where he built up a most lucrative practice, Hunter carrying out for him the surgical side of the work, more particularly the operation of transplantation of teeth. Hunter was therefore in constant touch with patients suffering from dental disorders, and with his peculiar mind, he would naturally attempt to unravel the pathology of those conditions which came under his notice. The results of his observations appear in the second part of the work on the teeth.

In estimating our debt to Hunter we must first of all attentively consider the series of specimens designed by him to illustrate "Teeth in General" for these specimens will help us to realize the wideness of his outlook and the comprehensiveness of his generalizations. Some of his views on "Teeth in General" have been preserved in the manuscript catalogue, and although these views are only in the form of rough notes, they nevertheless leave us in no doubt that the specimens were designed to illustrate a well-planned scheme.

The teeth, he says, are always placed "between what may be called the brim or margin of the mouth and the first intestine—viz., in the mouth, œsophagus and stomach." Those in the mouth are divided into two situations: "First, all those forming two rows in each jaw (i.e., one row in the right and another in the left jaw) and opposed by similar rows in the opposite jaws; secondly, where the teeth are placed in other parts, as the tongue. The first situation admits of divisions, as where those rows are single, as in the quadruped and amphibia: in others they are double, triple, &c., rows, as in many fish, where the four rows mentioned are composed of a vast number of rows of teeth."

The various functions of teeth are recognized, for he remarks that teeth "may be classed according to their uses, which I shall at present reckon four—viz., holders or retainers which may be called killers, dividers, crackers and grinders, the last two of which may be thought the same." He draws attention to the fact that in some animals "there is a regular succession of teeth by the falling off or destruction of the teeth and new ones continually growing and gradually coming into use."

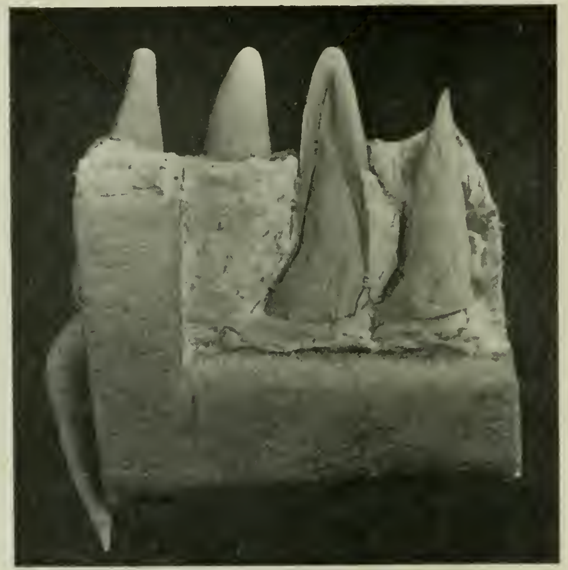

Fig. 1.

Transverse section of the mandible "of the small Bottle-nosed Whale of Mr. Hunter." (R.C.S.[1] Phys. Series, J 166.1.)

This phase of his work is illustrated by more than eighty specimens, which were prepared from thirty-two different types of animals.

Hunter failed to detect the fact that the cement is a separate structure, and, in his writings, he includes both cement and dentine under the term "bone." It is difficult to understand this oversight to differentiate between these two structures, especially when we examine his preparations of elephants' teeth in which the two structures are readily distinguishable. The teeth he divided into two main groups:—

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[1] R.C.S. is used as an abbreviation of Royal College of Surgeons of England.

(1) Those growing to a given size.

(2) Those growing continually.

The first group he subdivided into those composed of bone only and those composed of enamel and bone. Teeth composed of bone only he illustrated by preparations from the porpoise and the whale; and here, at an early stage of our review, we see in Hunter the born demonstrator, for a clearer demonstration of the structure of these teeth than the preparations shown in figs. 1 and 2 is difficult to imagine. The tooth to the right has been uncovered to give a general idea of its shape and its relation to the socket: the adjacent tooth has been split to show the pulp in position; the tooth to the left has been bisected and the pulp turned down in order that the relation of the pulp to the hard part of the tooth may be readily recognized.

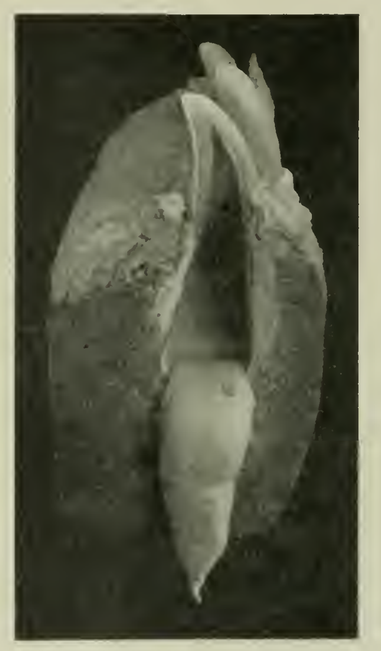

FIG. 2.

Side view of the specimen shown in fig. 1. (R.C.S. Phys. Series, J 166.1.)

Twenty-six specimens prepared from the lion, horse and elephant are employed to demonstrate the structure of teeth composed of enamel and bone. These specimens provide us with another object-lesson in the method of teaching, the importance of which cannot be too strongly emphasized. We are unfortunately constrained to teach dental anatomy in a great measure from dried preparations and there is consequently a tendency to lose sight of the relation between the hard and soft tissues. The necessity of preparing his specimens in such a manner as to show this relation did not escape Hunter. For example, examine the preparation from a young lion (fig. 3). It will be observed that the canine is cut through in order to show the pulp, and incidentally the relation of the erupting premolar to the deciduous tooth is demonstrated.

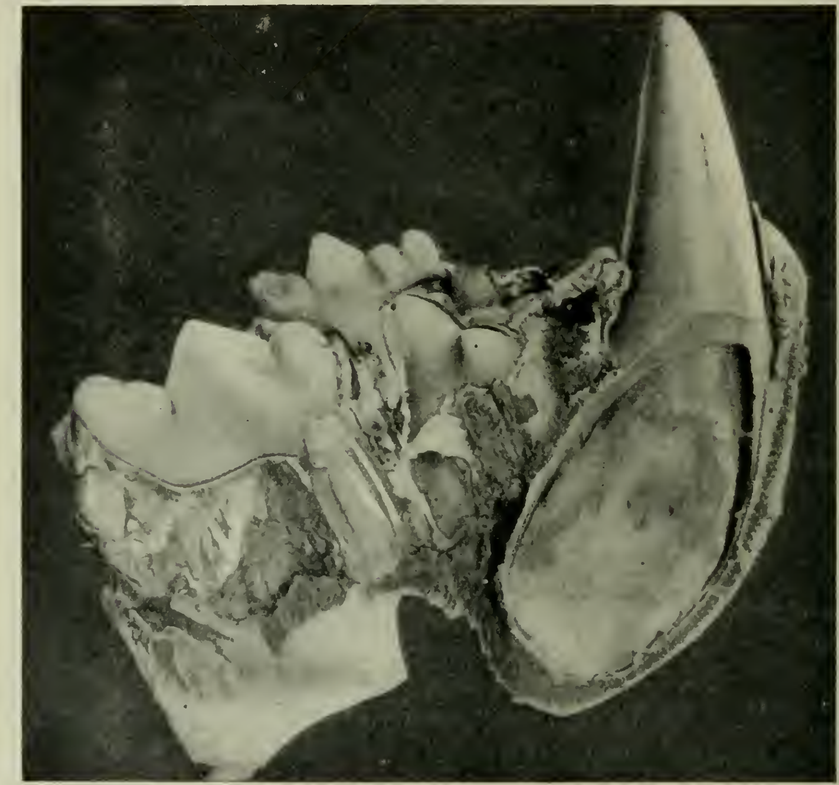

Fig. 3.

Portion of the upper jaw of a young lion. (R.C.S. Phys. Series, J 183.1.)

The complex character of the teeth of the horse is shown in a series of seventeen specimens. I will select two which must have required exceptional skill in their preparation. The first, fig. 4, is intended to show the relation of the bone-forming pulp to the growing tooth.

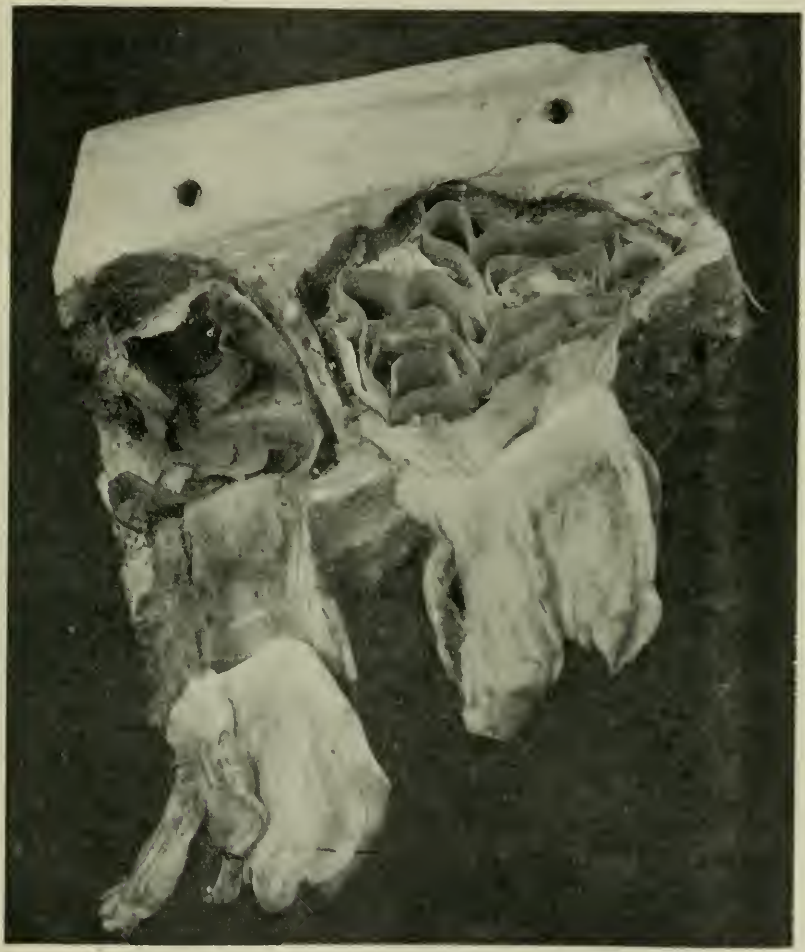

Fig. 4.

Horizontal section from the upper jaw of a horse. (R.C.S. Phys. Series, J 170.7.)

It is a "horizontal section of the upper jaw, with the two first grinders, of a horse. The bases of the sockets are removed to show the hollow growing roots of the teeth, of which the bone-forming pulps have been pulled out and hang down, attached at their bases to the bottom of the socket." The second, fig. 5, shows, in an ingenious way, the relation of the enamel-forming pulp to the bone-forming pulp. It is "a section of the upper jaw of a horse, in which are two grinders of incomplete growth, one of which is much further advanced than the other, having

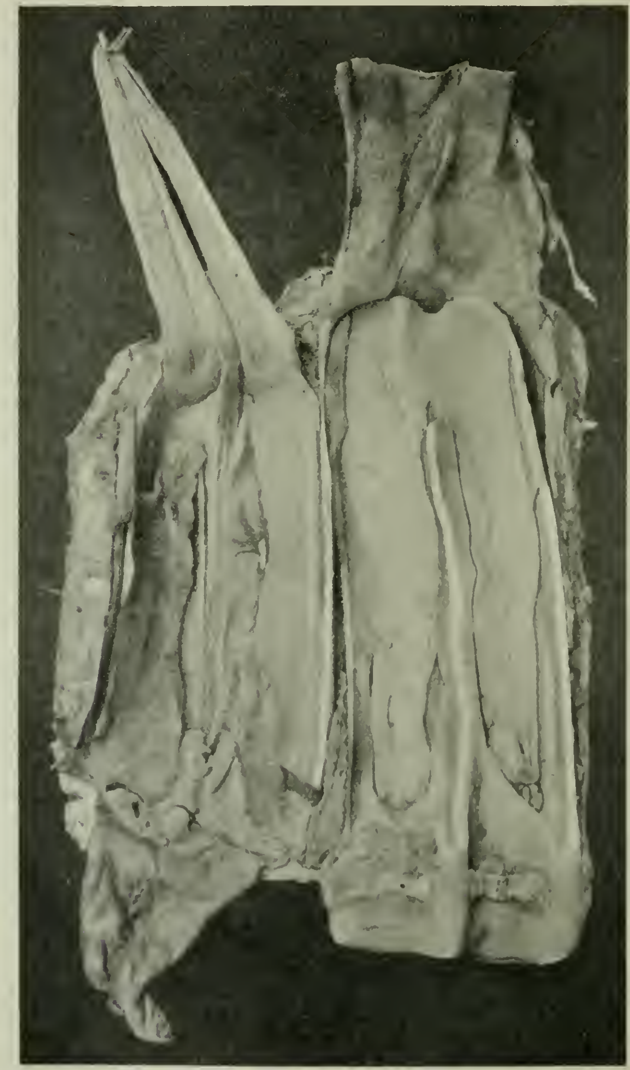

Fig. 5.

Section through the upper jaw of a horse. (R.C.S. Phys. Series, J 170.9.)

pierced the gum. The external part of the jaw has been removed to expose these teeth. From the most advanced tooth, the outer capsule and external lamella of the tooth have been removed to expose the bone-forming pulp, which adheres to the bottom of the socket, and as it passes into the cavity of the tooth, divides as those cavities divide. In the other tooth the forked bone-forming pulp is turned up, and another lamella of the tooth removed to expose the enamel-forming pulp attached at its base to the gum, the two pulps, as it were, interdigitating."

The group of specimens illustrating the teeth of elephants still further exemplifies the thoroughness with which he demonstrated his views. This group includes specimens to show (1) the enamel-forming pulp; (2) the bone-forming pulp; (3) the relation of the two pulps to one another; (4) the structure of the completed tooth.

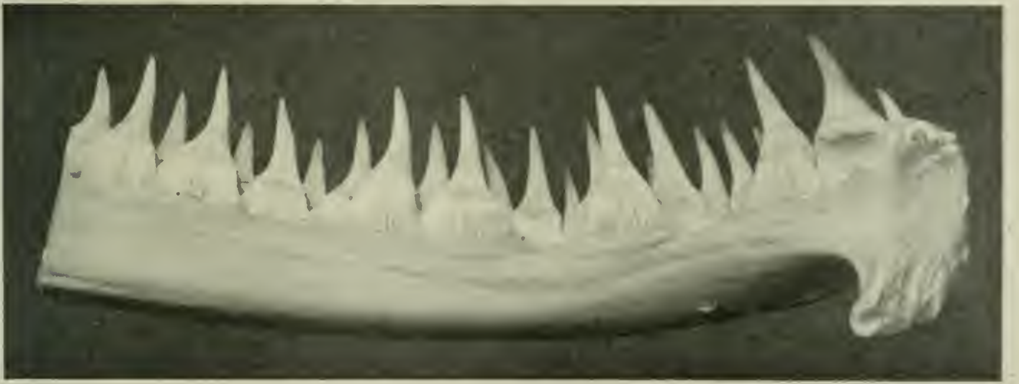

FIG. 6.

Portion of the mandible of an angler. (R.C.S. Phys. Series, J 102.1.)

Teeth of continual growth he divided into two species, first, the *dentes scalprarii* (chisel-edged teeth); and second, the tusks. In order to show the peculiar characteristics of these two types of teeth he chose the beaver, porcupine and boar.

Hunter has left us specimens to demonstrate "the growth of teeth," "the component parts of teeth," and "the process of shedding the teeth." In the last section there is an excellent preparation of the teeth of an angler illustrating the continual shedding of teeth which takes place in many fish and reptiles. This specimen, fig. 6, clearly shows the hinge attachment of the teeth to the bone.

The manuscripts contain a short note on the angler but no mention of the teeth. It is hard to believe that the curious mechanism of the hinge attachment could have escaped the notice of such an acute observer as Hunter, and it is highly probable that his views upon it were contained in the lost manuscripts.

The final group of specimens is designed to illustrate the various positions in which the teeth are situated. Examples are given of teeth situated in the mouth.

(*a*) In a single row in each jaw.

(*b*) In two or more rows in each jaw.

(*c*) In rows on the jaws, and also on other parts, as on the tongue.

(*d*) Where the teeth are scattered.

(*e*) Where they are disposed like a pavement.

(*f*) "This is a very singular class of teeth, where there is no jaw, the motion being in the teeth only."—Hunterian MS. catalogue. This is shown by a vertical section of an *echinus*.

The fauces of a carp and a large and small "annelide" are used to demonstrate teeth in the pharynx and œsophagus; a "mollusk," "Cape grasshopper," "river craw-fish," and a lobster, to show teeth in the stomach.

Close on one hundred and thirty years have passed since Hunter died and the facts which he demonstrated in these beautiful specimens have gradually been accepted and incorporated in the body of science. His clear intelligence evolved order out of chaos and his work laid the foundations of the science of odontology.

The specimens illustrating the "Natural History of the Human Teeth" have found a home in two museums, that of the Royal College of Surgeons of England and that of the University of Glasgow. It may be questioned whether the specimens in the Glasgow collection are the work of John Hunter. On this point I do not myself think that there is any room for doubt. We know that the subject of teeth attracted his attention early in his career when he was working in collaboration with his elder brother William in London. But the strongest evidence of their authorship is to be found in the fact that the plan of the specimens and the manner of their preparation indicate the mind and hand of the author of the specimens housed in the Royal College of Surgeons of England and the latter are indisputably John Hunter's. There is no evidence that William Hunter specially interested himself in the teeth or prepared specimens.

Hunter's specimens number ninety-three in all, of which fifty-six are in the Glasgow Museum and thirty-seven in that of the Royal College of Surgeons of England. Some subjects are illustrated in one of the collections only. In the Glasgow Museum there are series showing the soft tissues of the mouth; the periodontal membrane and variations of the teeth; but in the College of Surgeons' Museum these subjects are not represented. On the other hand the series in the latter museum showing the growth of the teeth at different ages and secondary dentine are not duplicated at Glasgow.

The existence of two separate and distinct collections may seem strange but is not, I think, difficult to explain. Hunter's earliest observations on the teeth were made, as previously mentioned, during his association with his elder brother William and any specimens prepared by John Hunter during that period would become the property of the elder brother who bequeathed his museum to the University of Glasgow. Subsequently John Hunter served in the Seven Years War and on his return he set himself the task of writing a work on the teeth. For the purpose of illustrating this work he had to prepare a fresh set of specimens, and it is these specimens which form part of the collection entrusted to the care of the Royal College of Surgeons.

In viewing these collections, we are at once attracted by a series of preparations showing the growth of the jaw from fœtal life until adult age, and then, emphasizing the lesson which the series teaches, there is a final preparation of four mandibles of different ages (fig. 7) which Hunter describes in the following words:—

> "Four lower jaws of different periods of life, from the age when the five shedding teeth are completely formed, to that of a complete set. This figure shows four things: (1) The lengthening of the jaw backwards which is seen by the oblique line made by the four condyles. (2) The gradual rise of the two processes above the line of the teeth. (3) The gradual increase of the teeth in proportion as the jaw lengthens. (4) The part formed always keeping of the same size."

Here, then, for the first time we are given an account of the growth of the mandible, and the views set forth are almost identical with those we hold to-day. A group of eleven preparations unfolds the progressive growth and loss of the deciduous teeth and the growth of the permanent teeth. These preparations display the teeth separately, as shown in fig. 8. Other specimens show the growth of single- and multiple-rooted teeth, and demonstrate the absence of any fundamental difference in the growth of the deciduous and permanent teeth.

In Hunter's day the manner in which the teeth are formed was little understood by the ordinary practitioner. Berdmore, who tells us that a perfect knowledge of the teeth is the basis of all rational practice, gives us the following account of the formation of the teeth:—

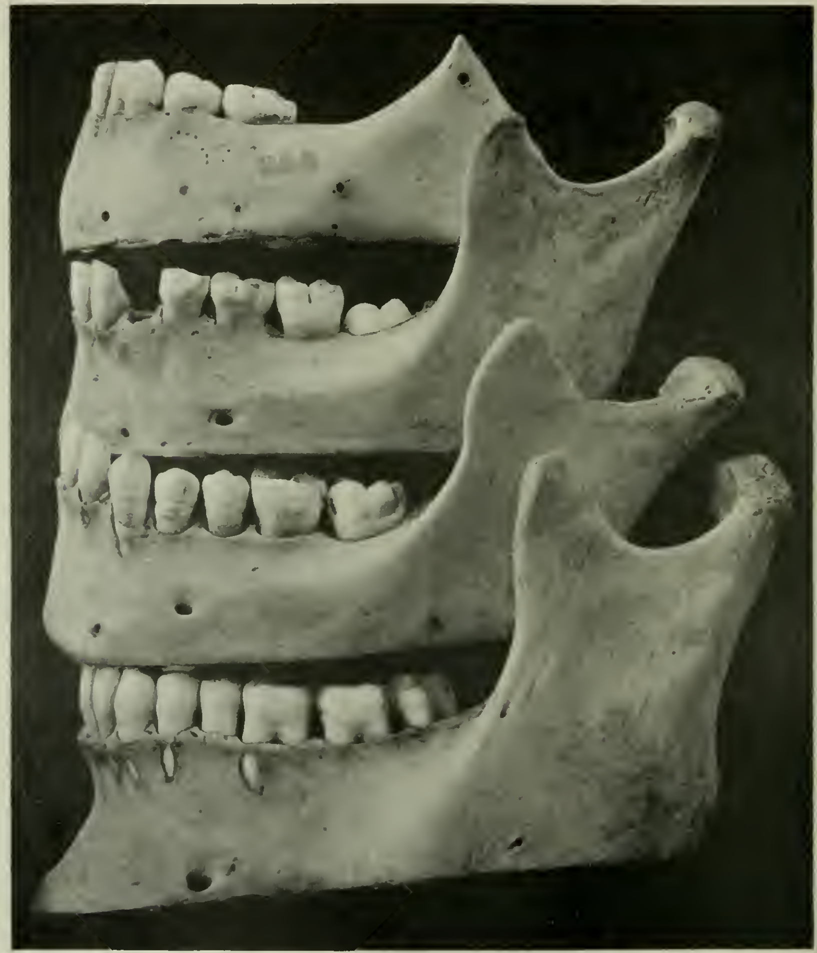

FIG. 7.

Mandibles of different ages to show the growth of the jaw. (R.C.S. Odonto. Series, H 64.)

"In infants, newly born, we observe, in each socket, a collection of soft white glairy matter contained in a small membranous sack, pierced on that side which corresponds with the bottom of the socket by a nervous and vascular chord, whose vessels presently divide, and are branched all over the membrane and contained substances."

This was written in 1770. Compare with this Hunter's masterly description published one year later:—

"The body of the tooth is formed first, afterwards the enamel and fangs are added to it. All the teeth are produced from a kind of pulpy substance, which is pretty firm in its texture, transparent, excepting at the surface, where it adheres to the jaw, and has at first the shape of the bodies of the teeth which are to be formed from it. These pulpy substances are very vascular. They adhere only at one part to the jaw—viz., at the bottom of the cavity which is to form the socket, and at that place their vessels enter; so that they are prominent, and somewhat loose in the bony cavity which lodges them.

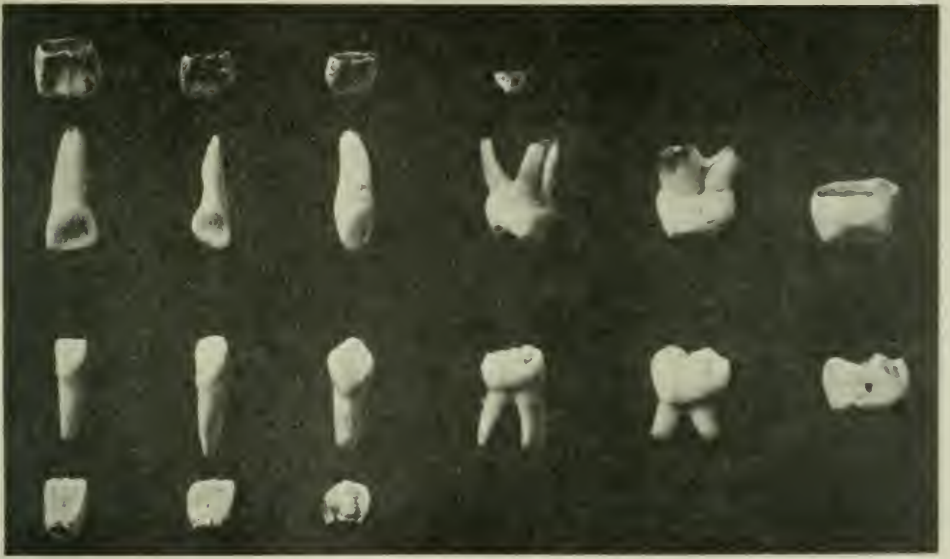

FIG. 8.

The teeth from a child about five or six years of age. (R.C.S. Odonto. Series. H 68.) This figure forms the subject of fig. 1, Plate X, "Natural History of the Teeth."

"They grow nearly as large as the body of the tooth before the ossification begins, and increase a little for some time after the ossification is begun. They are surrounded by a membrane, which is not connected with them, excepting at their root or surface of adhesion. This membrane adheres by its outer surface all around the bony cavity in the jaw, and also to the gum where it covers the alveoli.

"When the pulp is very young, as in the fœtus of six or seven months, this membrane itself is pretty thick and gelatinous. We can examine it best in a new-born child, and we find it made up of two lamellæ, an external and internal; the external is soft and spongy, without any vessels, the other is much firmer, and extremely vascular, its vessels coming from those that are going to the pulp of the tooth; it makes a kind of capsula for the pulp and body of the tooth. While the tooth is within the gum, there is always a mucilaginous fluid, like the sinovia in the joints, between this membrane and the pulp of the tooth.

"When the tooth cuts the gum, this membrane likewise is perforated: after which it begins to waste, and is entirely gone by the time the tooth is fully formed, for the lower part of the membrane continues to adhere to the neck of the tooth, which has now risen as high as the edge of the gum."

You will search in vain through the text-books of the present day for a more lucid and picturesque account.

Hunter was the first to describe the fibrous character of the enamel and to point out in what manner the enamel is distributed over the dentine. By ingenious experiments he demonstrated the difference in composition between the enamel and the dentine. He says:—

> "Let one half of a tooth be filed away, from one end to the other, then burn it gently in the fire. After this is done wash the filed surface with an acid, or scrape it with a knife. By this method you will clean the edge of the enamel, which will remain white, and the bony part will be found black."

Both collections contain teeth which have been treated in this way. The specimens in the Royal College of Surgeons' Museum are shown in fig. 9.

In the Glasgow Museum there are teeth which have been treated with nitrate of silver in order to show the difference in composition of the two tissues.

To Hunter we are indebted for the first accurate description of the individual teeth, and of the method by which they are attached to the bone. Here again we can only realize the advance he made by a comparison with his contemporary Berdmore, who describes the incisors as follows:—

> "The *Roots* of the incisors are long, pointed and single; the *Bodies* are wedge-like, convex on the outer side, a little concave within, flattened on the sides and so disposed that their sharp edges lie all in a line, making one uniform edge."

Listen now to Hunter's description of the lower incisors:—

> "The bodies of the *Incisores* are broad, having two flat surfaces, one anterior, the other posterior. These surfaces meet in a sharp cutting edge.

The anterior surface is convex in every direction, and placed almost perpendicularly; and the posterior is concave and sloping, so that the cutting edge is almost directly over the anterior surface. These surfaces are broadest and the Tooth is thinnest at the cutting edge, or end of the Tooth, and thence they become gradually narrower and the Tooth thicker towards the neck, where the surfaces are continued to the narrowest side, or edge of the fang. The body of an *Incisor*, in a side-view, grows gradually thicker, or broader, from the edge or end of the Tooth to its neck : and these coincide with the flat or broad side of the fang; so that when we look on the fore part, or on the back part of an *Incisor*, we observe it grows constantly narrower, from its cutting edge to the extremity of its fang. But in a side-view it is thickest and broadest at its neck, and thence becomes gradually more narrow, both to its cutting edge and to the point of its fang.

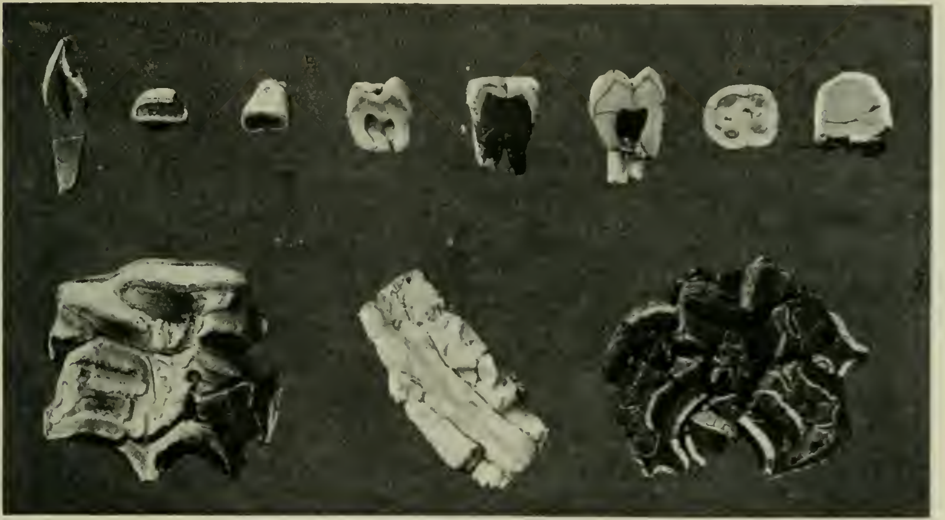

FIG. 9.

Specimens treated by heat to demonstrate the difference in composition between enamel and dentine. (R.C.S. Odonto. Series, H 85.)

"The enamel is continued farther down, and is thicker on the anterior and back part of the *Incisores* than on their sides, and is even a little thicker on the fore part than upon the back part of the Tooth. If we view them laterally, either when intire, or when cut down through the middle, but especially in the latter case, it would seem as if the fang was driven like a wedge into, and had split the body of enamel of the Tooth. They stand almost perpendicularly, their bodies being turned a little forwards. Their fangs are much shorter than those of the *Cuspidati*, but pretty much of the same length with all the other Teeth of this Jaw."

There are specimens to show the shapes of the pulp chambers (fig. 10) and drawings to illustrate the arterial supply of the pulp and alveolar process.

Hunter was the first to draw attention to the formation of secondary dentine. He says:—

> "A tooth very often wears down so low that its cavity would be exposed, if no other alteration were produced in it. To prevent this, Nature has taken care that the bottom part of the cavity should be filled up by new matter, in proportion as the surface of the teeth is worn down. This new matter may be easily known from the old, for when a tooth has been worn down almost to the neck, a spot may always be seen in the middle, which is more transparent, and at the same time of a darker colour (occasioned, in some measure, by the dark cavity under it), and is generally softer than the other."

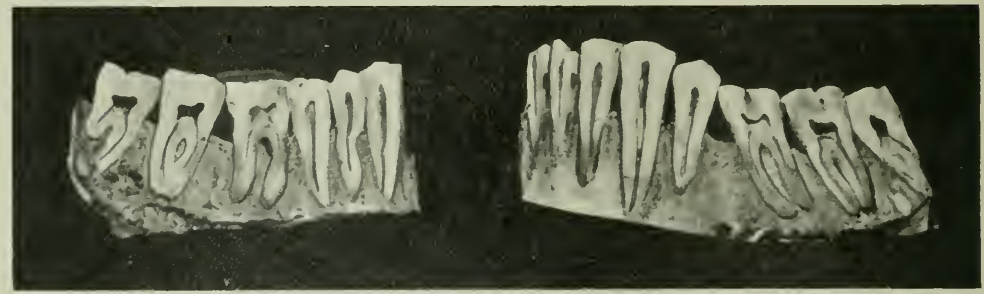

FIG. 10.

Sections through the mandibular teeth to demonstrate the shape of the pulp cavities (Museum of the Glasgow University, 29/29 and 29/30.)

The second part of the "Natural History of the Teeth," containing Hunter's views on the pathology and treatment of diseases of the teeth, was published in 1776. In criticizing this part of his work, we must remember the years that have past since it was written and we must view it in the light of the limited knowledge which existed in his day. The keynote of the work is this—treatment must be based on a sound knowledge of anatomy, physiology and pathology—and this principle we must take thoroughly to heart if we wish to be regarded as a scientific profession.

Four years ago I was afforded an opportunity of examining a collection of skulls of Londoners of the sixteenth to the eighteenth century and since then I have re-read Hunter's work with added interest because I have been better able to visualize the type and extent of dental disease in his day. It has always seemed odd to me that he should have devoted a section to "Deep-seated Abscesses in the Jaws"; but this is readily understood when one's attention is drawn to the large number of very severe abscesses that occurred in his day. He realized that these abscesses commonly arose from a disease in the teeth, "more especially in the cuspidati, these teeth passing further into the jaw than others. Their depth in the jaw being beyond the attachment of the lip to the gum, if an abscess forms at their points, it more readily makes its way through the common integument of the face, than between the gum and lip, which disfigures the face, and when in the lower jaw looks like the evil."

The clinical condition of the gums now called "Vincent's angina," which has been very prevalent during the recent war, was recognized by Hunter. It is true that he did not know its pathology as we know it to-day, but he gives us an excellent clinical picture of the condition in the following words:—

> "When the gums first begin to have a tenderness, we may observe it first on their edges; the common smooth skin of the gum is not confined to its very edge, but becomes at the edge a little rough like a border and somewhat thickened. The part of the gum between two teeth swells, and often pushes out like luxuriant flesh, which is frequently very tender. The inflammation is often carried so far as to make the gums ulcerate, so that the gums in many cases have a common ulcer upon them, by which process a part of the tooth is denuded. This is often in one part only, often only on one jaw, while in some cases it is on the whole gums on both jaws. In this case it often happens that the alveolar process disappears, after the manner above described, by taking part in the inflammation, either from the same cause, or from sympathy. In such cases there is always a very considerable discharge of matter from the inside of the gums and alveolar process, which always takes the course of the tooth for its exit. In many of these cases we find that while the gums are ulcerating in one part, they are swelling and become spongy in another, and hanging loose upon the teeth; and this often takes place where there is no ulceration in any part."

It would be difficult to find in modern literature a more lucid account of the progressive stages in inflammation of the gums, from the marginal gingivitis of the mouth-breathing child on the one hand to the acute ulceration of the gums seen in cases of Vincent's angina on the other.

Irregularities in the position of the teeth he attributes to "want of room in the jaw," and his treatment by extraction is based on that idea. The choice of teeth must, he states, be left to the judgment of the operator, but certain guiding principles are suggested:—

(1) "If there is any one Tooth very much out of the row, and all the others regular, that Tooth may be removed and the two neighbouring ones brought closer together."

(2) "If there are two or more Teeth of the same side very irregular (as, for instance, the second Incisor and Cuspidatus), and it appears to be of no consequence with respect to regularity, which of them is removed, I should recommend the extraction of the furthest back of the two, viz., the Cuspidatus, because if there should be any space, not filled up, when the tooth is brought into the row, it will not be readily seen."

(3) "If the above mentioned two Teeth are not in the circle, but still not far out of it, and yet there is not room for both, in such a case I would recommend the extraction of the first Bicuspis, although it should be perfectly in the row because the two others will then be easily brought into the circle, and, if there is any space left, it will be so far back as not to be at all observable."

For the treatment of superior protrusion he suggests the removal of a premolar on each side, by which means, he says, "the fore part of the circle will fall back."

Most interesting, also, is his recognition of the value of early extraction of unsaveable first permanent molars. The paragraph in which he refers to this is as follows: "It may not be improper, in this place, to take notice of a case which frequently occurs. It is a decay of the first adult grinder at an early age—viz., before the temporary grinders are shed, and before the second grinder of the adult has made its appearance through the gum. In this case, I would recommend removing the diseased tooth immediately, although it may occasion no kind of trouble; for, if it be drawn before the temporary grinders are shed, and before the second adult grinder has cut the gum, it will in a short time not be missed; because the bicuspids of that side will fall a little back, and the second and third grinders will come a little forward, by which means the space will be filled up, and these teeth will be well supported. Besides, the removal of this tooth will make room for the fore teeth, which are often very much wanted, especially in the upper jaw."

The first part of the "Natural History of the Teeth" is profusely illustrated, but the second part is without a single illustration; there are, however, twenty-six preparations in the two museums illustrating the pathology of the teeth. Attention is drawn to two of these preparations because I think they are unique. The first, fig. 11, is a maxillary canine with a curious spine-like projection from the labial aspect; the second, fig. 12, is the skull of a negress with a canine erupting in the nasal fossa.

Time will not permit me to refer at any great length to Hunter's contributions to comparative dental anatomy and pathology. In the osteological series there are over 500 skulls of animals belonging to nearly 150 different varieties.

There are a few notes on the dentitions of these in his posthumous papers. Most of the notes are brief—for instance, regarding the kinkajou he remarks, it "has three grinders like a monkey's; two sharp-pointed teeth which may be called little canines; all these are close together in the upper jaw; six incisors, which are close together in the lower jaw." In a few instances the descriptions are given in fuller detail, as in his account of the teeth of a *Phascogale*, which, Owen remarked, "could not fail to impress the reader with Hunter's faculties of accurate and minute description."

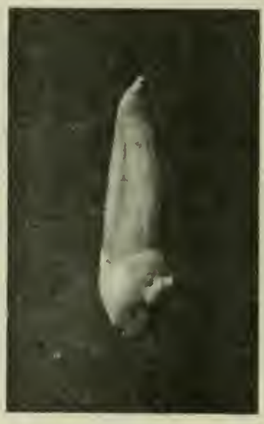

Fig. 11.

Maxillary canine with a curious spine-like projection from the labial surface. (R.C.S. Odonto. Series, H 105.)

Although Hunter's views on many of the specimens were never recorded, or, if recorded, were lost, nevertheless the specimens remain and it was mainly to those that Owen must have turned when he was preparing his classical work on "Odontography."

There are a few specimens of comparative dental pathology, but they are not referred to in any of his writings. There are examples of bullets embedded in the tusks of elephants, of secondary dentine, of overgrowth of teeth, and there is an extraordinary specimen of a malformed molar of an elephant.

Such, then, is a brief and, I am afraid, imperfect picture of Hunter's work on teeth, but I trust it is sufficient to show how vast is the debt that dentistry owes to this great man. It is true that his writings are not free from errors, but some of these are, I feel sure, to be attributed to want of care in the revision of the proof sheets. At times he is most lucid, at other times obscure, but he is always interesting. He is constantly suggesting explanations, and these "Hints of Hunter," which were full of meaning, pointed the direction in which investigation should be pursued.

Of Hunter it can truly be said that he was a student in the great university of Nature, he was a most accurate observer, he understood the value of experimental investigation and he possessed the power of making wide generalizations from the facts which came under his observation. We shall never know how far he had linked these facts together into a theory of life, but the specimens remain and they speak for themselves.

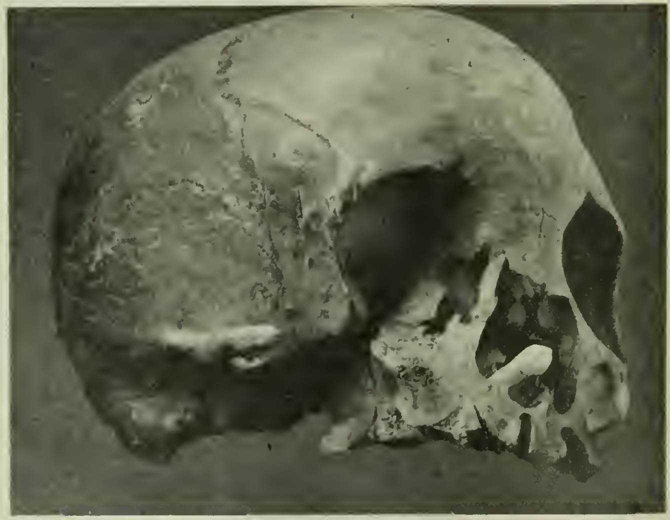

FIG. 12.

Skull of a negress with a canine erupting in the nasal fossa.
(R.C.S. Odonto. Series, H 88.)[1]

We must needs ever be grateful to him for placing the science of dentistry on a solid foundation, upon which the super-structure is gradually being built. The building is far from complete, but I am optimistic enough to believe that completed it will be, and that the day will come when our knowledge will enable us to prevent dental disease. The more closely we follow the methods of John Hunter the more quickly will that great ideal be realized.

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[1] For the loan of the blocks we are indebted to Messrs. Claudius Ash, Sons and Co., Ltd.

Section of Odontology.

President—Mr. J. F. COLYER, F.R.C.S., L.D.S.

Ankylosis of the Mandible and its Operative Treatment.[1]

By GILBERT CHUBB, F.R.C.S.Eng., M.B., D.Sc.

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THE ankylosed mandible is a condition which very directly affects the dental surgeon. Apart from the occasional dental cause of the fixation, and the dental complications its existence may produce, the non-operative treatment is a matter almost entirely for the dental surgeon. In those cases which fail to yield to non-operative treatment the success of an operation will very largely depend upon the post-operative treatment by the dental surgeon. In the cases now described their satisfactory condition is very largely due to the care and skill of my friend and colleague, Mr. Mendelson.

Ankylosis of the mandible may be due to infection or trauma. Opinion differs as to which of these causes is the more common, but it is more than likely that, even in the traumatic cases, infection plays an important part in the subsequent fixation. The numerous operations which, from time to time, have been described for the relief of this condition fall broadly into two groups :—

(1) The resection of the condylar neck, with or without the condyle.

(2) Resection of a wedge from the horizontal ramus, anterior to the cause of fixation.

Operations in the first group fail to give relief when the cause of fixation is situated wholly or in part anterior to the condyle. Those in the second group, in bilateral cases, leave the jaw so weak as to be practically useless for mastication. In the recent (3rd) edition of Brown's "Surgery of Oral Disease and Malformations" the following statement occurs:—

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[1] At a meeting of the Section, held October 27, 1919.

> "When the ankylosis is bilateral, and of such a character as to make it impossible to gain relief by operation upon the condyles, resection according to the Esmarch operation upon both sides leaves little or no muscular strength for the use of the jaw in mastication. If such patients are well nourished, and getting on comfortably without operation, it is the author's belief that they should be left undisturbed, for a bilateral Esmarch operation ought not to be performed except in rare cases or urgent necessity."

In each of the five cases now described the fixation was wholly or in part anterior to the condyle, and could not, therefore, be relieved by operation confined to the region of the temporo-mandibular joint. In four of the cases the original cause of the condition was trauma; gunshot wound in two and a kick or blow in the other two, all in the malar region. In the fifth case, which was bilateral, fixation followed acute arthritis of the temporo-mandibular joint, accompanying measles in infancy. In every case, however, there had been sepsis or infection in the zygomatic fossa. And in every case—and this is the important point—the coronoid was tied down to the surrounding structures, and especially to the pterygoid aspect of the maxilla, for an extent which, in the traumatic cases, spread far beyond the actual bony lesion. In two of the traumatic cases, and in the purely infective case, no definite lesion of the coronoid could be found, and in these cases the concentration of the adhesions into the region between the lower and anterior border of the coronoid process, and the lower part of the pterygoid aspect of the maxilla was very striking. In one of the remaining cases (Case III) in which the condition was of three years' standing, this anteriorly-placed adhesion had been replaced by a bony union of ivory hardness, although no definite fracture of this region of the coronoid process could be demonstrated.

The series strongly suggests that this element in the fixation of the mandible was the result, not of the trauma, but of the accompanying inflammatory processes in the pterygoid fossa. The close relation of the lower border of the coronoid process to the mucous membrane limiting the oral cavity may explain the tendency for adhesions to form in this region.

The condition was, in each case, relieved by the complete resection of the coronoid. In the purely infective case this was removed on both sides, together with the condylar neck region of the ascending ramus on each side. The operation was the same for each, and was devised to allow of the complete resection of the coronoid process down to its base, without unduly interfering with the muscles of mastication, and without risk of injury to the branches of the facial nerve. The operation was found to give an admirable approach to the pterygoid fossa, and to permit of the resection of the condylar region also, where this was necessary. The scar is almost entirely in the hair region, and therefore practically invisible. In none of the cases was there any involvement of the facial nerve. And in every case there was free and painless mastication of surprising efficiency within a few weeks of the operation.

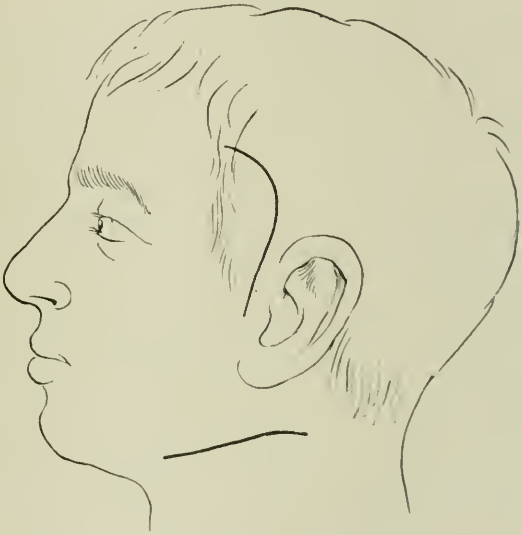

FIG. 1.

The incision (fig. 1) differs considerably from those hitherto described for operations in this region, and has the three following advantages: It avoids all branches of the facial nerve; it is confined almost entirely to the hair region; and it gives a free and wide approach to the pterygoid fossa. It starts in the pre-auricular fold of the lowest level of the external meatus, and passes vertically upwards to about the level of the top of the pinna, from which point it curves gently forwards a little below the superior temporal crest, terminating well within the hair area of the temporal region. The incision is carried directly down to bone. The skin, temporal fascia, and temporal muscle are separately defined and turned forwards (fig. 2). The deeper of the two layers into which the temporal fascia splits as it approaches the zygoma is divided from its deep aspect, and the more superficial layer—still working from its deep surface—is separated from the outer surface of the zygoma with the raspatory. The zygoma is resected piecemeal with the gouge forceps from immediately in front of the eminentia articularis posteriorly to as far forward as to include a portion of the malar bone anteriorly. For this resection and for the remainder of the operation the surgeon stands at the head of the patient and approaches the zygomatic fossa from above, that is, by way of the temporal fossa.

The tip of the coronoid can now be readily felt. Its upper and posterior portion is defined by severing the more posterior fibres of the temporal muscle close to the bone, upward traction of the muscle being performed at the same time. The coronoid is now cleared of its muscular attachments with the respiratory and is then resected piecemeal with gouge forceps (fig. 3), guided by the finger, until the anterior border of the ascending ramus passes in a smooth curve down to the commencement of the alveolar border of the horizontal ramus (*see* also fig. 9, p. 30).

In this resection the surgeon is working between the deeper fibres of insertion of the temporal muscle and the belly of the external pterygoid on the one hand, and the under surface of the masseter muscle as this passes to its insertion into the outer face of the coronoid and angle on the other. Anteriorly and inferiorly ordinary care is sufficient to avoid injury to the mucous membrane covering the lowest portion of the coronoid, and separating the latter from the oral cavity.

In actual practice, the difficulty experienced in the removal of the coronoid varies enormously according to the nature and extent of the injury, and the degree of ankylosis to the surrounding bones.

In cases in which only the coronoid is to be removed, the operation is completed by stitching back the temporal fascia and muscle into place and closing the skin incision, a small drainage tube being left in the wound for forty-eight hours. In the fifth case it was necessary to resect the neck of the condyle also. This was done, partly through the temporal incision already described, the neck being attacked on its anterior aspect with curved gouge forceps. On account, however, of the large and very hard mass of bone occupying the site of the joint in this case, and in order to minimize the risk of injury to the internal maxillary artery, the resection was completed from behind, the lower border of the angle being defined by means of a horizontal incision

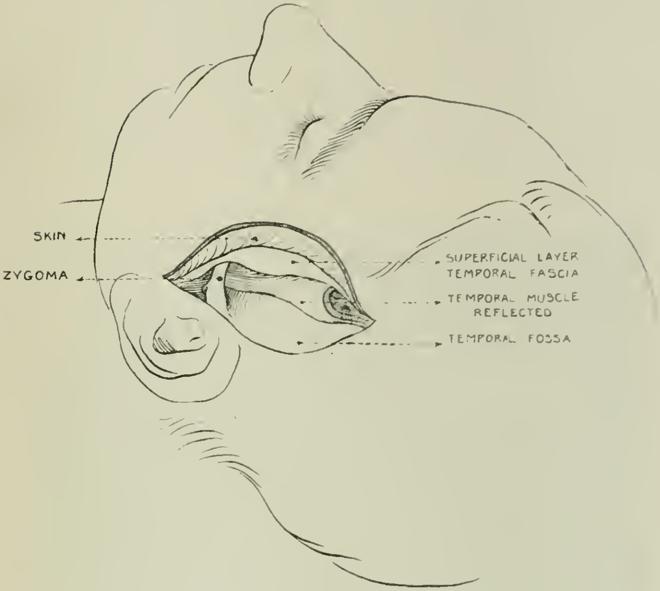
SKIN
ZYGOMA
SUPERFICIAL LAYER TEMPORAL FASCIA
TEMPORAL MUSCLE REFLECTED
TEMPORAL FOSSA

FIG. 2.

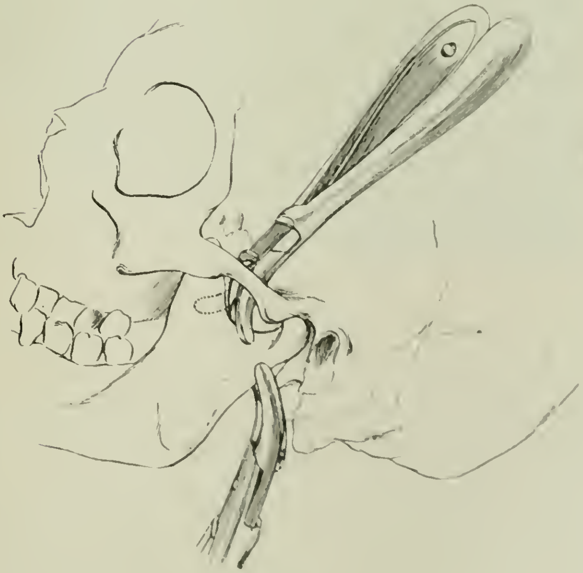

FIG. 3.

below this region (fig. 1), and the condylar neck reached, by a subperiosteal resection of the posterior border of the ascending ramus (fig. 3). This second incision was also useful for the "Murphyrization" of the future pseudarthrosis by means of a flap of the posterior portion of the temporal fascia and muscle, which was turned down, brought through the gap made by the resection of the condylar neck and secured in the deeper layers of the lower wound.

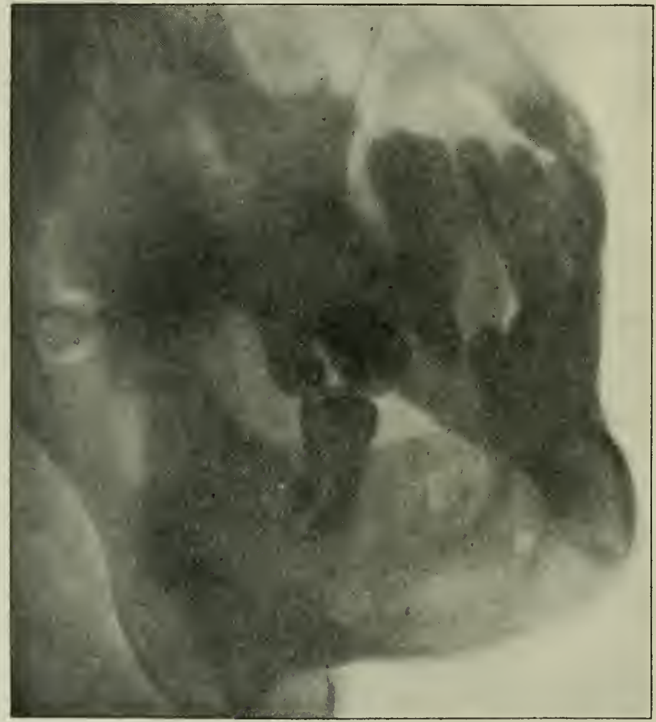

FIG. 4 (Case I).

Case I.—Pensioner W. was wounded in May, 1918, and a piece of shell removed from just below the zygoma at a Casualty Clearing Station, in France. At this time he could open the mouth wide enough to admit "the extreme tip of the little finger." From September to November of the same year, he was using spring splints but with very slight improvement and much pain. Later he used a screw gag daily, and the jaws were on three occasions wrenched open under gas. A note by Mr. Northcroft, of the First London General Hospital, states that in September the patient was only able to open his mouth 2½ mm. He was admitted to Queen's Hospital, Sidcup, in April, 1919, and fitted with a screw gag, but its use was painful, and when Mr. Fry brought him to me in the latter part of June, his gape was ¼ in., and he could only open and close his mouth through this interval with difficulty. There was a scar on the right anterior zygomatic region and the X-ray showed a fracture of the upper part of the coronoid (fig. 4). Operation, June, 1919: The tip of the zygoma was found to be ankylosed to the infratemporal crest and had to be chiselled away. Below this the coronoid expanded into a mass of sclerosed bone and fibrous tissue, quite ½ in. in thickness and extending sufficiently deeply into the pterygoid fossa to make the earlier stages of its removal somewhat difficult. After the complete removal of the coronoid the mouth could be readily gagged open. On the day following the operation the patient had a voluntary gape of 2 cm., and the mouth could be opened and closed through this interval without pain. He was fitted with a spring gag and in the following September had a gape of 2½ cm., eats "practically anything," and masticates his food without pain. He was by that time using the spring gag about twice a week (fig. 5).

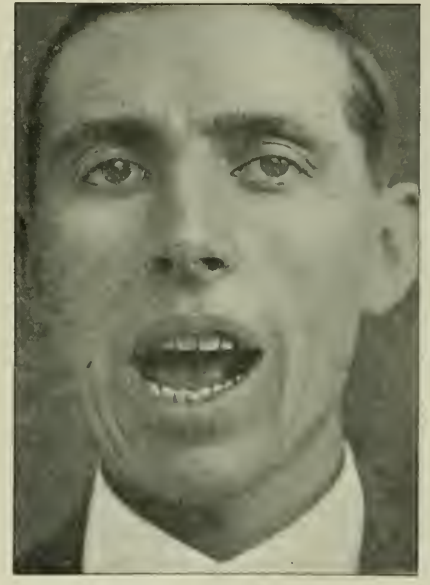

Fig. 5 (Case I).

Case II.—Pensioner G., was wounded in September, 1917, in the right malar region, and a piece of shrapnel was removed from inside the mouth in France. For the first four months after being wounded the patient received massage and gagging, but from then onwards he received no treatment until his admission to Queen's Hospital, in June, 1919. The gape then would with difficulty admit the tip of the little finger (fig. 6). At the operation, July, 1919, there was found a fracture of the anterior portion of the zygoma and a large boss of sclerosed bone projected from the anterior border of the coronoid and could be distinctly felt with the finger to impinge on the pterygoid aspect of the maxilla on an attempt being made to open the mouth (fig. 7). After the coronoid (figs. 8 and 9, pp. 29, 30) had been resected the mouth could be readily opened to the full extent of the gag. On the day following the operation the patient had a voluntary gape of 2 cm. In the following September he had a gape of 2½ cm. and his mastication was good and painless (fig. 10, p. 30). He still uses the spring gag for a short time each morning.

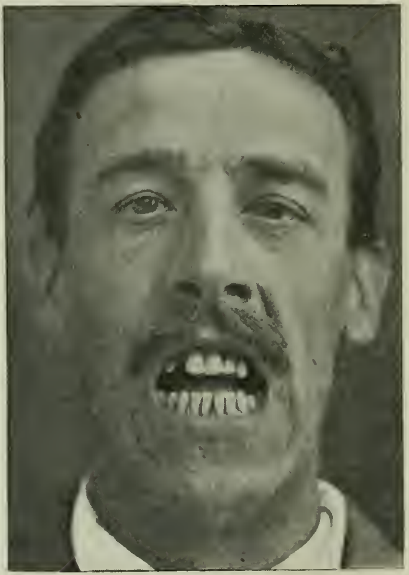

FIG. 6 (Case II).

Case III.—Private M., was kicked on the cheek by a mule in March, 1915, and from that time had been unable to open his mouth. He had had no treatment. On admission to Queen's Hospital in June, 1919, the mouth was found to be firmly closed (fig. 11, p. 31), and although a slight tilting of the left side could be obtained by the employment of considerable force, it was not possible to obtain any separation of the teeth on the right side. There was a scar and depressed area over the right zygomatic region and the X-ray clearly showed ankylosis of the coronoid to the overlying zygoma (figs. 12 and 13, pp. 31, 32). At the operation, June, 1919, the fractured zygoma was found to be firmly ankylosed to the coronoid, and both to the squamous temporal and sphenoid in the region

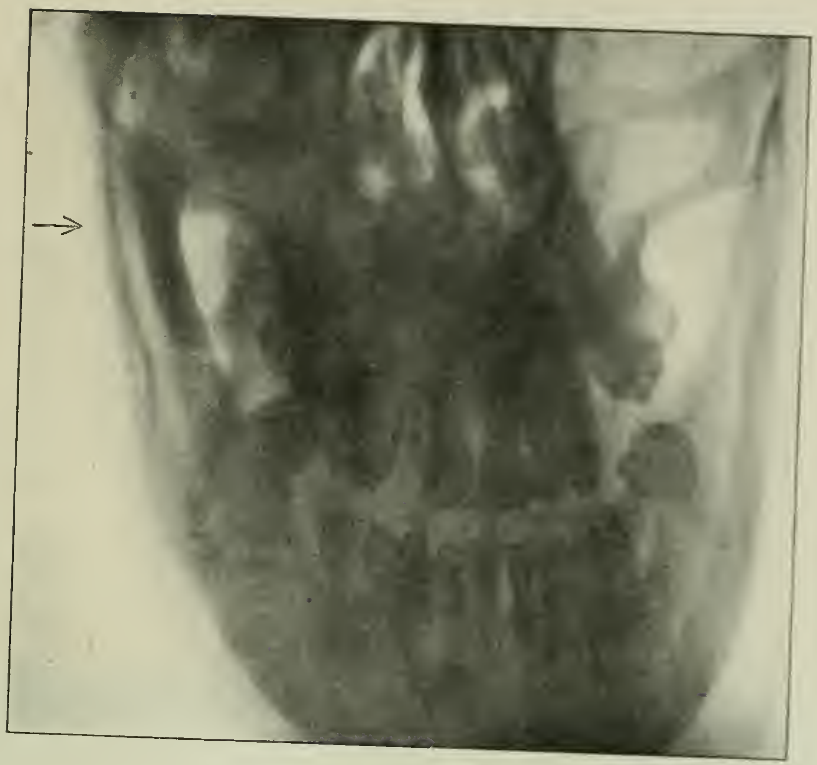

FIG. 7 (Case II).

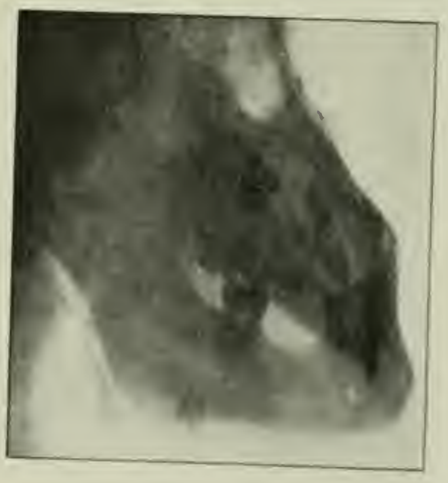

FIG. 8 (Case II).

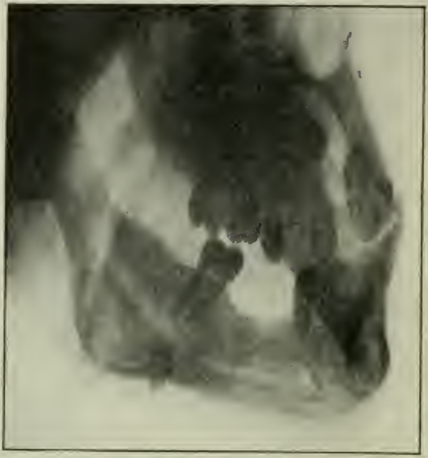

Fig. 9 (Case II).

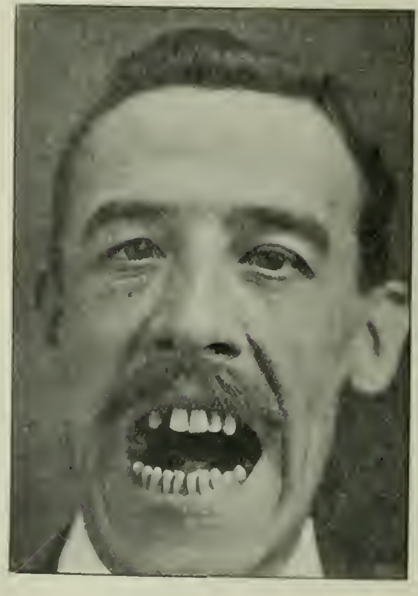

Fig. 10 (Case II).

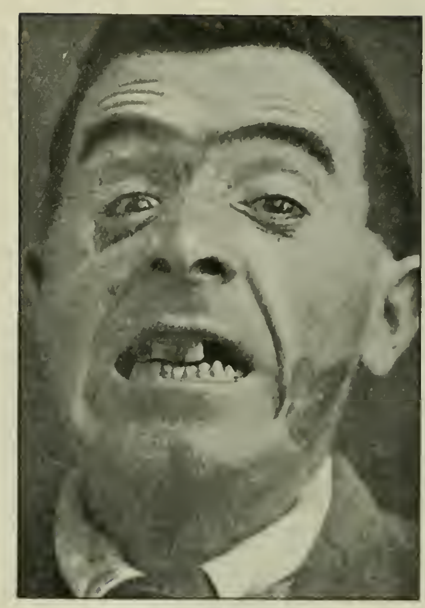

FIG. 11 (Case III).

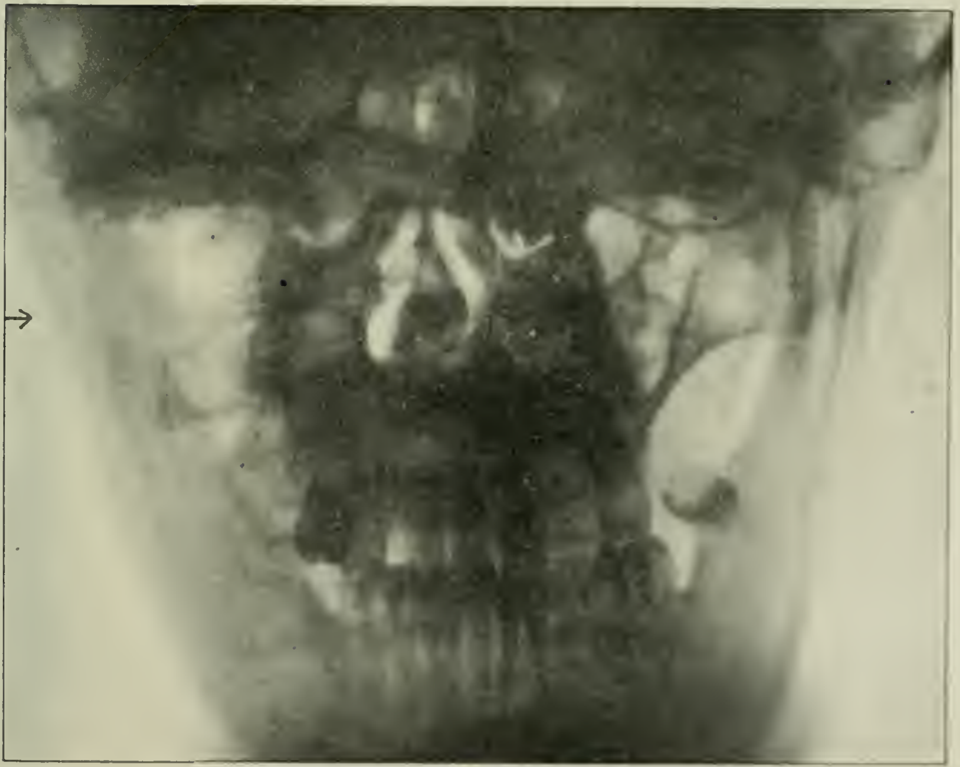

FIG. 12 (Case III).

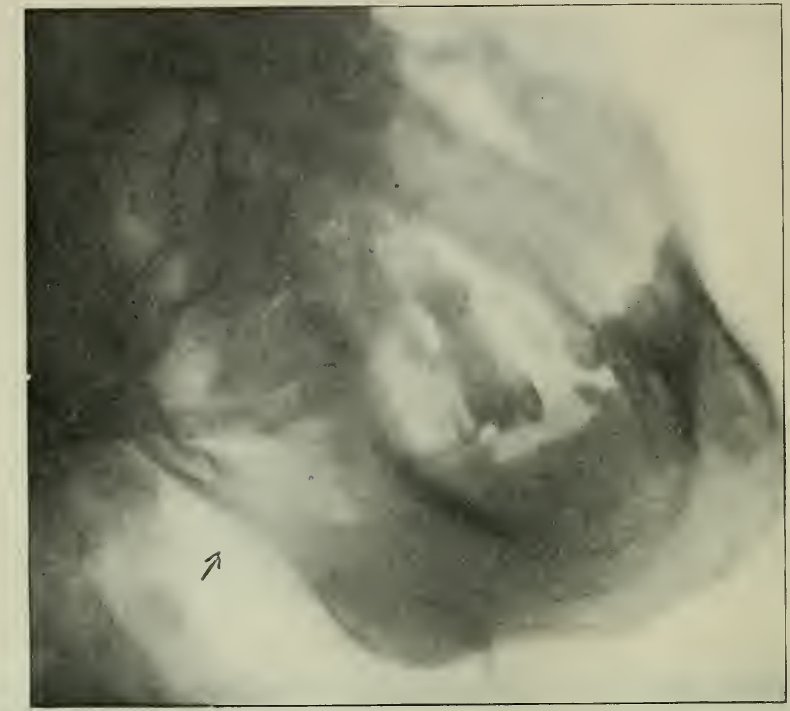

FIG. 13 (Case III).

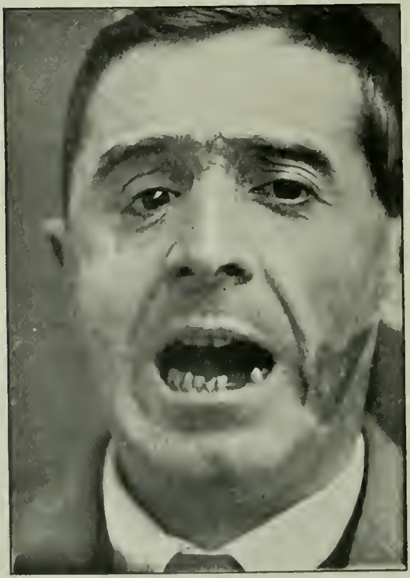

FIG. 14 (Case III).

of the inferior temporal crest, so as to reduce the entrance to the pterygoid fossa from the temporal region to a small rounded aperture. The depressed and ankylosed portions of the arch and malar bone were removed and the coronoid resected. The coronoid was found to be densely sclerosed and firmly ankylosed to the pterygoid surface of the maxilla as far down as the tuberosity, the bone in this region being of ivory hardness; no movement of the lower jaw could be obtained until this lower limit had been passed with the gouge forceps. The jaws then readily opened to admit a wedge 2½ cm. thick between the teeth. On the day following the operation the patient could open his mouth to this extent without assistance and on the second day was masticating soft food without pain. Two months after the operation the gape was 2½ cm. (fig. 14), and the power of mastication was steadily improving.

Case IV.—Lieutenant C., accidentally injured in the right malar region, with fracture of the zygoma, by bolt from bursting rifle. Since then patient had undergone several sequestrotomies, and in June, and again in July, his jaw had been forced open under an anæsthetic; this, according to the patient, failed to effect any improvement, and, indeed, had been followed by a steady reduction in his gape. On admission to Queen's Hospital the gape was 1 cm. There was a depressed scar in the infra-orbital region and marked prominence of the right malar region. On opening his mouth there was some deviation to the side of the injury; wedging merely increased this deviation without materially increasing his gape. At the operation, August, 1919, the posterior two-thirds of the zygoma was displaced downwards. The anterior part of the arch and malar bone was loose, and save that the zygomatic portion was removed, was left in position. The tip of the coronoid was found to be normal, but the anterior border was attached by a stout fibrous band passing upwards to the pterygoid aspect of the maxilla. As in the previous cases, the coronoid was removed, including the whole of the anterior border. A week after the operation he had a voluntary gape of 2 cm.

Case V.—Mr. X., aged 18, with a history of complete closure of the jaws following an attack of measles when aged 4. The right elbow and hip-joint were also affected at the same time, and were opened at the Hospital for Sick Children, Great Ormond Street, where the patient was under the care of Dr. Colman for some five months. When aged 9 the right condyle was resected through a transverse incision along the upper border of the zygoma, at University College Hospital, but without any marked improvement in the patient's condition being effected. He came to me in June, 1919. The molar teeth were firmly clenched and only partly erupted. Upper and lower incisors both protruded considerably, but the under development of the lower jaw, due to the early involvement of the epiphysis of either side resulted in the lower incisors lying so far behind the upper that a gap existed through which the patient had managed to feed himself (fig. 15, p. 34). A slight tilting of the right side of the mandible could be obtained, but none at all of the left. I operated upon him in September, 1919, and found the left temporo-mandibular joint

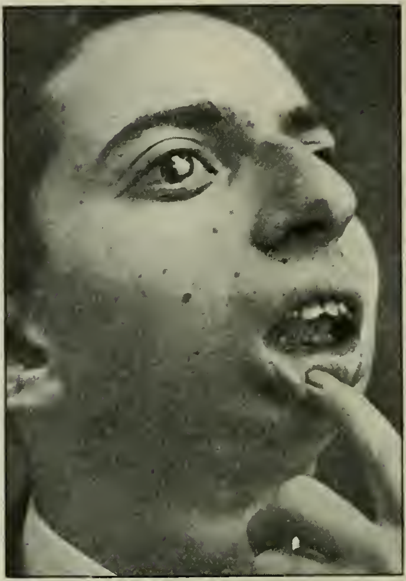

Fig. 15 (Case V).

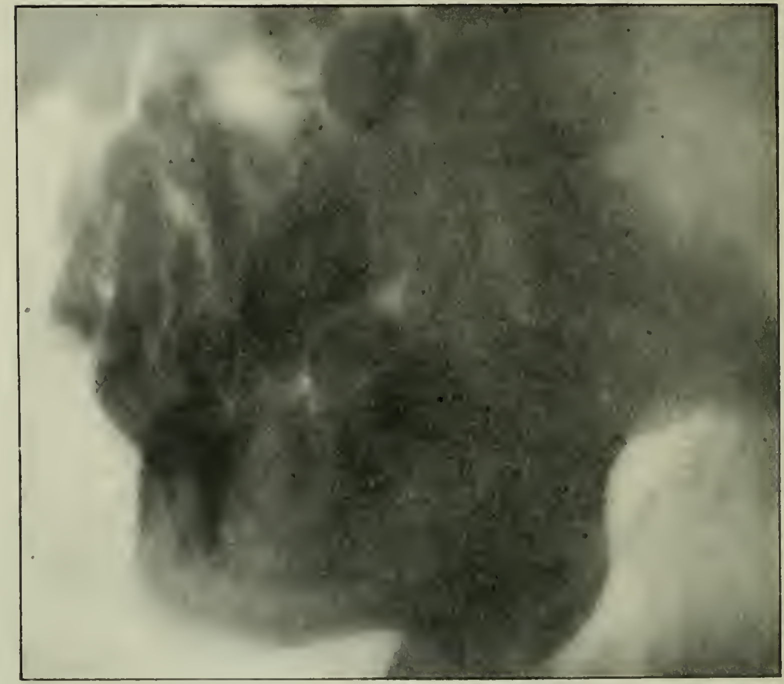

Fig. 16 (Case V).

represented by a mass of bone of ivory hardness entirely obliterating all trace of the joint, and causing the ascending ramus to blend smoothly with the surrounding bone (fig. 16). The neck was resected in the manner already described. No attempt was made to remove the mass of bone representing the condyle and glenoid fossa, this being preserved as a support for the future pseudarthrosis. The coronoid was not sclerosed but it was firmly bound by fibrous tissue, and, apparently, in places by soft bone also, to the surrounding structures, and especially to the lower part of the pterygoid aspect of the maxilla. It was not until the complete removal of the coronoid had been accomplished that any considerable degree of mobility of that side of the jaw was obtained. The right side was similarly treated at a second operation a fortnight later. Here in place of the bony ankylosis found on the left side there was a fairly firm fibrous union of the neck and region of the glenoid fossa. It was from this side that the condyle had been removed ten years previously. The fibrous union was completely resected, but, as on the left side, movement of the jaw was not free until the coronoid had been removed also. On account of the precarious condition of the patient's teeth no attempt was made to gag open the mouth at the time of the operation, but a few days later the patient was moving his jaw voluntarily and without pain through a distance of 1 cm., and could give the finger a very effective bite with the teeth. By means of a wooden wedge, which the patient used himself, a sufficient gape was obtained to allow of the front teeth being capped. The

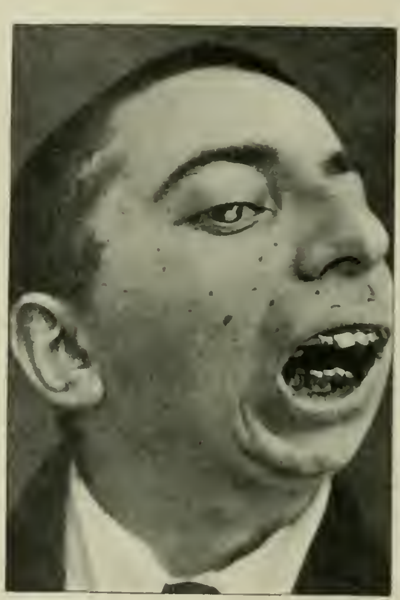

FIG. 17 (Case V).

jaw was then wrenched open under gas and fitted with an elastic gag. Two weeks later the patient had a gape of over 2 cm. between the cutting edges of his incisor teeth, was chewing soft food without pain, and had a power of bite that made it very unpleasant to leave the finger between his incisor teeth (fig. 17, p. 35).

SUMMARY.

Five cases of closure of the jaws are described. Four were traumatic in origin, with fracture of the zygoma, with or without direct involvement of the coronoid, but without involvement of the temporo-mandibular joint. The fifth case, which was bilateral, was infective in origin and was due to an acute arthritis of the temporo-mandibular joint in infancy. In each case, however, there had been infection in the zygomatic fossa, and in each case, quite apart from any direct involvement of the coronoid in the injury, there was a definite fixation of this structure, by the development of fibrous or bony adhesions, to the lower part of the pterygoid aspect of the maxilla.

The incision and mode of approach to the pterygoid fossa used in operating upon these cases not only allowed of an easy approach to this region without risk of injury, temporary or permanent, to the branches of the facial nerve, but allowed also of the complete subperiosteal resection of the coronoid and condylar region of both sides without materially affecting the subsequent power of mastication.

Section of Odontology.

President—Sir Frank Colyer, F.R.C.S., L.D.S.

Dental Sepsis in Children: Its Consequences and Treatment.[1]

By F. St. J. Steadman, D.P.H., L.R.C.P., M.R.C.S., L.D.S.

Among the many lessons which the great World War of the past five years has brought home to us is the important fact that the physique of our race is by no means as good as it ought to be; indeed, the Prime Minister, in a speech last year, stated that the physical examination of recruits had revealed an appalling state of affairs. In cotton weavers, for example, only 19·6 per cent. of those examined were placed in Grade 1. In one day's batch of fifty-nine men, only three were found to be in a fairly good physical condition: A struggle for our very existence as a Nation, such as that through which we have just passed, brings home to us in a way that nothing else can the gravity of this matter.

The causes of this deterioration of our national physique are doubtless many. Existing conditions entail the performance of a large amount of work in a vitiated atmosphere, and diseases such as tuberculosis, rheumatism and valvular disease of the heart are very rife. It is the duty of the medical profession (in which I, of course, include the dental) to determine as accurately as possible where we are at fault and to strive against those conditions which impair the health and strength of the community.

The Medical Society of London has commenced this investigation, but judging from the report of a meeting held on November 25, 1918,[2] in which the causes of rejection were classified and in which there is no mention made of dental disease, they are once again overlooking the commonest cause of all. When we realize the extraordinary prevalence of dental disease in all classes of the community, it is truly strange that the medical profession is, even to-day, not appreciating its vital effect on the physique of our race. In a recent article by Dr. J. D. Comrie,[1] however, analysing the physical defects among the general male population, the writer states that in 10,000 recruits 928 had artificial teeth, while another 1,120 had lost half their teeth at least, and that a septic condition of the teeth with deposit of tartar and gingivitis was frequently associated with dyspepsia and invariably with a deteriorated physique.

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[1] At a meeting of the Section, held January 26, 1920.

[2] *Lancet*, 1918, ii, p. 744.

I think it will be generally admitted that chronic disease in childhood, the effects of which spread over a period of years, must have a profound influence upon mental and physical growth. Of all chronic diseases of this character, dental disease is by far the most common, as something like 80 per cent. of the children of the race are suffering from dental diseases.

It is my hope that the discussion, which I trust will follow this paper, will give a clear lead to the dental profession on the treatment of dental sepsis in children. Many discussions fail in their object because the limits are not clearly defined and speakers are apt to wander from the point at issue; and too often discussions become merely academic and of no practical value. I now propose, therefore, to lay down the limits of this paper.

I assume that we are all agreed upon the tremendous importance to the child of a perfect dental arch; that better than combating dental diseases is prevention by securing a clean mouth in the pregnant mother, and her proper feeding, breast feeding in infancy, the establishment of proper nasal breathing, and later proper diet—a diet requiring efficient mastication. Further, that we are agreed upon the immense importance of early and constant dental supervision so that caries can be treated early, either by silver nitrate or, when the cavities are shallow and do not involve the pulp, by suitable fillings; that it is generally a reflection upon the parents, and more especially upon the dental adviser (provided that his inspections have been sufficiently frequent), if a pulp is allowed to become infected or if treatment is delayed until infection of the pulp has occurred. I want to emphasize these points because it has been said that I do not believe in the conservative treatment of teeth in children. I want to make it quite clear that I do all I can in my practice to conserve the deciduous as carefully as I do the permanent teeth.

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[1] *Lancet*, 1919, ii, p. 958.

In my paper to-night, I propose to deal with the effect of marked dental sepsis on the health of children, and with the treatment of those all too common cases in which the prophylactic and remedial measures just enumerated have not been carried out, and the child has been brought to us with advanced caries in one or many teeth. Moreover, I want it to be clearly understood that I have in my mind the treatment of this sepsis from a broad national point of view. I am not concerned so much to-night with the treatment of the children of the rich, for although of course the fundamental principles are the same for both rich and poor, it is clear that in dental diseases, like all other diseases, there is often one means for the rich and another for the poor. The advice given to a wealthy, leisured mother (who is aided by nurses whose sole work is to look after her children) may be different from the advice given to the busy working-class mother, whose day's work begins when she gets up in the morning and ends only when she goes to bed at night. It is as futile to tell such a mother with five or six children that she must see that they clean their teeth carefully after every meal with a tooth-brush, aided by floss silk, as it is for a physician to order the bread winner of a family, earning £3 a week and suffering from phthisis, to take a twelve months' sea voyage.

THE EFFECTS OF DENTAL SEPSIS UPON CHILDREN.

The effects of dental sepsis upon children can be considered under two main headings: (A) General; (B) Local.

(A) *General.*—It was during my work at the Belgrave Hospital for Children some years ago that I first began to appreciate the profound effect that dental sepsis may have upon the general health of the sufferers. When I first took up this work I thought that the state of the pale-faced, tired-looking, under weighted and mentally inefficient children whom I saw was the result of semi-starvation, due to poverty. One day, however, I happened to notice a mother who had brought such a child to me and who had with her another of her children, who had not this half-starved appearance but who looked healthy and well. Thinking that this marked difference in appearance might be due to favouritism on the part of the mother, I questioned her closely, but found that her treatment with regard to the food of both children was the same. I then looked at the mouths of the two children. My little patient was suffering from advanced caries, with its consequent gingivitis, while the mouth of the other was healthy. I removed the septic teeth from the one, and a few months later it was as healthy in appearance as the other. This very common and ordinary case illustrates very well the effect dental sepsis frequently has upon children. Mental and physical growth is retarded. Pale faced and anæmic, their eyes have not the normal lustre of healthy children, and they have a tired and sleepy look about them.

Sir Frank Colyer was, I think, the first to weigh these children before and after treatment, and in a paper read before the Manchester Odontological Society, on December 6, 1910,[1] gave the weights of some of the cases he had treated for sepsis. He showed that there is frequently a marked increase in weight, too great to be accounted for by the normal increase of growth. He gives details of the following four cases:—

M. L., aged 4 years 6 months. When first seen was suffering from gastro-intestinal trouble. Teeth removed $\frac{\mathrm{DCBA} \mid \mathrm{ABCD}}{\mathrm{ED} \mid \mathrm{DE}}$. In this case the $\underline{\mathrm{E} \mid \mathrm{E}}$ were not carious and were retained until the $\underline{6 \mid 6}$ erupted. This patient lost her gastro-intestinal trouble, and between June 8 and October 2, 1909, increased in weight from 2 st. 4 lb. to 2 st. 8 lb. The normal weight should have been 2 st. 8 lb., and the normal increase in four months 1½ lb. This patient increased 4 lb.

W. G., aged 7 years 4 months. Stated always to have indigestion. Teeth removed $\frac{6\mathrm{EDC} \mid \mathrm{CDE}6}{6\mathrm{EDC} \mid \mathrm{CDE}6}$. The teeth were removed on December 6, 1909, the patient weighing 3 st. 2½ lb. On February 7 the weight had increased to 3 st. 5 lb. and all symptoms of gastro-intestinal trouble had ceased. The average weight of this boy should have been about 3 st. 9½ lb., and the increase in two months a little under 1 lb. The weight, however, increased in two months 2½ lb.

A. R., aged 6 years 7 months. Complained of constant feverish attacks. Teeth removed $\frac{\mathrm{EDBA} \mid \mathrm{ABDE}}{\mathrm{ED} \mid \mathrm{DE}}$. Between December, 1909, and October 23, 1910, the weight had increased from 3 st. 5 lb. to 3 st. 10 lb. The feverish attacks had ceased and the child was sleeping and eating well. This patient was about average weight and the increase in weight about normal.

The rapid increase in weight following extraction is at times very marked.

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[1] *Dental Record*, 1911, xxxi, p. 67.

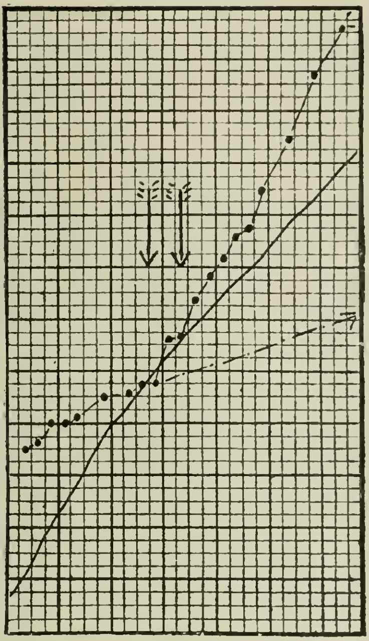

CHART I.

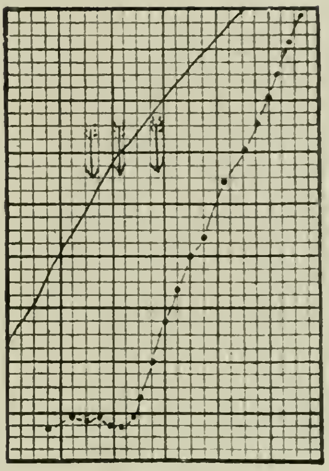

CHART II.[1]

Chart I.—Weight curve of child. The continuous line is Budin's curve. The interrupted line gives the record of the child's weight. The arrows indicate the extraction of maternal teeth; three in the first case, two in the second. The direction of the interrupted line before the extraction would lead to a weight of 14 lb. in one year; after the extraction to one of 22 lb.

Chart II.—Weight curve of child. Lines as before. The arrows indicate the extraction of eight teeth, twelve teeth and four roots, and eight teeth respectively.

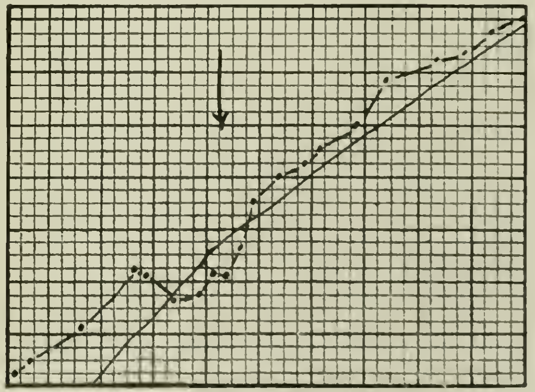

CHART III.

Weight curve of child. Lines as before. The arrow indicates the extraction of six maternal teeth.

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[1] Charts I, II are reproduced from Mr. J. F. Colyer's "Dental Pathology and Surgery," by permission of Messrs. Longmans, Green & Co.

For example, in his fourth case, a boy aged 10 years 2 months, it was found advisable to remove $\frac{EDC|CDE}{EC\ |CDE}$. Between June and October this boy boy increased from 4 st. 6 lb. to 4 st. 11 lb. The normal weight should have been about 4 st. 11½ lb. and the average increase in four months about 2 lb. This child in the period increased 5 lb.

I have weighed a considerable number of children and have been able to amply confirm Sir Frank Colyer's results. As he points out, the increase in weight is not always shown. In a few cases there may even be a slight decline. This is frequently traceable to sepsis in other parts; for example, tonsils, or nasopharynx.

One of the chief ways by which dental sepsis in children produces this loss of mental and physical growth is by the loss of sleep. A careful investigation of the history of these cases will frequently disclose the fact that for months the child has not slept well. Its nights have been disturbed by pain. This important symptom has to be looked for, as children do not as a rule complain of it. When one considers the effect of a series of bad nights upon oneself, the importance to the child of this lack of sleep can be dimly realized. I wish to emphasize this point with all the power that I can. Its importance cannot be exaggerated.

Some years ago, the father of one of my young patients at the Belgrave Hospital for Children took the trouble to find out where I lived to visit me for the purpose of thanking me for my treatment of his son, aged 9. Some fourteen months before, I had removed his twelve molar teeth, $\frac{6ED|DE6}{6ED|DE6}$. The father told me that up to that time the boy had always been delicate and ailing. He had had many restless nights from toothache, and had constantly needed medical care. He was small, under weight, pale faced and very backward at school. He had "suffered terribly." A few days after the operation he began to change rapidly. He has now rosy cheeks, looks well, has regained his normal weight, has not once been ill, and has made rapid strides at school. It seemed, as his father said, as though a millstone had been removed from about his neck.

A common result of oral sepsis in children consists in gastro-intestinal disorders, evidenced by gastric and abdominal pain, diarrhœa of offensive character, with much undigested food in the motions, marked wasting, fretfulness, night terrors, loss of appetite, sleeplessness and pallor of the face. In treating a case such as this, it would appear to be a common-sense thing first to inspect the mouth and to remove any possible source of infection in it before proceeding to treat the stomach; yet many medical practitioners still neglect to do this. In one case of this kind which came under my care, a child, aged 7, had been treated for no less than fourteen months by her doctor, who had made only a very cursory oral examination. Indeed, as the child was frequently awake at night with toothache, the mother had more than once asked him whether he thought that the condition might be due to the teeth, and whether he thought it advisable to have the teeth treated, but the doctor had replied that the teeth must on no account be removed, because that would injure the permanent teeth, and, moreover, would prevent mastication! At last the mother, finding that the doctor's medicines did no good, determined to have the teeth attended to. On examination I found advanced caries with exposure of nearly all the pulps in $\frac{\mathrm{E\,D\,C} \mid \mathrm{C\,D\,E}}{\mathrm{E\,D} \mid \mathrm{D\,E}}$ with definite chronic abscesses over $\frac{\mathrm{D} \mid \phantom{\mathrm{D}}}{\mathrm{E} \mid \mathrm{D}}$. Three or four weeks after the removal of these teeth the child was practically well.

This gastro-intestinal condition is due to sepsis and not to lack of mastication, as is proved by the fact that the patients frequently get well very soon after extraction of the teeth, and almost before the gums have healed, that is to say, while the gums are still too tender to allow of mastication upon the teeth that remain. This is borne out, I think, by the most interesting observations of Dr. H. Waller upon dental disease in nursing women. He shows that in many instances in which a breast-fed baby ceases to thrive while still at the breast, the trouble can be traced to sepsis in the mouth of the mother. He gives details accompanied by charts (here reproduced) of three cases selected from nearly 200, demonstrating the marked gain in weight of the infants after the extraction of septic maternal teeth[1] (*see* Charts I-III, p. 41).

In these cases, it is clear, I think, that the digestive apparatus of the child is unchanged; that the improved health has been brought about by the removal of sepsis in its food. Similarly, as Dr. Sim Wallace points out, anæmia of other than dental origin is liable to be aggravated by the chronic toxæmia and septic absorption which may be associated with dental sepsis.[2] Other chronic diseases such as tuberculosis cannot but be prejudicially affected by an insanitary state of the mouth. Typhoid and scarlet fever and other zymotic diseases have been shown by Hunter to be much more dangerous when dental sepsis is present.[1] Further, sepsis from the mouth may be absorbed into the blood-stream, giving rise to such diseases as septicæmia or endocarditis.

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[1] *Lancet*, 1916, ii, p. 785.

[2] "Dental Diseases and Public Health," p. 18.

(B) *Local.*—Under this heading I include the adjacent parts to which infection sometimes spreads by continuity of tissue. Pharyngitis, tonsillitis, otitis media, and—much more rarely—meningitis and such like troubles. Infection may pass down the lymphatics to the submaxillary and cervical glands. Some hold that glands are so commonly enlarged in children that it is not worth while even examining for them. In my opinion, such teaching is absolutely wrong. I think (although I know that I am on controversial ground) that normally the submaxillary and cervical glands are not palpable in children; that if they are, there is sepsis somewhere. Carious teeth frequently produce enlarged glands, as is proved by the fact that these glands disappear after the removal of the teeth. At the same time, I am well aware that we see enlarged cervical glands in children with perfectly clean mouths.

We dental surgeons are apt to forget that these glands drain a very large area apart from the teeth and oral mucosa—for instance, the tonsils, the nasopharynx, the orbits, the skin of the face, the anterior part of the scalp, &c. We also commonly see children with very septic teeth in which the glands are apparently not affected, but as Mr. G. Morgan points out, this freedom from injury to the gland is often more apparent than real:—

> "A gland constantly irritated by micro-organisms from a carious tooth is potentially weaker; it is more vulnerable than a gland not so irritated. This greater susceptibility to injury is shown by the effect of strain on such glands. I think we may take it for granted that a gland becoming tender and remaining for some weeks enlarged from simple strain is not a healthy gland. Its resisting power has been lowered by having to deal with micro-organisms, perhaps in no great amount, and I think one may say of low virulence, but spread over long periods. A girl, aged 8, coming of a very healthy family and living in a good house, slipped whilst using dumb-bells and strained the muscle of her neck. The next day there were two tender glands a little in front of the right angle of the jaw. The six-year-old lower molar on this side was badly decayed and had been so for a year, yet until the strain there was no sign of enlarged or tender glands. After the strain the glands remained enlarged for several months, and one finally became tubercular. Glands in the groin will sometimes enlarge from the strain of football, but careful examination of the

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[1] "Oral Sepsis as a Cause of Disease in Relation to General Medicine," *Brit. Med. Journ.*, 1904, ii, p. 1358.

area of drainage and going into past history will nearly always prove that the glands were not in their normal state at the time of injury. Measles and whooping cough often play the same rôle as strain—i.e., glands injured by carious teeth, and yet not visibly showing any change will enlarge more quickly and remain enlarged longer than glands not so injured. I could supplement these cases by others proving that strain alone does not cause inflammatory enlargement of healthy glands." [1]

It should be remembered that a very slight septic focus in children will often lead to enlarged glands, and if glands are palpable we should endeavour to find the cause. It is true that the glands often rapidly diminish in size after the removal of septic teeth, but they do not always quite disappear. They appear to be fibrosed, and years later, at the age of puberty, they occasionally break out again, and examination may show them to be tubercular. Occasionally these infected glands break down, and the infection is carried to other parts of the body, giving rise, possibly, to tubercular lesions in the bones, or it is even possible that tubercular meningitis may be set up. Mr. J. G. Turner has shown that sepsis in the deciduous teeth not uncommonly leads to damage of the permanent teeth beneath them, as shown by hypoplasia and some of those deformities which have been classed under the term dilaceration, commonest among the premolars and fairly common amongst the incisors.[2] I have examined a number of radiographs of the bone round septic deciduous teeth to ascertain whether any rarefying osteitis is to be observed, especially in those cases in which the pulps have died under fillings. I am not able to make a definite statement upon this at present, but hope to bring this point up again on a future occasion.

Lastly, the carious tender teeth by disuse bring about a gingivitis which may spread eventually to the periodontal membrane of the permanent teeth, with all its consequent ill-effects upon the health of the individual. Many of our citizens, owing to neglect of their teeth in childhood, have had dirty septic mouths all their lives, so that we need scarcely wonder at their poor physique.

Such, then, is a by no means complete picture of the possible consequences of sepsis in children and upon the national physique. No doubt one frequently sees children with exceedingly septic mouths

[1] "Lymph Glands in Relation to the Teeth and Gums," *Brit. Dent. Journ.*, 1903, xxiv, p. 525.

[2] "Injury to the Teeth of Succession by Abscess of the Temporary Teeth," *Brit. Dent. Journ.*, 1909, xxx, p. 1235.

apparently in robust health. I have reason to suspect, however, that in some of these cases at any rate this robust health is more apparent than real. Then arises the interesting question, What percentage of children with marked sepsis in their mouths have their general health thereby affected? It is a question which I fear in the present state of our knowledge we cannot answer, but one worth trying to solve in the future.

I have treated between seven and eight thousand children in the way that I am about to describe, but these have been mostly hospital patients; consequently, I have only been able to follow up the history of a comparatively small number. Of these I can definitely state that a considerable percentage have shown marked improvement in their general health, and I know of none whose health has been adversely affected by the removal of their septic teeth. The illustrative cases I am giving this evening are merely examples of very many others of the same kind which have come within my experience.

Would that I had the power of a Raemaker in depicting these things graphically! I would draw a picture showing the enormous number of children of our race wrapped in a pall or dense fog, the fog of dental sepsis, retarding their mental and physical development. Drastic treatment of this sepsis is like snatching away the pall or lifting the fog. Normal weight is regained, their eyes once more shine brightly, and the palest cheeks become rosy. These children have been given a chance to grow.

THE TREATMENT OF DENTAL SEPSIS IN CHILDREN.

All deciduous teeth in which the decay is sufficiently advanced to infect the pulp, I extract, and generally their antagonists also. I only fill teeth when I feel sure that the pulp is not affected. This condemns at once the majority of decayed deciduous teeth, because the pulps of these are relatively larger than in permanent teeth, so that a comparatively small cavity is sufficient to infect the pulp. By infected pulp I do not necessarily mean an exposed pulp. Many pulps are infected long before the actual exposure has occurred.

I treat the permanent molar teeth in the same way as the deciduous teeth if the roots are incomplete at the time of the pulp infection. I know of no way of dealing with infected pulps in children which I regard as sound. The treatment recommended by many practitioners of sterilizing and capping the pulp is, in my judgment, dangerous, as in the majority of cases so treated the pulp eventually suppurates and dies under the filling and the teeth remain tender and uncomfortable, so that the child's confidence in them is not restored and he does not use them for the purpose of mastication; frequently they ache and he remains with his nights disturbed. The following case which came under my care illustrates these points:—

The patient was a boy, aged 5, and was brought to me by his mother on account of pain in his teeth which caused him to have restless nights. On examination, I found he had no less than eleven amalgam fillings inserted into his deciduous molars. Three of these teeth $\begin{array}{r|l} & \text{E} \\ \cline{2-2} \text{E} & \text{D} \end{array}$ were obviously dead and tender and pus could be squeezed out from the pockets around them. The gums all round the mouth were inflamed and dirty. It was clear that the child had not used these teeth for the purpose of mastication for some time. The submaxillary lymphatic glands on either side were swollen and tender, as also were the cervical glands on the left side. The mother said the child never seemed well, was always irritable and cried himself to sleep when put to bed and frequently woke during the night, at such times complaining of pain in his teeth; that he had not had a good night's rest for months, and that he did not care for his food and did not eat enough. The child's general appearance amply bore out his mother's statements. He was small for his age, pale faced, and his eyes lacked the brightness and intelligence of the normal child. The dentist who filled these teeth had told the mother that nothing further could be done until the child was aged 10 or 11, when these teeth would be shed, and that it was absolutely necessary to retain them for the purpose of mastication. A second dentist had confirmed this opinion. In the face of these opinions of two of my fellow practitioners, I had some difficulty in getting the parents' consent to the immediate extraction of the deciduous molars. Fortunately I was supported on this occasion by the family doctor (more often than not, I regret to record that I have found in him a strenuous opponent). The operation was performed on March 18, 1913. The effect was startling. On that night and each following night the child slept well; there was no crying out and no restlessness whatever. On April 22 the glands were only just palpable, and were no longer tender, and the beneficial effect of the good nights on the child was obvious. The pallor of his cheeks and the sleepy, tired look had vanished. The mother said that the improvement in health "seemed marvellous" and the child was "altogether brighter in himself." He was eating well and seemed always to be hungry. This child was not weighed, but it was apparent that he was steadily putting on weight.

The argument against extensive extraction in children, that mastication is lost, is futile. I cannot understand how it is that medical and dental practitioners, who use this argument, can fail to see for themselves that the power of mastication is already lost. No child will masticate on tender teeth and exposed pulps; but one may actually take up the other line of argument and say that by the removal of tender teeth the area of mastication is increased. An exposed pulp in a deciduous molar means that the child will not masticate on that side of the mouth, so that the important six-year-old molars behind them are not functional; remove the tender tooth and these teeth will be restored to usefulness.

Experience shows that the child regains its normal weight and health after extraction of all septic molars. In other words, experience shows that it is better for the child, if food has to be bolted, that the food be as clean and as uncontaminated by pathogenic organisms as possible. Here, in passing, I would like to point out that if it has been decided to extract the six-year-old molars, it is best to do so early, when the child is about 10 years old, and not wait until the twelve-year-old molars have erupted, so as to allow these latter teeth to come forward.

If you take thirty children and arrange them in a group, and ten of these children have all of their teeth present and their teeth and mouths perfectly healthy, and ten have had most or all of their teeth removed but their mouths otherwise healthy, while the remaining ten have septic teeth and inflamed gums; it will be easy for the trained eye—aided if you like by the weighing machine—to pick out these last ten children, but it will not be possible to separate the remaining twenty.

Another argument against the extraction of teeth is that it prevents the proper growth and development of the jaws. If the extraction of teeth adversely influences the growth of the jaws, it is said to be clear that if several teeth are removed on one side and not on the other, the jaws on the one side from which these teeth have been extracted will show a deficient growth; whereas, the side from which the teeth have not been removed will grow normally, giving the whole face a lopsided appearance. Experience shows that save in exceptional cases nothing of the kind happens; on the contrary, the jaws still reach their normal development on both sides, in spite of the loss of the teeth on the one side. Or again, if we extract teeth from the upper jaw and not from the lower, it has been said that the upper jaw will not grow properly but the lower will, so that we shall have brought about an "inferior protrusion" or vice versa; but nothing of the kind happens, which shows that the normal growth of the upper jaw has apparently not been affected by the extraction of the teeth.

A more important argument which requires attention is that the early extraction of deciduous teeth will lead to the forward movement of the six-year-old molars, thus causing crowding and irregularities of the arch later on. If the teeth are extracted before the permanent molars have occluded, there is no doubt that this will take place, but provided the patient comes frequently for dental inspection, at the right moment one of the permanent teeth in each corner of the mouth can be sacrificed. These teeth may be either the four six-year-old molars or the four premolars, according to circumstances. If the extractions take place after the permanent molars have occluded, there will be very little forward movement.

I have said that, as a general rule, I remove not only the carious teeth but also the antagonists. The importance of this was brought home to me by a demonstration by Sir Frank Colyer at one of the "At Homes" of the Royal Dental Hospital some years ago. A young child who had one lower deciduous molar tooth missing but all the rest of the teeth present and healthy, was given a meal of soft food (bread and butter) and was then given an apple. The eating of the apple mechanically cleansed away all the soft food which remained around the teeth after the meal of bread and butter, except that around the tooth which was opposite the place where the tooth was missing. Around this tooth was a soft film of carbohydrate food, and when this was scraped off, a little ring of inflamed gum all around the tooth was seen, due to the frequent lodgment of food. This, to my mind, clearly demonstrates the importance of removing this functionless tooth, for if left, it is liable, owing to the tenderness of the gum around it, to prevent mastication and to force the child into the pernicious habit of unilateral mastication. In those cases, however, in which the four lower deciduous molars and the first upper, but not the second upper, deciduous molars are carious, I leave the latter (in children aged under 6) in position, following the teaching of Sir Frank Colyer, until the permanent molars have erupted and occluded.

I think we do not realize sufficiently the importance of preventing unilateral mastication. It means that on the unused side we shall get a marked gingivitis, and more important still, this habit is liable to become permanent, so that later on, when the permanent teeth have erupted, they quickly become septic also. I am convinced that many cases of periodontal disease can be traced back to tender teeth in the child.

There is another argument in favour of extraction which, however, I bring forward with some hesitation—that of expediency. One would perhaps hardly be justified in acting upon expediency alone were it not supported by the other and more powerful arguments that I have already stated.

Of the 7,000,000 or so children of this country, approximately 6,000,000 require dental aid. I think if I allow, on an average, one hour's work for each child to do what is required, I shall not be accused of overstating the case. If this work is carried on at the rate of five and a half hours a day, it will take one man nearly 3,000 years to complete. This rough calculation will show that we have nothing like enough dental surgeons to get through the work. On the other, if we extract all teeth with infected pulps, a tremendous amount of time will have been saved. It is difficult to estimate the beneficial effect this work would have upon the health of the children, and consequently, upon the physique of the Nation; although it is true we should have a greater number of crowded arches to deal with. A result cheap indeed, at the price! Of course, there is no doubt that as the years go on this huge mass of dental work in children will gradually lessen as the teaching of prevention becomes general in schools.

It takes a considerable amount of courage to condemn a large number of teeth in a child. This necessary courage can only be acquired by observing, recording and pondering over the evil consequences of leaving these septic teeth. Many members of the dental profession at the present day appears to lack this courage, or have not yet realized sufficiently the great harm they are doing to their patients by leaving septic teeth. Four times during the past six months parents have brought children to me simply and solely because they have heard that I am willing to treat children. One case was that of a boy, aged 5; every tooth in the child's mouth was carious almost down to the gum margin, causing a septic gingivitis and profoundly affecting his health. The father told me that he had taken the child to two dentists and a doctor, each of whom said that nothing could be done. The father told me that common sense convinced him that something could and ought to be done. Removal of all the deciduous teeth has had a very marked beneficial effect upon this boy's health, but will no doubt lead to crowding later on and the sacrifice of four permanent teeth. I have explained this to the parents, but they do not seem to think that this is worth considering in face of the immense improvement in the child's general health—an attitude which I cordially endorse. In another case, a little boy was brought to me with a large swelling in connexion with his left lower deciduous molar. A doctor had treated this for eight weeks, stating that the tooth, being a temporary one, must on no account be removed. During this time the swelling on the jaw had become hard and brawny; pus had burst through the skin, and when I came to remove the tooth I found a large piece of necrosed bone at the bottom of the sinus. Had the tooth been removed eight weeks sooner the patient would have been spared permanent disfigurement.

It is true that a considerable number of dentists do not wish to treat children. It is not given to everyone to have the patience and tact that is necessary to deal with young children, but if a practitioner has not these qualities he should manfully recognize the fact and send children to those who will undertake their treatment.

The ignorance of a small but harmful minority of medical practitioners concerning dental matters is, without exaggeration, nearly as great as that of the general public. These men cannot yet appreciate their own ignorance and sometimes give advice on dental matters—advice which is not only wrong but often positively extremely harmful. Progress is being made, but it is slow. In the war against dental disease and its train of consequences; in the effort to snatch off the pall and clear away the fog of sepsis which envelops our children, I have sometimes thought that these medical practitioners are like evil spirits, doing their utmost to prevent the clearance of that fog. On more than one occasion when I have urged the removal of abscessed teeth which are keeping their possessor awake night after night with pain, besides causing severe general ill-health, the medical adviser has stepped in and said that nothing must be done to the teeth until the child's health has improved. It is difficult to follow what is in the mind of the giver of such advice. What would one say if, when his house was burning, the firemen told him that they must wait until the heat had cooled down a bit before they commenced to put it out?

Lastly, I would like to emphasize one more point, which I consider very important, namely, that if extensive extraction has been decided upon, it should be carried out at one time under one general anæsthetic such as ethyl chloride. In this way we put much less strain upon the nerves of the little patients. In one case which came under my observation a dentist had condemned twelve septic teeth and proceeded to remove them, one at a time, under gas. After five teeth had been removed in this way the child's nerves had been shattered. I can see no great objection and very little risk in removing all that are necessary at one operation.

DISCUSSION.

Dr. ARTHUR SAUNDERS: I should like in the first place to take the opportunity of stating that from the point of view of a physician I am in complete agreement with what Mr. Steadman has said as to the far-reaching constitutional effects of dental sepsis in children, and personally to thank both him and Mr. Lloyd Williams for the valuable service they have rendered to the children in my department in removing in so many cases the primary cause of their ill-health. He has well described the dull, thin, undersized, anæmic patients who are the victims of this condition. The wasting and anæmia are often so marked and accompanied also by headaches, feverishness, night sweating, and slight cyanosis that the suspicion of early pulmonary tuberculosis is often aroused. The cyanosis is usually associated with slight dilatation of the heart, both no doubt being due to a toxic myocarditis. Albuminuria also is frequently present, and by no means rarely the presence of albumin in considerable quantities, together with casts and red and white blood-cells, point to a more definite nephritis. And it is to dental sepsis as a not infrequent and less commonly recognized cause of nephritis, and particularly of a hæmorrhagic nephritis, that I would like especially to refer. Such a nephritis, sudden as a rule in its onset, may present early a smoky or bright red appearance of the urine from the presence of blood, and closely resembles the glomerular nephritis seen in scarlet fever. When the condition is treated at once by the removal of the septic teeth, it is remarkable how quickly the blood disappears and the urine becomes normal. In other cases, however, where the damage to the kidney is of long standing, the injury may be so great as to lead to a progressive or even fatal nephritis. I am very strongly in favour in these cases of the immediate removal of all the septic teeth at one sitting under a general anæsthetic.

Mr. E. B. DOWSETT: With regard to the charge brought by Mr. Steadman against medical practitioners generally, I think his experiences in that respect must have been rather unfortunate, as I must say I have always had the greatest possible support from the doctor on every similar occasion. Apart from this, I fully agree with the views expressed in the paper, and had been practising this method of treating children for some years before the War. I was fired with enthusiasm by our President about that time by seeing some of his cases of children radically treated and exhibited at a conversazione at Leicester Square. The results of early and multiple extractions of the temporary teeth, in such cases as described, so impressed me that I adopted the same procedure at once. Before the War I had a whole series of cases so treated at Guy's Hospital, all showing distinctly beneficial results therefrom. Recently we have instituted a special children's department at Guy's, under Mr. Bull, who I trust is now carrying on the good work. Of course, the two criticisms that have always been directed against such treatment are: (1) The small amount of masticating power left to children, and (2) irregularities arising later in the permanent dentition. With regard to the first, it is extraordinary how wonderfully well these children will and can eat with practically no teeth, for they all thrive and fatten without their teeth as they never did while carious and septic teeth were still in their mouths. Apropos of this, Mr. J. G. Turner, who will I am sure correct me if I am wrong, some few years ago, I believe, went so far as to declare that not even permanent teeth were necessary. With regard to the second criticism, the only irregularities that can arise consist in a slight crowding in the premolar region—a condition easily capable of treatment. This is a very small price to pay for a clean mouth and a healthy constitution, for a healthy constitution is the main and proved justification for the treatment described in the paper this evening. Treatment of exposed pulps in the temporary teeth should never be undertaken; there is no sound treatment except extraction. With regard to infection of the pulp before actual tangible exposure—although it is pathologically possible for the pulp to become infected through the tubes of hard dentine—nevertheless the general method I adopt is to fill cavities in temporary teeth,—provided I find a layer of *hard* dentine still overlying the pulp, but to extract the teeth when there is only a layer of decalcified and softened dentine over the pulp, or when the pulp is actually exposed.

Mr. A. T. PITTS: Mr. Steadman has brought a serious indictment against both the medical and dental professions in stating that some members of both professions do not sufficiently recognize the serious results of dental sepsis in children. During the last few years I have seen a large number of children at the Hospital for Sick Children, and I endorse all Mr. Steadman's views. The effects of dental sepsis in children, both local and general, are indeed serious, and the improvement in the general health, when the infected teeth are removed, is striking. Even in those cases in which it has been necessary to render the mouth practically edentulous, the children gain in weight, and are able to eat their food in comfort. It is not uncommon to see mouths in which the apices of necrosed roots have ulcerated through the gum, a condition comparable to sequestra being exfoliated. In any other part of the body this would be regarded as a serious condition, and yet in the mouth it will sometimes pass unheeded. I am in entire agreement with Mr. Steadman when he says that extraction is the only treatment for deciduous teeth which are septic or have exposed pulps; I do not know of any way in which they can be treated so as to leave the mouth free of infection. There is no doubt that premature loss of the deciduous teeth frequently leads to crowding of the permanent teeth, but compared to the evils of sepsis it is quite negligible. It has been stated that this crowding of the permanent teeth is a predisposing cause of periodontal disease in later life: even if this be so, I would say that an infection occurring in adult life when growth has ceased and metabolism has reached an equilibrium, is less serious than that occurring in a growing child who requires nourishment over and above that needed to repair the daily loss of energy.

Mr. ARTHUR COLYER: I regret that owing to my late arrival at the meeting I missed the opportunity of hearing the early part of Mr. Steadman's paper. With the part I did hear I am almost in entire agreement. One is naturally of opinion, after the teaching of the last twenty years, that the chief principle to keep in mind when treating children's teeth is that of prevention of local and general infection of the individual. I must say that I am rather surprised that it was necessary to emphasize the necessity of adopting such treatment as recommended by the reader of the paper. When stopping children's teeth, I believe in stopping them well. There is just one method of treatment, not new, which occasionally I find very efficacious. In nervous children in whom proximal cavities, either in the incisor or molar regions, are well developed thorough treatment by means of stoppings is sometimes almost impossible. In such cases I cut with fine stones broad V-shaped spaces and treat the proximal surfaces with nitrate of silver. Such treatment, if efficient use is made of the toothbrush, leads to arrest of caries and restoration of a healthy interdental gingiva. One benefit of fairly early removal of the second temporary molars is the opportunity afforded of stopping the medial surfaces of the six year molars before the carious condition, which is almost always present in these cases, has become extensive. One is able to insert early a thin line filling, and so to give the remaining unaffected structures a better chance of remaining sound. In private practice I much prefer gold as a filling in these small line cavities, because amalgam, by changing shape, creates spaces for the lodgment of food. One should not forget the use of Woodhouse plates, which hold back the permanent molars while the premolars and canines scramble into place. With reference to the knowledge possessed by doctors of the scientific treatment of children's teeth, my experience is not in accord with that of Mr. Steadman. They may not hold such advanced views as the writer of the paper, but the advice they give is based upon a good knowledge of the pathological conditions. As to dental surgeons who do not treat children's teeth, I would go further than a previous speaker. The practitioners who do not treat temporary teeth, and hold the opinion that such treatment is not necessary, and at the same time do not advise that the children be placed under the care of others, are in my opinion guilty of malpraxis, and should be placed on a separate register.

Mr. J. LEWIN PAYNE: There is one aspect of the subject upon which I think sufficient stress has not been laid in the course of this discussion, and that is the question of prophylactic treatment. If the paper is to give a clear lead to the profession in regard to dental sepsis in children, and especially if Mr. Steadman is to bring the matter before a meeting of medical men, it is most important that the methods of prophylaxis shall be fully and clearly stated. I cannot agree that it is futile to instruct either the mothers or the children in prophylaxis. On the contrary, I am convinced this is a most important part of our duty. How many children are taught to keep the mouth really clean? How many of them have a toothbrush of proper size and shape and with the bristles correctly arranged? How many children are taught natural cleanliness of the mouth by the employment of suitable food and drink, and the use of the tongue at the end of a meal? In my experience not 25 per cent. of the children of even well-to-do people know how to clean their teeth effectively. After all, when we recommend, as too often, alas, we are compelled to do for the sake of the general health, the extraction of large numbers of children's teeth we really acknowledge the defeat of surgery just as much as the general surgeon does when he is driven to amputate a limb. Prophylaxis alone can save us from this position, and it is our duty to make this more clear to our patients and to the public. Moreover, if prophylaxis is not taught, the dirty condition of the mouth often seen in young children, and so graphically described by Mr. Steadman, may be repeated four or five years after all the temporary teeth are gone.

Mr. J. G. TURNER (in reply to Mr. Dowsett): In a paper on the "Value of Teeth to the Human Economy," I said that from childhood to old age teeth were non-essential; foul teeth were the enemy, not want of teeth. This provoked an astonishing torrent of abuse, for which this evening's proceedings have made some amends. With the main thesis of the paper I must heartily agree. Till prevention comes into its own extraction will remain the chief means of treatment. There are not enough dentists in England to deal with the saveable teeth of school-children alone. When clinically investigating hypoplasia of teeth I came to the conclusion that dental sepsis in early childhood was in some cases the cause of hypoplasia of the permanent teeth. Unlike other tissues, the teeth are incapable of repair and show a permanent record of the injury caused by the prolonged sepsis. Karl Pearson, I believe, has shown that in cases of scarlet fever the cranial measurements are smaller than normal. The same reasoning can be applied to prolonged dental sepsis, and it thus becomes obvious that serious physical and mental disability may be caused thereby. In this connexion I suggest that myopia of school-age may be due to dental sepsis, the strain of the extra-ocular muscles in conveyance acting on a sclera weakened by malnutrition or actually inflamed by toxic poisons or true bacterial infection, causing it to stretch. A similar case is to be seen in flat-foot. I would also draw attention to the fissured tongue often seen in children. This seems sometimes to be congenital, but most often it is part of a general chronic septic stomatitis due to foul teeth.

Mr. GEORGE NORTHCROFT: Other speakers have dealt with Mr. Steadman's paper from the point of view of the method of dealing with certain forms of oral sepsis, with which most of us are in hearty agreement, and have been for years. There are, however, one or two points of detail that seem to need modification. For instance, the statement that dilaceration is commonest among premolars and is due to sepsis, as far as I know, lacks confirmation. Mr. Steadman seems to be inconsistent as to his views on the evil effects of what might be called minor sepsis and on the development of the jaws after the removal of the temporary teeth. He draws attention to the fact that gingivitis results from the accumulation of food round isolated temporary teeth, implying that general sepsis will result if these are left, and then says, following the President's practice, he leaves isolated $\underline{E\,|\,E}$ in place, with a view to holding back the $\underline{6\,|\,6}$. He also maintains that if the $\underline{6\,|\,6}$ and $\overline{6\,|\,6}$ are once in occlusion they do not move forward, whereas it is a well known fact that these teeth do alter in their antero-posterior relationship in the jaws during the growing period. In one portion of his paper Mr. Steadman says that the growth of the jaws will undoubtedly be affected and it will be necessary to remove permanent teeth, either the first permanent molars or the premolars, and in another place he says the normal growth of the upper jaw has apparently not been affected by the extraction of the teeth. You certainly cannot have it both ways. He also says that if unilateral extraction is resorted to normal development will still take place. Now, it is a well known fact that unilateral extraction causes deviation of the centre. If Mr. Steadman's powers of observation have not led him to see that a very large proportion of the population have asymmetrical faces in any case, it is scarcely to be expected that he would see the effect of asymmetrical extraction on the development of the jaws or the features; and, therefore, to make a claim that the effect of extraction does not produce a disharmony of the features, because it has not been observed by him, is a statement one cannot allow to pass unchallenged.

Mr. STEADMAN (in reply): Dr. Saunders has made a valuable contribution to the discussion. I am sure that I am expressing the feelings of members in stating how pleased we are to welcome members of other branches, especially when they add to our knowledge in the way that Dr. Saunders has done. Mr. Arthur Colyer appears on the whole to have been more fortunate in the medical men that he has met than I have been, but I wish to emphasize the fact that I have been careful to point out that my remarks refer to the minority only. In reply to Mr. J. Lewin Payne, I quite agree with him in the importance of teaching prophylaxis, but it should be carried out on the lines advocated by Dr. Sim Wallace, that of proper dietary—self-cleansing detergent food—rather than by toothbrushes. In the case of the poor, at any rate, toothbrushes often do more harm than good through their not being kept clean; for instance, it is not uncommon to find one toothbrush being used for a whole family. A toothbrush for each member of a large family costs money and is often an item of expense which cannot be met. In reply to Mr. Northcroft, the evidence for dilaceration and hypoplasia being frequently due to sepsis is to be found fully described in Mr. J. G. Turner's paper from which I was quoting. With regard to Mr. Northcroft's criticism about leaving the $\dfrac{E\,|\,E}{|}$ in position, I thought that I had made it clear that I did this only when the patient was aged about 6, that is to say, just before the eruption of the first permanent molars, and then merely during the time of their eruption, in order to hold them back. I consider this course to be the lesser of two evils.

I cannot accept Mr. Northcroft's statement that it is a well-known fact—or indeed that it is a fact at all—that teeth move forward to any great extent once they have occluded. Mr. Northcroft has not grasped my meaning about the growth of the jaws. I am convinced that the growth of the jaws is unaffected by the extraction of teeth. The reason that permanent teeth have sometimes to be extracted subsequent to the extraction of deciduous teeth owing to the resultant overcrowding, is not due to lack of growth of the jaws but to the forward movement of the first permanent molars, which I have admitted takes place in cases in which the second deciduous molars are extracted early before the eruption and occlusion of those teeth. In certain cases it is very much better to allow this forward movement than to leave in septic teeth. With regard to Mr. Northcroft's remarks about asymmetry, I am fully aware that asymmetry of the face is very common, this being due, however, not to extraction of teeth as stated by Mr. Northcroft, but to moulding at birth as pointed out by the President, as it is very common in persons who have never had any extractions performed. Mr. Northcroft has admitted that this asymmetry is so slight that it has to be carefully looked for in order to discover it. This being so, it is surely better to extract the septic teeth and give the child a chance of full development with a slight asymmetry (even if this were due to extraction), than to retain these teeth during the important years in which growth is taking place, to the lifelong injury to that growth, both mentally and physically. Here again it is the lesser of two evils.

Section of Odontology.

President—Sir FRANK COLYER, F.R.C.S., L.D.S.

Case illustrating the Need for Conservative Treatment of Bone Fragments in Compound Fractures of the Mandible.[1]

By GILBERT CHUBB, F.R.C.S.

THE patient had a very extensive wound laying bare the mandible from the middle of the ascending ramus almost to the symphysis; the mental foramen being clearly visible. There was a double fracture of the mandible; posteriorly just above the angle, and anteriorly in the canine region. The loose fragment carried two molars and one bicuspid, all of which were clearly visible in the wound: the latter, of course, opening widely into the mouth (fig. 1). The loose fragment was supported in correct occlusion by dental splints, and subsequent radiograms show it firmly united at either end with the teeth in good position (fig. 2). During the process of union the fragment threw off a superficial flake-like sequestrum, after which the wound was closed by a plastic operation which also closed the mouth cavity (fig. 3).

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[1] At a meeting of the Section, held February 23, 1920.

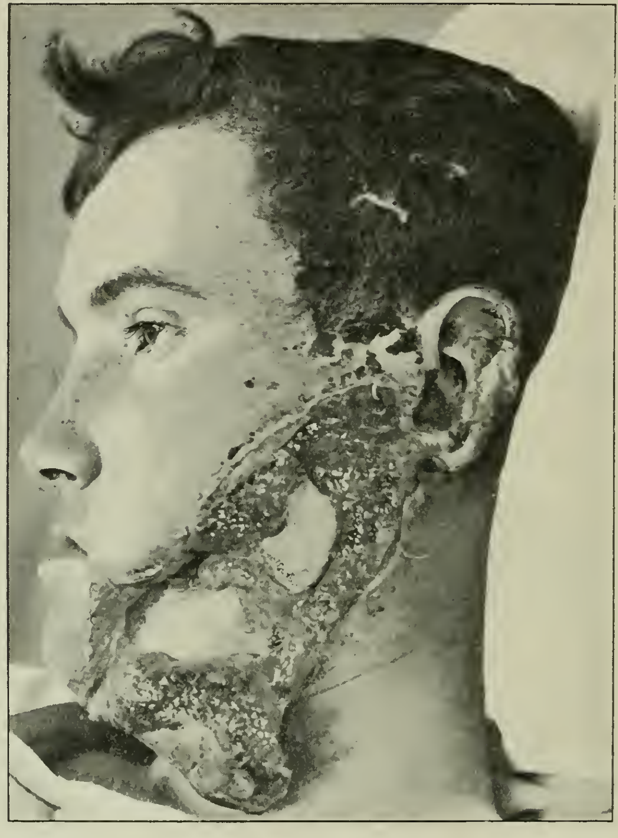

Fig. 1.

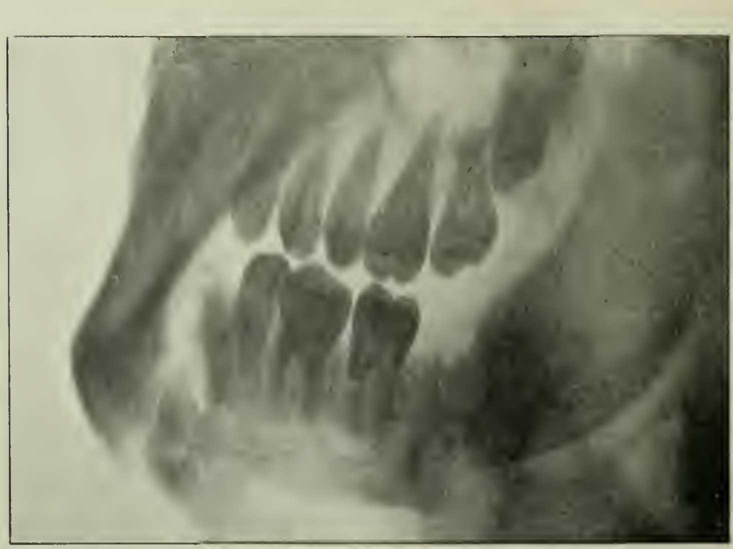

Fig. 2.

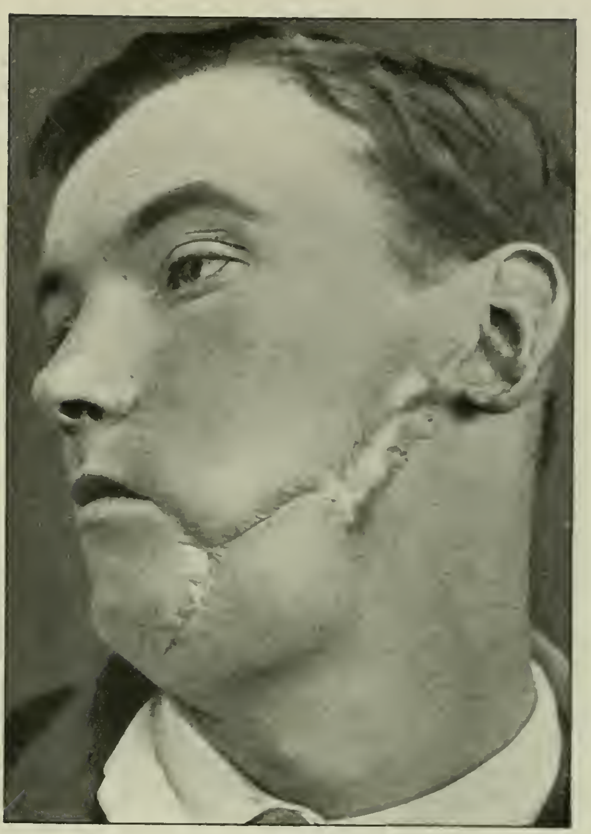

Fig. 3.

Case of Cystic Swellings in the Mouth.

By F. B. Bull, L.R.C.P., M.R.C.S., L.D.S.

E. D., aged 11. The patient was sent to Guy's Hospital from the dental surgeon at the Popham Road Treatment Centre, Islington. The present condition shows the presence of four cystic swellings in the mouth, situated one in each of the temporary molar regions in both upper and lower jaws. The second temporary molar teeth are in good condition, but the condition of the first temporary molar teeth is as follows: T.M. 1 one root present, T.M. 1 one root present, T.M. 1 carious, T.M. 1 carious.

The swellings are about the size of a walnut, and the outer walls of those in the lower right and upper left regions appear to be broken down. There is a history of twenty-four months from the time when they were first noticed, and eighteen months ago the mother was advised to have the condition attended to, but as the boy was suffering from shingles at the time she took no steps in the matter. He has had no pain.

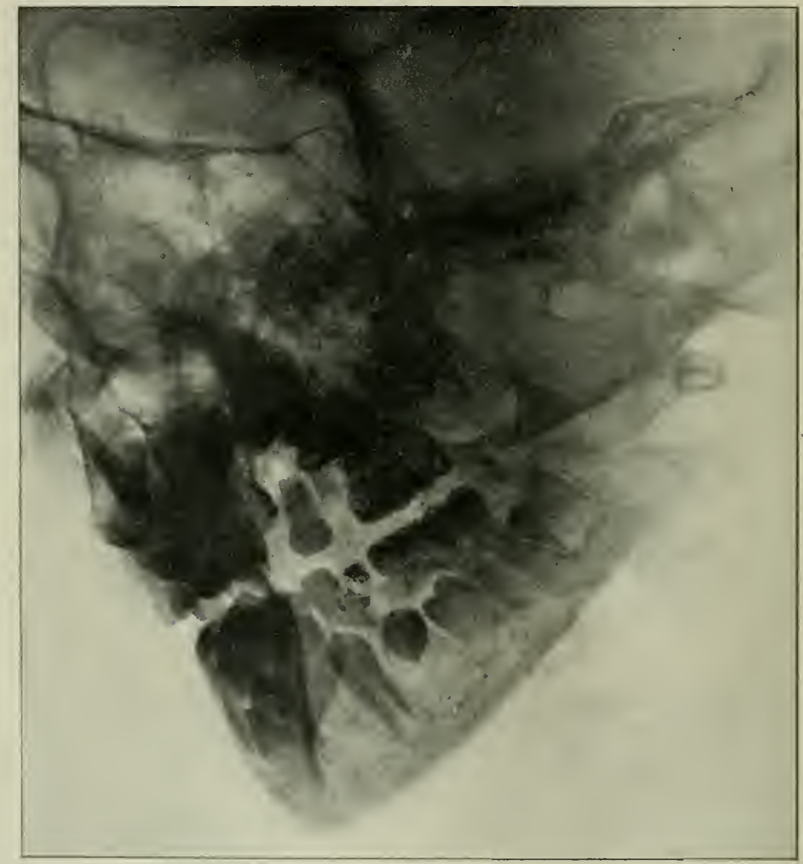

Fig. 1.

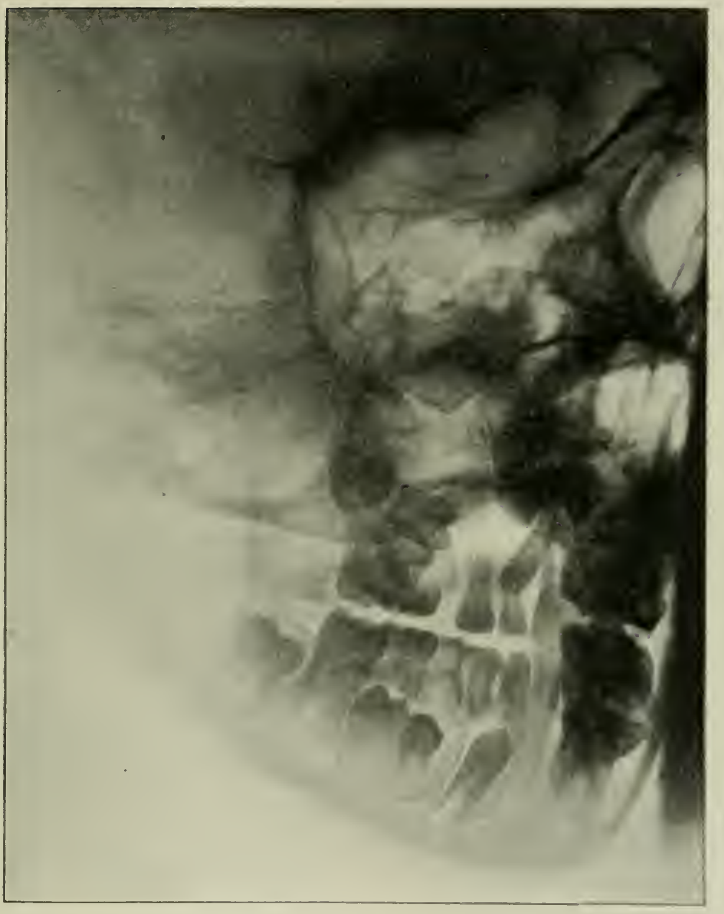

FIG. 2.

Section of Odontology.

President—Sir FRANK COLYER, F.R.C.S., L.D.S.

Chronic Infective Osteitis of Maxillæ in a Male, aged 40.[1]

By H. WATSON TURNER.

THE man was shown at the last clinical meeting of the Odontological Section. The history of the case is: Twelve years ago enlargement of the right maxilla was noticed in the molar region, and has since been increasing gradually along the whole alveolar region of both maxillæ. In May, 1919, he was sent up to the Middlesex Hospital under the care of Mr. Gordon Taylor. On examination, though there was huge hypertrophy of the maxillæ, it did not appear to pass beyond the suture line and to be entirely confined to the alveolar region. It is impossible to definitely say in any case how far enlargement has spread, except post mortem in the macerated specimen.

Mr. Gordon Taylor asked me to see and to take charge of the case as a likely one of dental origin. I have here a photograph of the patient when I first saw him (fig. 1, *a*, *b*), and also an impression of his maxillæ. The lips and cheeks were so stretched I could not get an impression of the entire growth. The teeth had deep pockets in places and were somewhat loose. I extracted all his teeth under a general anæsthetic and decided to wait six months and see what result followed. The man returned to hospital on February 23, 1920, and showed marked improvement in appearance, the lips and cheeks being much less stretched and lines of the face more marked. I took fresh photographs and impressions. The photograph I think shows the improvement (fig. 2, *a*, *b*).

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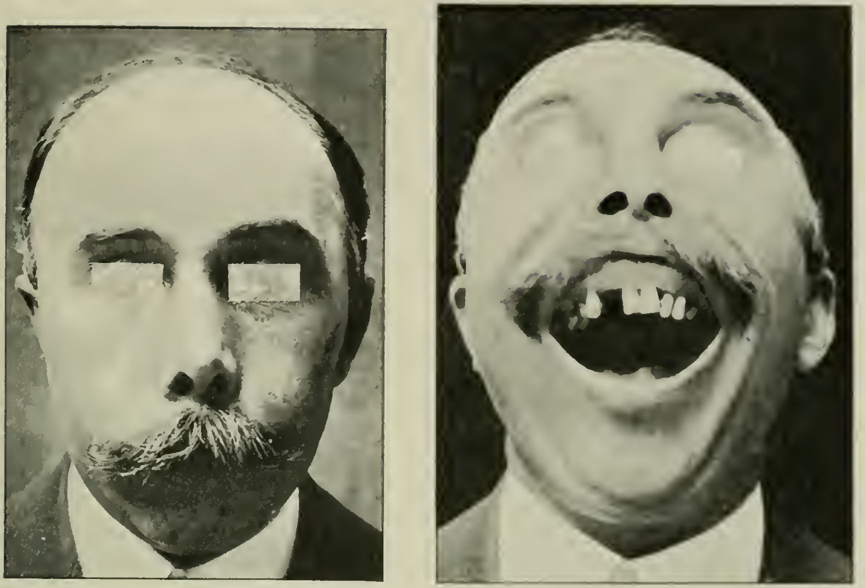
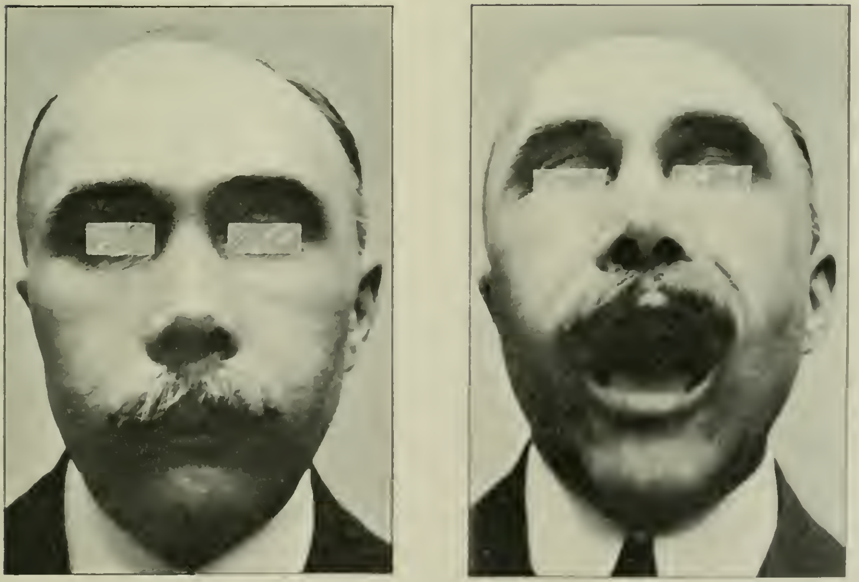
[1] At a meeting of the Section, held March 22, 1920.



a *b*

Fig. 1.



a *b*

Fig. 2.

I decided to remove as much of the growth as I could. Before doing this I brought the man to the last clinical meeting of this Section. On March 11 I operated and removed large quantities of the bone. Photographs and impression taken since the operation show his present appearance (fig. 3, *a*, *b*). The bone was friable and granular to touch and easily cut. Transillumination showed no suborbital illumination. Mr. Charles Clark very kindly took X-ray photographs for me which show the antra to be completely obliterated. The nasal passages are clear and also the orbits. There has never been any pain or neuralgic trouble or trouble with nasal breathing. No epiphora nor eye trouble. No other bony enlargements are to be found in other parts of the body. The only trouble beyond the growth has been depression, which is now gone, this probably being mostly mental.

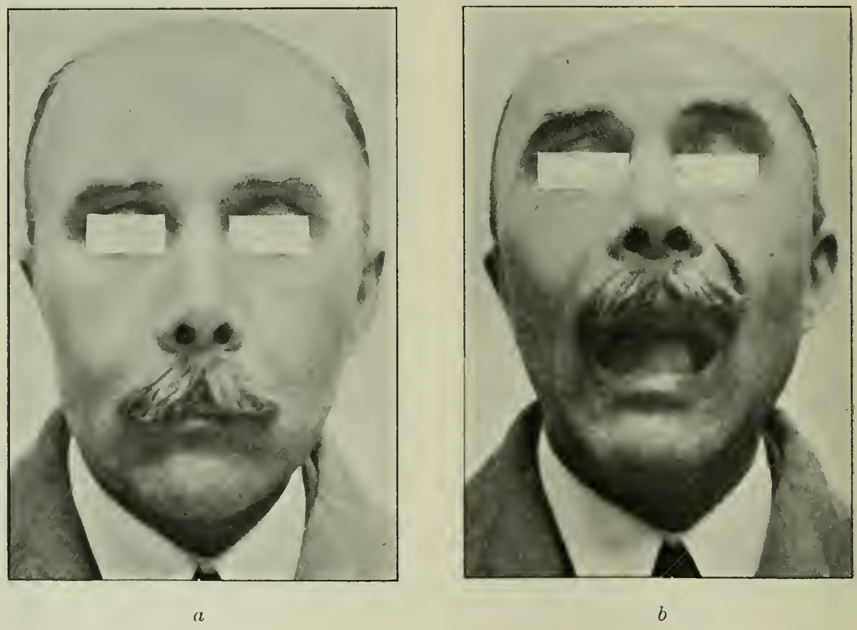

a *b*

Fig. 3.

Dr. Drew very kindly prepared and cut sections of the bone for me. He reports: "It appears to be an inflammatory hyperplasia; there is a good deal of bone absorption in parts. No bacteria could be demonstrated, either by Gram, the ordinary stains, nor with Nile blue."

Two sections are shown and were cut without decalcifying, the bone being soft and crumbly. In the one—a paraffin section—bone cells, shrunken away from their surroundings, are well shown. For the same reason, shrinkage, the myeloid cells are shown but shrunken. In the other section, a frozen one, there being no shrinkage in preparation, multinucleated myeloid cells and formative cells are well seen.

I hope in a year's time to again see the man and see the final result. Seeing the improvement resulting from the removal of the cause—the infection from the teeth—I hope for an entire arrest of the hypertrophy and ultimately a normal condition again.

Hemihyperplasia of Face, Jaws and Teeth.

By Montagu F. Hopson, L.D.S.Eng.

Examples of hemihyperplasia of the hard and soft tissues of the face, including the teeth and more rarely the tongue, are sufficiently uncommon to be worth recording. The condition would appear to be germinal inasmuch as it is observable at birth, and in this limited sense, inherited, but so far as I have been able to ascertain there is no evidence of transmission.

Christopher Heath, in his well-known Jacksonian Essay, describes a case of hyperostosis of the jaws with hypertrophy of the tissues of the corresponding side of the face, which had been under his notice for fourteen years. When first seen the boy was aged 12. The enlargement had been remarked on from the age of 3 months, the left side of the face being involved. Heath gives a figure showing the appearance of the lad when he first saw him. The left superior maxilla had shared in the deformity, being distinctly hypertrophied. A woodcut of a cast of the maxilla shows that the deciduous incisors and canine are retained on the affected side, although they have been replaced by the permanent teeth on the healthy side. The premolars on both sides have erupted the left first molar appears to be just erupting, but that on the right is not to be seen. There is no apparent difference in the size of the right and left premolars. Heath excised the affected maxilla " in the hope that the removal of the bone and the necessary incisions in the cheek would lead to a permanent relief of the deformity." Later an attempt was made to open the left eye and to destroy a portion of the tissue of the cheek " but without much permanent success." A figure is also given of a section of the excised maxilla showing the unerupted teeth embedded in the bone. Mr. Charles Tomes examined the specimen microscopically, and reported the bony structure as being remarkable on account of the absence of well-developed regular Haversian systems. "The bone is everywhere excavated by large irregular spaces, around which there is but little appearance of lamination; the lacunæ are arranged somewhat irregularly."

Another very similar case occurred in the practice of Richard Barwell at Charing Cross Hospital, and is recorded by him in the *Transactions of the Pathological Society*, vol. xxxii. The patient was a lad, aged 7, the right side of the face being affected. The corresponding half of the tongue was also involved. Barwell tied the common carotid artery, but the lad died from a secondary hæmorrhage. His skull is now in the museum at Charing Cross, and it shows that all the parts concerned in the function of mastication are hypertrophied. I would draw your attention, however, to the fact that the deciduous teeth on the affected side are retained.

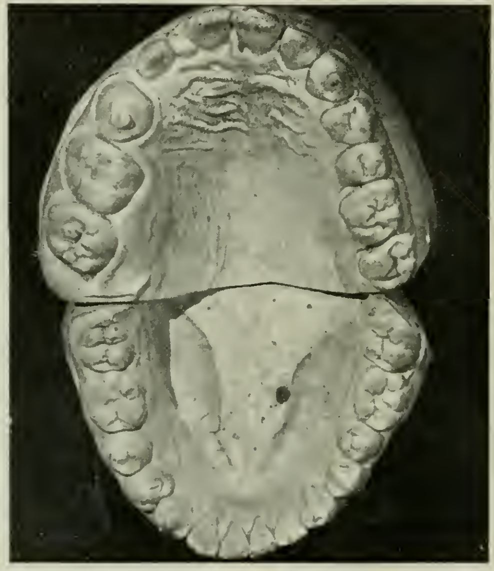


Fig. 1.

By the kindness of the President, Sir Frank Colyer, I am permitted to bring before you another example, not hitherto recorded, which was brought to his notice by Mr. H. O. Dickin. The patient is present so that you can examine him yourselves. He is not quite 17 years of age, and, as you see, it is the right side of his face which is so very hypertrophied. There is the usual hyperplasia of the soft parts, including the right half of the tongue. I would draw your attention to the fact that the frontal bone is also involved, so much so, that there is a very distinct ridge running vertically down the centre of his forehead, where the two bones articulate. The models of his mouth (fig. 1) show that there is considerable enlargement of the bone of the right maxilla. There is an enormous permanent canine, the first premolar and the second are missing, whilst the first molar is half as large again as its fellow of the opposite side; the second molar is larger than its fellow, but is smaller than the first molar. In the mandible we see some

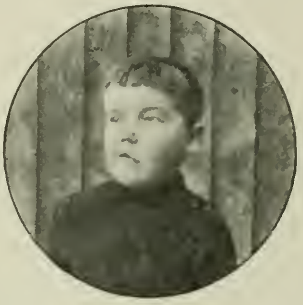


FIG. 2.

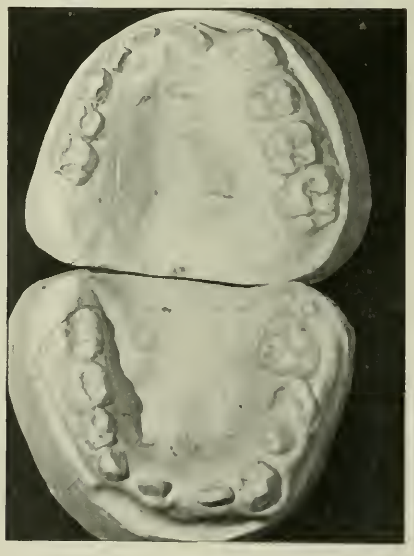


FIG. 3.

thickening of the bone. There is an exceptionally large canine, both the premolars are larger than those on the left, but there is not so marked a difference in the size of the molars.

Last autumn Dr. W. O. Roberts sent a small boy to my out-patients at Guy's Hospital. He is aged 7½. His parents and the other members of the family are normal, and so is the patient with the exception of the hemihyperplasia of the hard and soft tisues of his face on the left side. The condition was noticed at birth and has steadily increased. There was no difficulty about the mother's labour. The photograph (fig. 2) shows his facial appearance. From the models of his mouth (fig. 3) you will see that there is an even greater enlargement of the maxilla than in Sir Frank Colyer's case. The deciduous teeth are still in place on the healthy side, but on the left side he has already erupted an enormous permanent canine, a large first premolar and a very large first molar. A radiograph shows that the second premolar is in place over the retained second deciduous molar. In the mandible again we have the same permanent teeth erupted, and equally large, and here it is possible to compare the size of the first molars as that on the right has also erupted. There is a distinct overgrowth of the mandible on the affected side.

Case of Osteomyelitis of the Mandible.

By H. Stobie.

The following notes of a case will illustrate the enormous powers of regeneration the mandible has—an already well established fact.

I first saw the patient on July 26, 1916, when he gave me the following history: About May 18, 1916, an aching upper tooth was "extracted," but contrary to expectations the pain was not relieved, but increased, and his face began to swell. The swelling rapidly involved the left cheek and side of neck and on May 25, 1916, a week later, "some lower teeth" were extracted; still the swelling increased and by May 29, 1916, had spread under the chin. An abscess pointing in the mid-line in this region was opened and pus evacuated with no improvement, and three days later the swelling had spread across the mid-line and involved the side of neck and face. On June 7, 1916, an abscess in the right cheek was incised and on June 28, 1916, openings were made behind the right and left angles of the mandible and drainage tubes inserted. No bone was removed on any occasion but several teeth "fell out" at different periods. I could obtain no history of any previous disease or habits usually associated with necrosis, which could be assigned as a cause of the trouble.

This history covers a period of roughly nine weeks, and on examination I found his appearance was suggestive of diffuse lipomatosis. The face and neck were considerably swollen and œdematous, sinuses were present behind the right and left angles of the mandible, in the right cheek, two in the sublingual region, all discharging copious quantities of thick yellow offensive smelling pus. There was a considerable degree of anæmia and the patient's general condition was poor. On examining inside the mouth, the superior maxillæ were well formed, all the teeth were present excepting the $\underline{\ \ 4}$, which had been extracted on May 18, 1916. The teeth were free from caries, and the gum healthy and presenting no evidence of periodontal disease. In the mandible were present $\overline{8\,7\;\;2\,1|1\qquad}$ and the left premolar-molar region was occupied by a large partially exposed sequestrum which was loosening. The teeth were firmly attached to bone which was itself, however, freely mobile and the whole was bathed in pus.

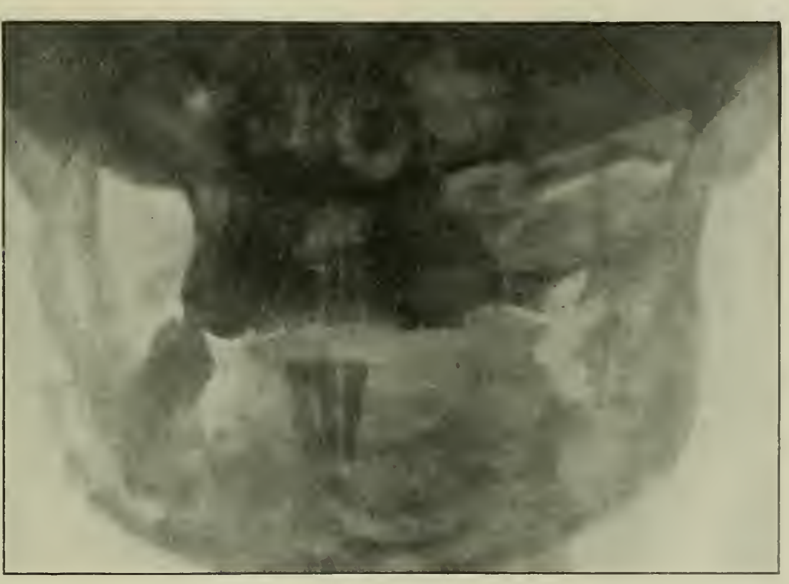


Fig. 1.

On July 27, 1916, an X-ray was taken and it was found (fig. 1) that the bone from the right to the left condyle presented a cribriform appearance suggestive of diffuse osteomyelitis.

Treatment was commenced immediately by having the inside of the mouth thoroughly cleansed by frequently syringing with ac. carbolic and liq. potassæ, and the external application of hot fomentations, three hourly. I did not consider the drainage already present sufficient, and the patient's general condition forbidding a general anæsthetic, on July 30, 1916, with ethyl chloride spray I dilated the sinuses at the angles with forceps, explored with my finger and introduced ½-in. drainage tubes. Hot fomentations and irrigation being continued.

Three months later, on October 5, 1916, the sequestrum inside the mouth, being quite separated, I removed it from inside but had some difficulty on account of its length and the fact that the posterior extremity "came first," for with the mouth widely open there was barely sufficient room between the maxillary and mandibular sulcus to rotate it and only by fracturing about ½-in. did I succeed (fig. 2, A). It proved on examination to be practically the whole of the outer portion of the left side of the body of the mandible from the symphysis to the angle. This removal was followed by marked improvement both local and general until November 17, 1916, when the left side of the face became much swollen and the discharge more copious. Bare bone could be felt at the bottom of the sinus at the left angle, and on X-ray examination it was shown that a large portion of the left ramus was separating but new bone was present on its inner aspect (fig. 3). This plate also shows the considerable amount of regeneration of bone

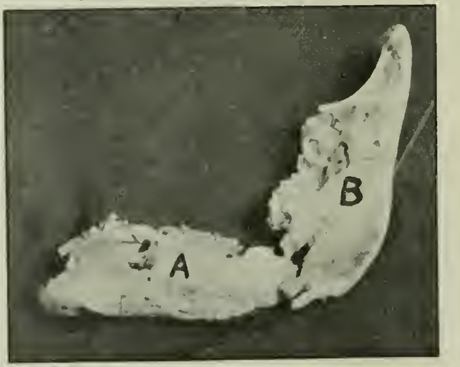


FIG. 2.

on the right side from which only some small sequestra were separated. The general condition of the patient was now so much improved by attempting to keep the sepsis under control, and a process of intensive feeding, that active interference was decided upon for its removal to relieve the symptoms, which were severe, and on December 12, 1916 (anæsthetic, C. and E. followed by $CHCl_3$ with Junker's inhaled), the sinus behind the left angle was extended upwards by an incision about 1½ in. long along the posterior border (fig. 5) of the ramus, and the sequestrum freed by blunt dissection from surrounding tissues and removed. This proved to be the left ramus from the condyle to the angle (fig. 2, B), with the exception of the coronoid process.

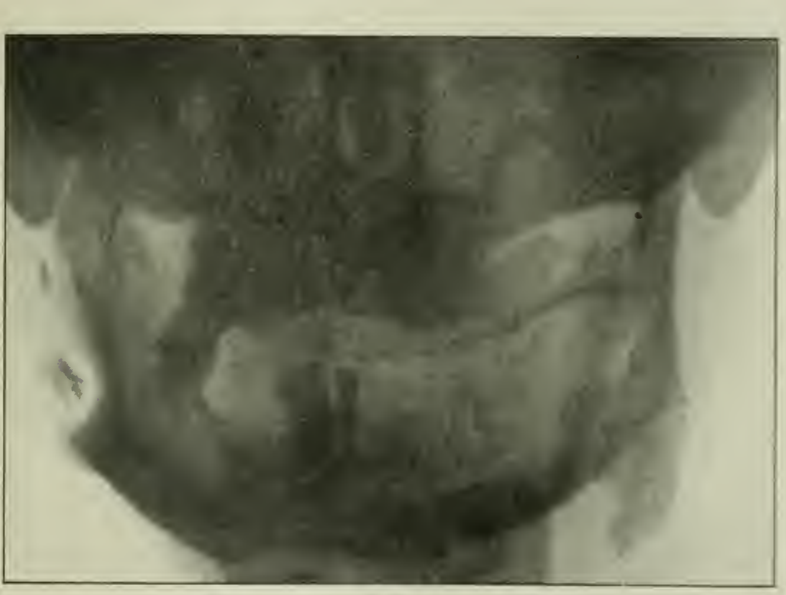


Fig. 3.

An X-ray taken the next day shows the condition following this removal (fig. 4). It will be seen that there is continuity from right to left sides; the quantity of bone being more perfect on the right than the left side, and certainly doubtful in the regions where mandibular teeth remain. No attempt was made during this period to remove these teeth for fear of injuring the tissues and retarding the regeneration which was going on, but their removal was felt to be desirable and improvement both local and general being continued, they were removed on February 12, 1917.

The process of regeneration already well established was distinctly improved by this measure, and an X-ray taken on February 27, 1917, shows this (fig. 5). By the end of March, 1917, all sinuses had completely healed; the movements of the jaw were normal, but naturally there was no regeneration of alveolar process and consequently no ridge. A weighted denture was inserted to restore the function of mastication, which was performed moderately well, the patient being able to eat soft food. An X-ray taken at this period shows the final condition, and comparing it with the initial state it will be seen that the bone, besides consisting of homogeneous tissue, is considerably narrower from side to side (figs. 6 and 1). The final appearance of the patient in July, 1917, is here shown (fig. 7), fourteen months from the onset of his disability.

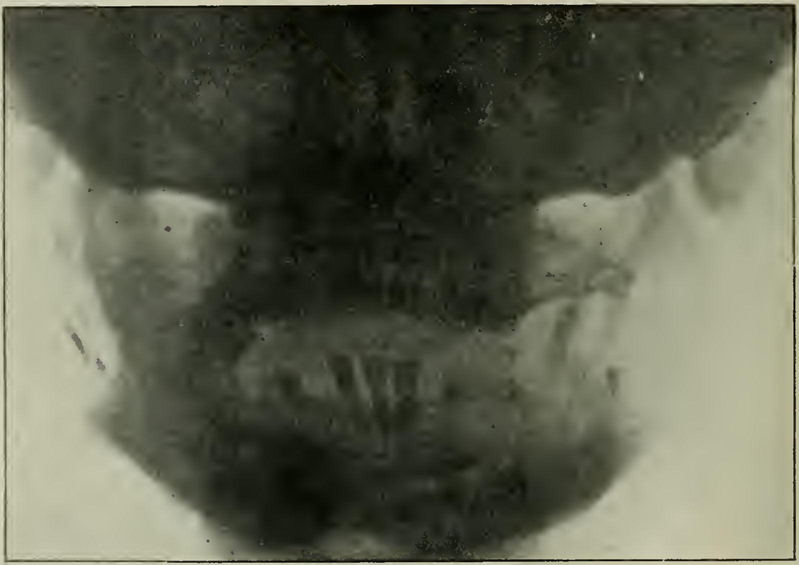


Fig. 4.

The history of this case is unsatisfactory, insomuch as it is difficult or impossible to assign the origin of the trouble to the superior maxillary tooth, and probably it would be correct to assume that there was infection from a lower left molar tooth, either via the pulp or periodontal membrane, and thence to the medulla, the pain being referred to the corresponding upper tooth. No bacteriological examination was made, but the causative organism must have been of virulent type.

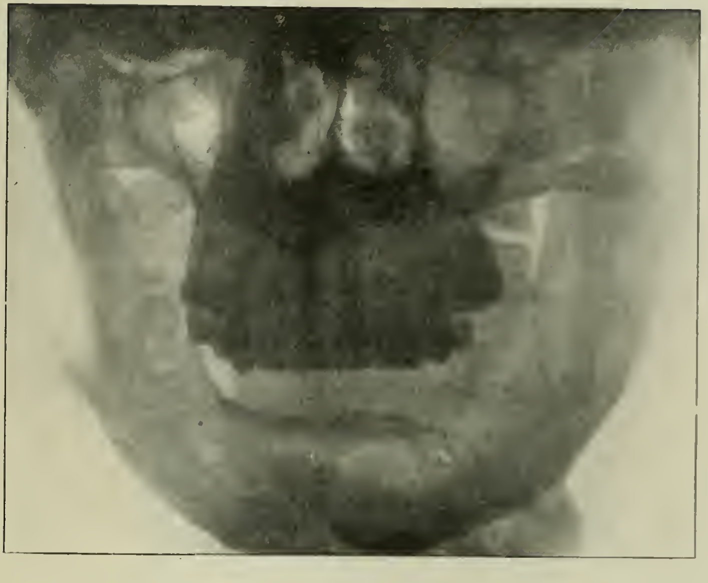


FIG. 5.

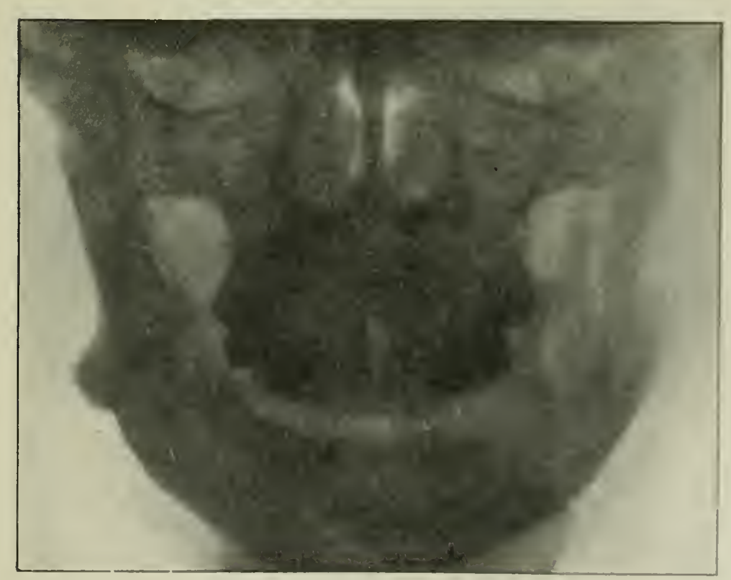


FIG. 6.

The progress made and the result obtained illustrates the value of patiently waiting for sequestra to be naturally separated in preference to immediate operation for their removal, although with the experience of some other cases, similar in nature but not so extensive, the process was arrested by the removal of sequestrum and teeth up to the limit of the portion affected. The general condition of the patient in this case decided the course to be taken, and it is more than likely earlier interference would have jeopardized the patient's chances of the complete recovery he made.

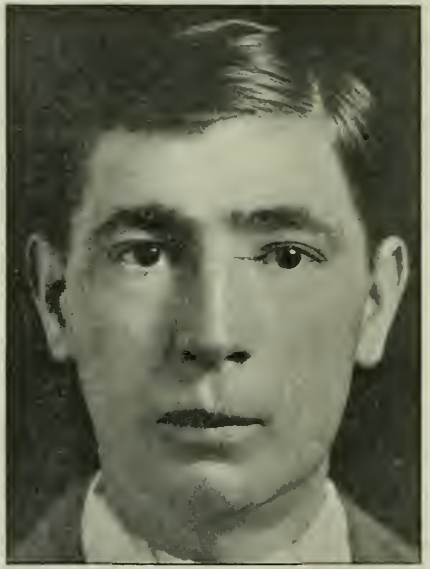


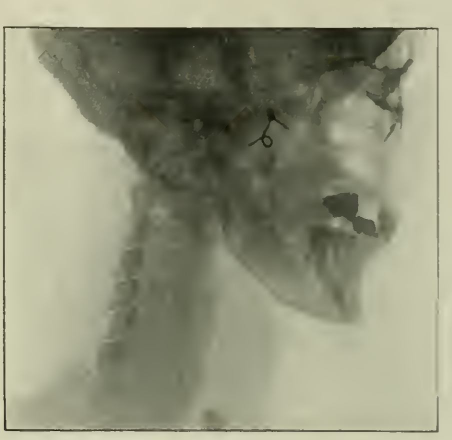
Fig. 7.

A Mandibular Third Molar situated in the Coronoid Process.

By Montagu F. Hopson, L.D.S.Eng.

The vagaries of mandibular third molars are both numerous and curious. My patient was a man aged over 60, whom Dr. Collingwood Andrews asked me to see. For twenty-eight years he had suffered with trouble in and about his left temporo-mandibular joint—recurrent attacks of pain, some swelling, with difficulty in opening his mouth.

The condition was attributed to arthritis, and once he was treated for mumps. Eventually, after a larger swelling than usual, a sinus appeared below the angle of the mandible and on its inner aspect. This sinus persisted and had been present some time when Dr. Andrews saw him. The patient was sent to Sir James Mackenzie Davidson for X-ray examination, and I show you the beautiful stereoscopic radiographs he secured. They show the third molar situated in the coronoid process, with its crown directed forwards and a large absorption cavity beneath the tooth, from which pus had tracked downwards beneath the internal pterygoid and found exit through the sinus I have mentioned. I made a futile attempt to reach the tooth from the inside, with the mouth widely opened and the coronoid process tilted forwards and downwards. Subsequently we sought the assistance of Mr. McAdam Eccles, and the tooth was removed from the outside. The tooth was much absorbed and eroded, thus showing that it had lain in a pus cavity for a very long time.



Mandibular third molar situated in the coronoid process.

Successful Case of Bone-grafting.

By Stanley Mummery.

The case which I am bringing before you this evening, presents, I think, two features of rather unusual interest. The first was an attack of tetanus, which nearly proved fatal; the second was the occurrence of prolonged sepsis after the graft had been inserted, which did not, however, prevent a completely successful result.

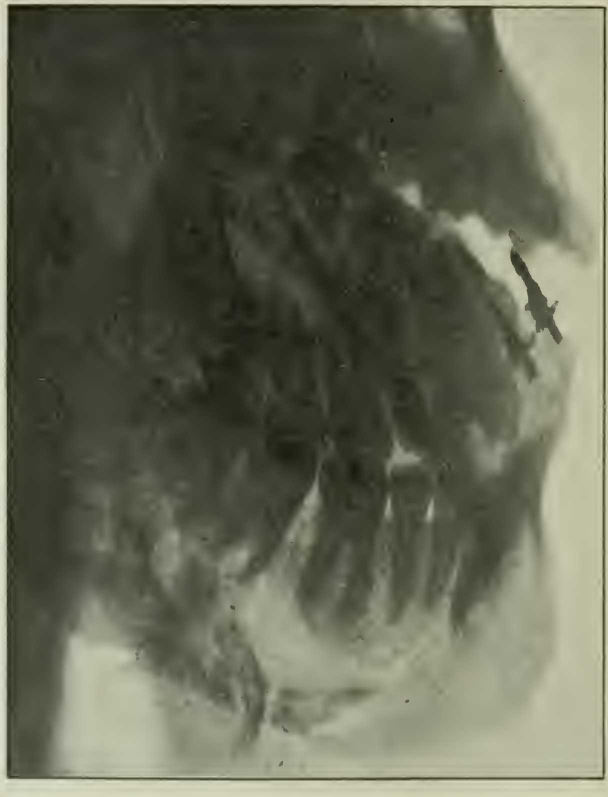


Fig. 1.

The patient, an officer in the Sixth Northumberland Fusiliers, was admitted to the Endsleigh Palace Hospital under me on September 14, 1916, with a severe shell wound of the face. Upon examination I found a large wound on the right side of the mandible, which would admit three fingers. The chin had dropped and receded, and the mouth was fixed open with the third molars only in occlusion. An X-ray examination (fig. 1) showed an extensive comminuted fracture on the right side from the canine back as far as the angle, with a fissure extending from this region up the ascending ramus nearly to the sigmoid notch. The neck of the condyle was also fractured.

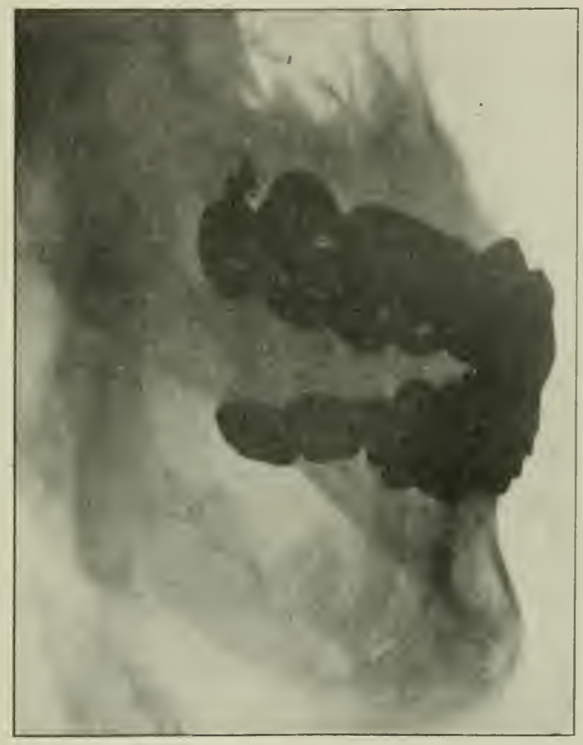


FIG. 2.

Within a few days of admission I made cast metal splints which were cemented to the teeth of upper and lower jaws, and by means of elastic traction the fractured mandible was brought into correct position in four or five days. The right second and third molars were involved in the fracture and were removed previously.

On October 9, just three weeks after admission, tetanic spasms developed in the muscles of the back. Chloral was administered, and Dr. Weston injected tetanus antitoxin. The tetanus, however, gradually developed and soon spread to the muscles of the neck and jaws. On the 19th the patient was delirious with a rapid pulse and obviously in a very serious condition. The splints became loose owing to the violent muscular spasms, and I removed them entirely. The patient remained in an extremely critical condition, and was delirious without intermission, for six weeks. He was under the care of Dr. James Taylor during this time who took a great interest in his case. Towards the end of November a marked improvement set in, and by the 27th of that month the patient was fully conscious and able to sit up. The mandible had during this time swung right round to the right, and the condition was naturally far worse than before. The splints were refixed, and before the end of December the mandible was drawn back into position again, when it was fixed by wiring the two splints together.

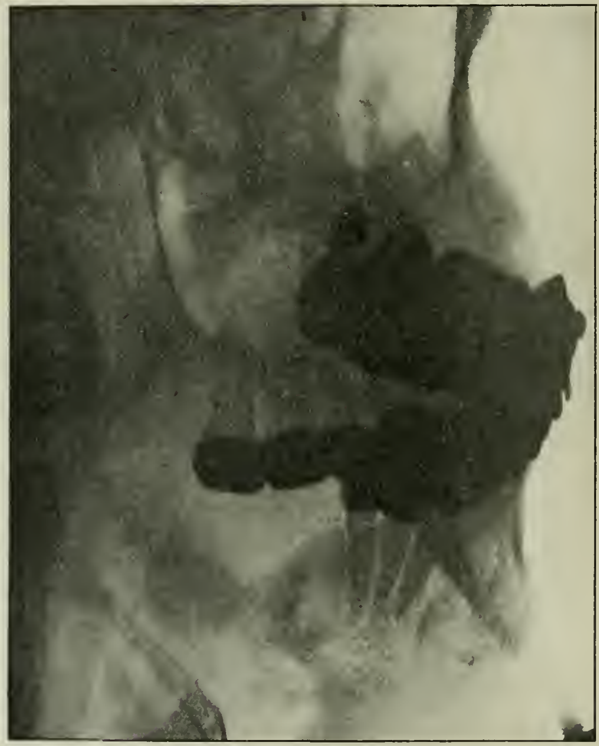


Fig. 3.

I may say that the patient had an unusually perfect set of teeth with very well developed cusps, and the articulation of the upper and lower teeth very materially assisted in immobilizing the mandible.} 'By the end of January all suppuration had ceased, and the condition was very satisfactory with the exception of the large missing portion of the mandible on the right side. New metal splints of a slightly different design, with studs soldered at intervals, were now substituted for the old ones, and the upper and lower firmly wired together preparatory to an operation for grafting. This was performed towards the end of April by Mr. Walton. A rib graft was inserted, mortised at each end and firmly fixed by pegs cut from the same rib (fig. 2).

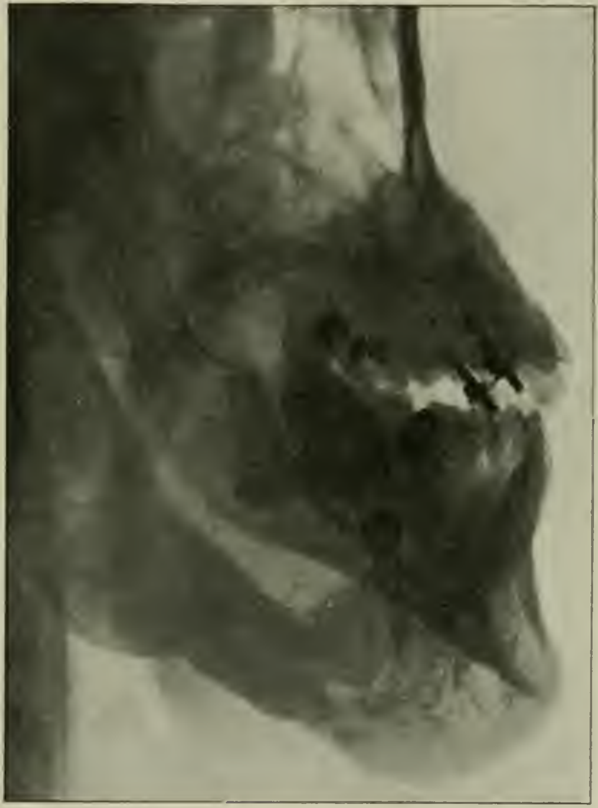


FIG. 4.

All went well until twelve days after the operation, when there was a sudden rise in temperature and considerable swelling of the face on the affected side. The next day a quantity of pus discharged through the operation wound and also into the mouth, while the swelling extended behind the ear and up into the temporal region. At the end of a week the discharge ceased, the swelling subsided and the temperature fell to normal.

On June 5 the swelling and discharge recurred, the pus escaping opposite the anterior end of the graft; a sinus became established at this spot, which communicated with another sinus inside the mouth below the first molar (fig. 3, p. 80).

On July 16, about eleven weeks after the operation, I unwired the splints and tested the union, which appeared quite firm, although the internal and external sinuses still persisted. The movement at the right temporo-mandibular articulation was also very good, which was hardly expected, as the neck had been fractured and the joint immobilized for over six months. I therefore removed the splints entirely and encouraged gentle movements of the mandible. The sinuses were treated by daily syringing with peroxide of hydrogen and afterwards swabbed out with 40 per cent. lactic acid.

It was not until the end of September, exactly a year after admission, that the sinuses finally healed up, and the patient was discharged from the hospital on October 7 with a fully efficient jaw which opened to a normal extent and possessed excellent masticating power (fig. 4).

The patient has kindly consented to be present this evening, and I will ask any of you who are interested to examine him.

Case of Metallic Mercury in the Region of the Right Mandible.

By H. A. Eccles, M.D.

The case I wish to bring before your notice is that of a young woman, aged 22, a domestic servant. The history as gathered from the patient herself is as follows: Pain was present in the right premolar region of the mandible for about eighteen months. At the end of this period she went to a dentist, who was said to be unqualified. He extracted the tooth after having pricked the gums several times. The pain was not deadened at all by the injection. About a week after the extraction of the tooth, she noticed some tiny pieces of "quicksilver" on her tongue and teeth. Three months later a swelling formed on the right cheek. A doctor was then called in, who opened the swelling on several occasions, metallic mercury in some quantity exuding instead of pure pus. The doctor states that she was at that time suffering from symptoms of mercurial poisoning, salivation, spongy gums, swelling of the tongue, colic and diarrhœa.

Eventually a sinus formed in the region of the centre of the body of the right mandible, which persistently discharged minute particles of mercury.

She first came under my notice some nine months after the extraction of the tooth. I then made the two skiagrams I now show. She was suffering from stomatitis even at this period. I sent a specimen of the discharge to Dr. Hurtley, the lecturer on chemistry at St. Bartholomew's Hospital, who reported as follows: "The particles on the dressings were removed. After cleaning, the separate particles adhered to one globule. Parts of this globule gave (1) a characteristic sublimate with iodine of mercuric iodide; (2) on solution in nitric acid and after replacement of the nitric by hydrochloric acid the following characteristic tests for mercury: (1) Yellow precipitate, with caustic soda; (2) scarlet precipitate, with potassium iodide; (3) white to grey precipitate with stannous chloride. There appears to be, therefore, evidence that the particles are metallic mercury and not an amalgam."

I then carried out the following simple experiments:—

(1) I injected a piece of meat with metallic mercury by means of an ordinary dental syringe, and made a skiagram. This showed the mercury divided into small particles exactly as seen in the skiagram taken of the patient. It is, therefore, possible to inject mercury with a dental syringe, and the appearances produced radiographically are similar to those seen in the case now described.

(2) I made a skiagram of a piece of lint on which I had spread a thick layer of unguentum hydrargyri B.P. containing 30 per cent. of metallic mercury. This showed a few shadows due to the presence of the metallic particles. But how many of these can be induced to pass through the skin by inunction?

By what channel, then, did these particles reach the patient's tissues, and by whom were they inserted?

Two channels would at once suggest themselves, namely, the mouth and the skin. Entry through the stomach and bowel may be put out of court, as it would not seem possible for a heavy metal to ascend into the region of the jaw. It would be very difficult, if not impossible, to insert so large a number of particles by inunction. Injection by means of a syringe seems to be the most probable solution of the problem. If this is correct, was the mercury self-injected, or is it possible that it was injected by mistake instead of cocaine or an allied substance?

The case appears to be of interest on account of its rarity, and of the problems it suggests. The question of treatment, also, is one of interest. Merely allowing the particles to discharge themselves through the sinus did not appear to be satisfactory. At the suggestion of a colleague of mine at the First London General Hospital, removal of the mercury by means of a gold probe was tried with, I think, some measure of success. My subsequent skiagrams showed the particles to be becoming less numerous, but when I lost sight of the case, there were still some remaining, and the sinus was still open.

Three Cases of Misplaced and Unerupted Teeth.

By FRANK COLEMAN.

Case I.—The patient, a man, aged about 40, was admitted to the Royal Dental Hospital on December 18, 1919, complaining of a swelling on the right side of the upper jaw. History: On January 14, 1917, the man received a through-and-through rifle bullet wound on the right side of his face, the entrance and exit wounds being respectively at the infra-orbital foramen and angle of the jaw; the bullet traversing the antrum between those points. Condition on admission: On the outer side of the alveolus in the right premolar region there was an inflamed, tender swelling, the size of a walnut, discharging pus through several sinuses. Through these sinuses a hard substance could be felt (? bone or tooth substance). A radiograph (fig. 1) showed the swelling to contain a buried canine tooth lying horizontally with its crown directed forward. On January 1, 1920, nitrous oxide was administered by the nasal method, the tooth defined and removed and the cavity scraped out. A portion of the crown of the tooth was missing, it having been carried away by the bullet traversing the jaw.

Case II.—The patient, a middle-aged man, was admitted to the Royal Dental Hospital on December 5, 1919, complaining of tenderness and swelling on the left side of his jaw. Condition on admission: On the left side of the maxilla in the premolar region there was a hard rough substance, the size of a dried pea, resembling bare bone, and surrounded by a ring of granulation tissue. A radiograph (fig. 2, p. 86), taken on the following day, showed a second premolar lying obliquely (with its crown directed upwards and forwards) in the substance of the jaw. The bare bone presenting in the mouth was a flattened plaque on the surface of the root of the tooth due probably to a previous periostitis. The tooth was extracted under nitrous oxide a week later, the forceps being applied to the apical portion of its root, which was distinctly bent on itself, suggesting past trauma and possibly a factor in its misplacement.

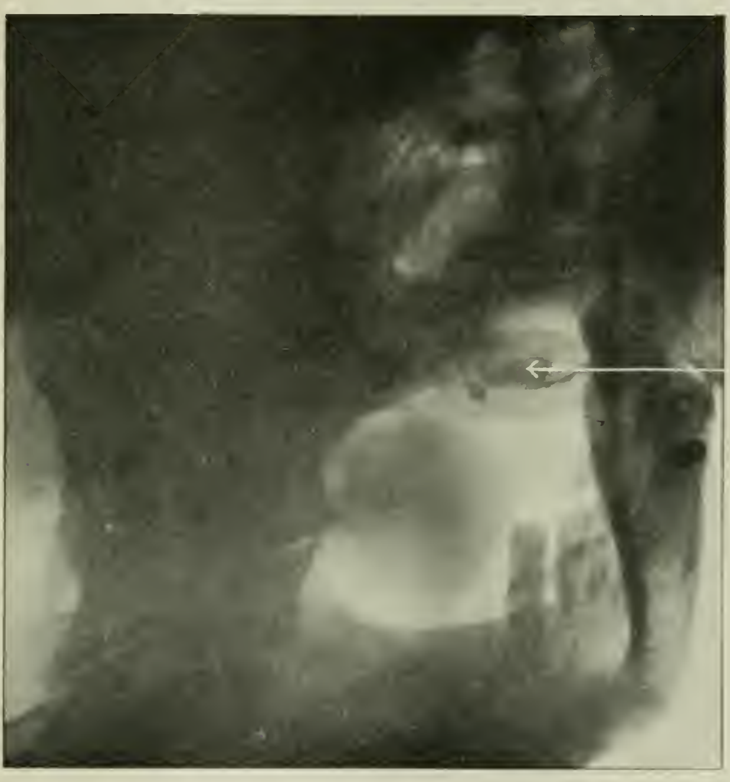


FIG. 1.

Showing embedded canine tooth (lying to left of condyle of jaw in skiagram).

Case III.—I saw this patient, a lady, aged about 50, early in January of this year. She was complaining of a good deal of discomfort in the lower left wisdom tooth region, discomfort which was increased by the wearing of a denture. A radiograph (fig. 3, p. 86) had already been taken which showed a premolar lying almost horizontally in the jaw with its crown abutting against the base of the coronoid process. The third molar had been removed seven years previously. The tooth was extracted under ether, but only after the removal of the bone covering it, as the position and direction of the tooth (against the coronoid process) would not permit of extractive movements in its axis.

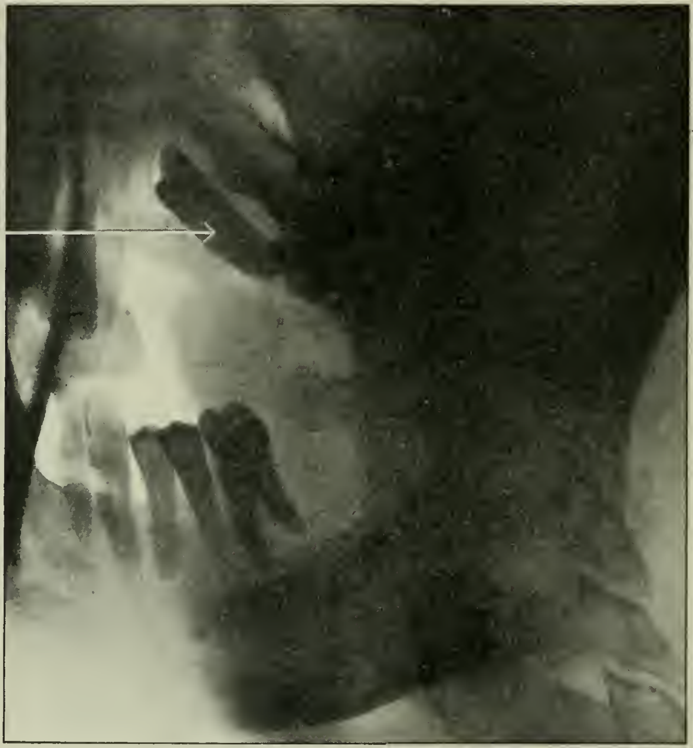


FIG. 2.

Shows maxillary premolar tooth (above mandibular molar in skiagram).

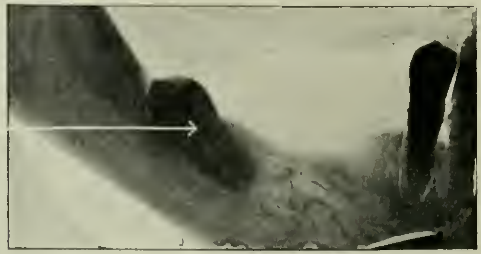


FIG. 3.

Showing mandibular premolar.

Case of Unerupted Premolar.

By E. Lloyd-Williams.

Patient, a lady, aged about 45, who had suffered for some time from a persistent sinus presenting over the apex of the left upper lateral incisor, which it was intended to crown. All forms of treatment having failed, an X-ray photograph was taken, when a missing second premolar was discovered with its crown lying on the apex of the lateral incisor. This is the first time that I have found a premolar in this position. The lateral incisor has been extracted, with the hope that an effort at eruption will make the removal of the premolar a simpler operation, with less disfigurement to the patient.

Traumatic Opening of Left Maxillary Antrum.

By E. Lloyd-Williams.

This case is one of traumatic opening of the left maxillary antrum, after an attempt to extract a first premolar from a lady aged 60. The human antrum is very variable in size, as was shown by the late Mr. Arthur Underwood some time ago in a paper read before this Section. In this instance the cavity was particularly large and easily entered by any ordinary attempt at tooth extraction. The interest of this case lies, not in my lack of skill in extracting the tooth, but in the resultant condition of an open antrum, and the question whether it were wiser to open up thoroughly and drain this cavity or leave the treatment to Nature. I decided on the latter course, and with good results. The fractured root was removed and all trouble cleared up. Incidentally, it may be of interest to relate that when this patient last visited me, on an attempt being made to wash out the mouth after the completion of two simple fillings, we discovered that the mandible was completely dislocated unilaterally. Repeated attempts by myself and a medical *confrère* to reduce the dislocation failed utterly. Under full chloroform anæsthesia the jaw was easily put into place. It is, perhaps, worth noting that I found the easiest way of reducing the dislocation was to close the mouth partially and exert firm pressure, directly downwards, at a point immediately over the angle of the mandible.

The following communication has also been made before the Section:—

> "On the Structure of the Dental Follicle and its Relation to Pathological Conditions." By J. HOWARD MUMMERY, D.Sc.(Hon.), M.R.C.S., L.D.S.Eng.

The above paper is published *in extenso, with illustrations*, in the *British Dental Journal*, January 1 and 15, 1920.

Section of Odontology.

President—Sir FRANK COLYER, F.R.C.S., L.D.S.

A Factor in the Pathology of Periodontal Disease.[1]

By GERALD B. ASH.

THERE is a factor in periodontal disease which apparently has been largely overlooked. To my mind it is of paramount importance, and that is why I wish to bring it to your notice that the subject may be aired and its importance gauged.

In a recent Editorial in the *British Dental Journal* the phrase was used: "Hypothesis preceding research." I must confess that my contribution this evening falls within that category.

I wish to draw attention to the peculiar significance of the oral epithelium. Most writers on periodontal disease after recognizing the initial gingivitis pass on to consider the injury to the periodontal membrane, which is a sub-epithelial tissue, and in no text-book nor in any treatise on the subject that I know of, with one exception, can I find mention of the epithelium, which must first be affected. The exception occurs in Hopewell-Smith's "Dental Histology and Pathology," where he does mention ulceration of the surface of the gum with destruction of the epithelium.

It is a fact of great physiological importance that the body has a complete covering of epithelial tissue, and that whatever is absorbed by the body must pass through this tissue. This covering is absolutely complete—externally by the epidermis and internally by the various forms of mucous membrane from the lip to the anus. There is no exception to this, and whatever apparently protrudes from it is simply an adaptation of the epithelium itself—eye, nails, hair, crowns of the teeth.

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[1] At a meeting of the Section, held April 26, 1920.

It has been termed "the first line of defence," and exercises a power of discrimination in allowing substances through it. Take, for example, the bowel: Although normally it teams with septic organisms, septic absorption only takes place in the condition of stasis when the accumulation of septic matter overcomes the resistance. You can plunge your hand into septic matter and suffer no harm unless there is an abrasion of the epidermis. On the other hand no wound is healed until the epithelium has grown over it, and the epithelial continuity is restored.

Now, the teeth are no exception to this rule of an epithelial covering. We have seen that that portion of the tooth which projects above the gum margin is epithelial in origin, and by analogy with all other dermal appendages I maintain that the connexion between the epithelium of the gingival margin and the crown of the tooth must be absolutely continuous and complete.

I am afraid I cannot demonstrate this continuity, and I am not aware that it has ever been demonstrated. I have made attempts by the Koch-Weill process but without success. But it should not be impossible if only there were better opportunities and means of research. However, there can be no real doubt about it, and it is not necessary to go very deeply into the histology of the developing tooth to show how it is brought about by Nasmyth's membrane.

Bödecker considers that Nasmyth's membrane is in direct union with the outermost epithelial layer of the gum. Mummery has shown the extensive distribution of epithelial cells in the follicle. Three points in his paper on the structure of the follicle which bear on this subject are: (1) The connexion between the surface oral epithelium and the enamel organ is never lost; (2) in addition to the sheath of Hertwig there is an enveloping layer of epithelial cells which is an extension of the enamel organ; (3) the stellate reticulum atrophies very early, and the external and internal epithelium of the enamel organ lie together. He considers the cellular layer of Nasmyth's membrane to consist of the external and internal epithelium and stellate reticulum. To put these points another way, the enamel organ persists as Nasmyth's membrane, and is in connexion with the oral epithelium on the one hand and with Mummery's enveloping sheath on the other.

Thus the connexion between the surface oral epithelium and Nasmyth's membrane is apparent. We can now see the continuous connexion between the oral epithelium and the dermal appendage in the form of the tooth—viz., the cellular layer of Nasmyth's membrane. It is possible that at the actual point of union at the neck of the tooth there is a thickening of the epithelial layer caused by the remains of Mummery's enveloping layer of the follicle and the sheath of Hertwig.

Bearing in mind the function of the epithelial tissue as the first line of defence, we can appreciate the significance of this connexion at the neck of the tooth. It is obviously a very weak connexion because, Nasmyth's membrane having no blood supply in itself, when a break has occurred there is no living epithelial tissue for the gingival epithelium to unite with. This is evidently the one spot in the whole body where a break in the epithelium, when it has once occurred, must be permanent, and can never be closed over so long as the tooth is in position. It is only by the removal of the tooth and the granulation of the socket that the epithelium can spread across and once more become continuous.

By saying that Nasmyth's membrane has no blood supply, I mean that as far as we know it has *no* means of obtaining nourishment, and, whatever the conditions at the point of union with the oral epithelium, the cellular layer as a whole is dead, and the renewal of cells is impossible.

We must also appreciate that there is some protection afforded by the position of the point of union—viz., in a sulcus under the slight bulge of enamel at the neck of the tooth. This bulge is only slight in man, except in the milk dentition, but it is very pronounced in the carnivora, where the gum is more liable to damage from splinters of bone, &c.

I need hardly go into the causes that would bring about this break; they are only too well known. It is sufficient to point out that an acute gingivitis alone would be sufficient to cause the death of the cells on the borderline between the living oral epithelium and the dead Nasmyth's membrane. But the break having once occurred it is very necessary to recognize that we have now a quite peculiar condition which cannot occur anywhere else in the whole body. We have now a lesion that is analogous to a wound and absorption can now take place. The best that can happen is that scar tissue may form and contract tightly round the neck of the tooth as fibrous tissue. But no wound is healed until epithelium has grown over the surface and become continuous, and at the best the joint can no longer be called "tight" against absorption.

When it is considered how large the area of absorption is when the condition is general or a large number of teeth is affected, what ideal conditions exist in the mouth for the growth of pathogenic organisms, and, further, that absorption is taking place into cancellous bone, richly supplied with thin-walled capillaries, is it to be wondered at that marked general symptoms of septic absorption are often present, and that many ailments, the origin of which is obscure, improve with the treatment of the septic foci in the mouth? On the other hand, when it is considered from the weak nature of the epithelial connexion that probably everyone sooner or later must have this break of the continuity of the epithelium in his mouth, and when it is recognized that numerous people have a very obvious and often advanced periodontitis and are apparently none the worse for it, it may be asked why these people do not suffer from septic absorption. The answer is that it depends largely on their power of resistance and the virulence of the organism present. The first line of defence, the epithelium, having broken down the patient is thrown back on to the second line, the blood stream. When there is a good local reaction and the gingival margin recedes there is good drainage and the septic foci are reduced to a minimum. But even where the local reaction is poor and the recognized symptoms of marked periodontal disease are present in the mouth, the resistance of the blood may still be tuned up sufficiently to cope with the absorption, so that no serious or inconvenient general symptoms are present. There comes, however, a time when the general resistance is lowered, perhaps by advancing age or some illness, then the balance is disturbed, probably never to be recovered, and whether the condition in the mouth becomes worse or not, general symptoms appear, and in the variable forms that might be expected from an infection carried in the blood-stream.

This is the peculiar significance of periodontal disease that here is a unique condition which is peculiar to the mouth and impossible elsewhere in the body, in which there is a permanent break in the epithelial covering so that septic absorption is taking place incessantly and has to be dealt with by the blood-stream.

I do not propose to go into the question of treatment, but a remark above about the removal of the tooth might lead you to think that I am advocating extraction in every case. It was only intended to show that short of the extraction of the teeth it is foolish to speak of a *cure* for periodontal disease. The primary condition, the break of the epithelial connexion, is permanent, and even after successful local treatment, there is a potential pocket and the local symptoms may recur.

It has not required the experience of this war to teach us the value of drainage in surgical cases. Even when there is a large area where absorption is taking place the provision of efficient drainage will reduce pyrexia and remove other symptoms of absorption. Similarly in periodontal disease local treatment which aims at the reduction of the pockets, their sterilization and effective drainage is of great value, but in all cases the general condition of the patient must be the guide as to the necessity of more radical treatment.

I maintain that the real lesion from which arises the condition commonly spoken of as pyorrhœa is this break in the continuity of the epithelium. It is immaterial whether pus is present or not. Indeed the presence of pus may be the sign of a healthy local reaction, and I think it is a common experience that the cases which show most marked general symptoms are not those in which pus is much in evidence. What is material is the amount of bone destruction that has taken place and whether the patient is showing symptoms of septic absorption.

When we have once grasped the fact that a break in the epithelium means septic absorption and that this break in the continuity of the epithelium in the mouth is permanent, we shall not talk so glibly of "cures for pyorrhœa," but, on the other hand, we shall be able to recognize the possibility of septic absorption in the mouth even if we cannot squeeze a little detritus from round the teeth.

Section of Odontology.

President—Sir FRANK COLYER, F.R.C.S., L.D.S.

The Relation of Teeth to the Floor of the Antrum.[1]

By CHARLES A. CLARK.

THIS subject appears to me to be of interest, not only to the dental surgeon, but also to the laryngologist, owing to the variation in the size of the antrum and to its position in relation to the teeth, which are sometimes a cause of antral empyema. To the dental surgeon the distance of the floor of the antrum from the apices of the roots of the teeth, is interesting, not only in the case of extractions, but also from the fact that occasionally in filling a root a probe or bristle may pass through the apical foramen for a considerable distance, and it is consesequently not possible to state whether it has entered the antrum or a cyst. And again there is the liability of the antrum to infection from periodontal disease.

Let us consider what is the normal antrum. In "Quain's Anatomy" there is no reference to this subject of relative position of the teeth to the antral floor, but in other text-books the following information is given, as follows :—

"Cunningham's Anatomy," 4th ed., 1913, p. 186 : "The *floor* corresponds to the alveolar border of the maxilla, and differs from the other walls in being *stout* and *thick* ;[2] it is, however, deeply pitted inferiorly by the alveoli for the teeth."

"Gray's Anatomy," 1918, p. 226 : "The floor is formed by the alveolar process of the maxilla, and its lowest part is usually about 1·25 cm. below the level of the floor of the nasal cavity. In a large proportion of cases radiating septa of varying sizes spring from the floor of the sinus in the intervals between adjacent teeth ; in some cases the floor is perforated by the fangs of the molar teeth. The number of teeth whose roots are in relation to the floor of the antrum is variable. The sinus may extend so as to be in relation to all the teeth of the true maxilla, from the canine to the third molar (Salter)."

[1] At a meeting of the Section, held May 31, 1920.

[2] This is as a general statement incorrect, such cases forming the minority.

"Morris's Anatomy," 5th ed., 1915, p. 90 : "The *floor* of the sinus is usually very uneven, due to prominences corresponding to the roots of the molar teeth. In most cases the bone separating the teeth from the sinus is *very thin*, and in some cases the roots project into it. The teeth which come into the closest relationship with the sinus are the first and second molars, but the sockets of any of the teeth lodged in the maxilla may, under diseased condition, communicate with it."

Colyer, in his "Dental Surgery and Pathology," divides antrums into three classes—normal, contracted, and expanded. The latter in my opinion are more usually found, probably forming 60 per cent. of the total.

In extraction of the back teeth it is in some cases impossible to prevent taking away a small portion of the antral floor, adhering to the root or roots, and I have seen quite a few cases in which the antrum was infected by the extraction of a tooth. In one such case a patient whom I had to radiograph was very bitter against his dentist for having opened into the antrum when extracting a tooth, and causing empyema. Of course, he went to someone else. I explained that it was not always possible to prevent it, and I think, somewhat mollified his feelings, especially when I showed him his own radiograph. Whether he would have succeeded if he had brought an action against his dentist, no one can say, but certainly in any legal case of this kind, a radiograph would be essential evidence for the dentist. Again the attempt to remove an apex may in the case of a thin antrum floor, be a cause of infection through the apex entering the antrum.

Cases have arisen in which on taking radiographs the dentist has seen the imminent danger of opening into the antrum in extracting a tooth, and has taken precautions accordingly. In fact, if dentists were previously to see radiographs of the maxilla from which they had to extract a tooth or teeth, they would sometimes be filled with fear and trembling as to the result, and would not be so anxious to carry out the usual procedure—"get the forceps well up."

To the laryngologist the subject should be of interest, and upon his opening an antrum each case should be treated by him on its own merits as shown by a film radiograph, for the time is not far distant when a radiograph will be required before opening an antrum. But the point to which I wish chiefly to draw your attention is the enlargement of the antrum that takes place in the majority of cases. (A paper was written some years ago on this subject by Professor Keith, and I regret I have not had an opportunity to read it.) This enlargement will take place after the extraction of a tooth or teeth, which causes a dipping down of the floor of the antrum, and it is most frequently symmetrical if the teeth present are symmetrical: it is possible for the antrum to dip down very deeply, in fact there will only be an egg-shell thickness of bone between it and the gum. As teeth are subsequently removed, again the antrum will enlarge or dip down, occupying the space formerly occupied by the extracted teeth; hence the ease with which the antrum may be infected by an apical abscess or by periodontal disease. The cause of this expansion I cannot discover. At first I ascribed it to the excessive use of the voice, as in singers or public speakers, or in officers in the Navy or Army, who have to give orders in a loud voice, but I am unable to substantiate this theory. Again in some respiratory diseases I looked for a cause such as asthma, in which the patient, in order to obtain momentary relief, checks the expiration, thus producing pressure in the antrum; but I am not satisfied that this is the explanation.

Again I would draw your attention to the fact that a line of demarcation is formed, beyond which the antrum will not expand on one side and beyond which the alveolus in the edentulous maxilla will not shrink. It would, therefore, appear that the alveolus, being an adventitious tissue, is liable to absorption from both sides. This line of demarcation is fortunate for the dentist. No dentist has yet had the experience of an upper set of teeth, after some months of wearing, becoming a misfit owing to enlargement of the antrum. This line of demarcation appears to be a point at which the shrinking alveolus and the expanding antrum meet, neither encroaching on the other, but with only an egg-shell thickness of bone separating the soft tissues.

When we consider how great a factor oral sepsis is in the cause of ill-health, this thinness of the antral floor is important, and although laryngologists may possibly be of opinion that antral infection is more often due to nasal than dental causes, yet I would urge on them the necessity of eliminating (if possible) dental causation by means of a film radiograph.

In conclusion, let us quote one case: I was asked to radiograph some upper molar teeth, and the patient told me she had had antral trouble for years, and that the whole of the nasal wall had been removed a few years previously. On taking a film radiograph, a root in the antrum was to be seen. How this escaped the many washings out of the antrum, I cannot explain. In such a case a film radiograph would have saved both patient and operator much trouble.

Section of Odontology.

President—Sir Frank Colyer, F.R.C.S., L.D.S.

DISCUSSION ON THE PATHOLOGY AND TREATMENT OF DENTAL CYSTS.[1]

Mr. J. G. Turner.

When fully formed, a dental cyst comprises a wall and fluid contents. The wall always has an inner lining of epithelium, by the breaking down of which the fluid is formed, and an outer lining of fibrous tissue. The cyst is entire; there is never the bare dead end of a tooth-root projecting into the cyst, but there is or has been, always, a septic tooth of the permanent set at the point of origin. In the fibrous wall of the smaller cysts there is always to be found a more or less extensive spongework of epithelium. The earlier the cyst is examined, and the smaller it is, the more abundant is this spongework and the less conspicuous is the cystic cavity in relation to the whole of the tumour. At the same time the cyst fluid, which in the larger specimens is quite liquid and transparent, is found to become more tenacious in composition and to hold in suspension so large a quantity of cholesterin crystals as to give it a superficial resemblance to pus. This resemblance to pus is further increased in the earlier stages by the fact that there is in the first formed liquid contents a certain amount of dead mesoblastic tissue and of inflammatory cells. Such small cysts as we are now thinking of are extremely common, and are still, I believe, very generally referred to as abscess-sacs on account of the opaque whitish appearance of their contents. They are always found in connexion with septic teeth of the permanent set. The cyst may be developed at any part of the root, and a single tooth, especially multi-rooted, may have on extraction more than one cyst attached to it. Further, if the sockets be examined after extraction under a local anæsthetic containing suprarenal extract, it is not infrequently possible to see a small cyst which has been left behind, torn away from the root. This point of frequency is one upon which I wish to lay special emphasis. The fully developed dental cyst is probably the commonest tumour of the body; the small, undiagnosable cyst is many times more common—at a venture fifty times; while the earliest stage of all, which I shall next consider, is practically as common as septic teeth.

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[1] At a meeting of the Section, held June 28, 1920.

The earliest stage of a dental cyst consists of a spongework of epithelium, or in rare cases of a single bulb. This spongework of overgrown epithelium is practically always to be found in the inflamed and thickened alveolar-dental ligament surrounding septic teeth. It is included in a bed of interstitial granulation tissue, the result of a chronic infection of low virulence. I need not labour the point of the extreme frequency of these granulomata. Both the granulation tissue and the epithelial spongework are obviously the result of the same chronic irritation. I have examined microscopically many hundred such granulomata, and only twice found no sign of epithelium; one was a small mass and showed a typical picture of a commencing abscess, and the other was a large granulomatous bed surrounding pyorrhœic lower molars, suggesting the possibility of sarcoma by its unusual extent.

There is thus a diminution of frequency from the beginning to the end of the process of dental cyst formation. Epithelial granulomata (or as I first called them, epithelial root-tumours) are very common in connexion with septic teeth of both sets; small cysts are common in connexion with septic teeth of the permanent set; fully developed cysts, clinically diagnosable, are less common, but still so common as to be probably the commonest tumour of the body, again in connexion only with the permanent teeth. The reasons for this are, I think, obvious. The temporary teeth, alveolus and all it contains, are swept away before cysts have time to form. Acute destructive inflammation and timely extraction account for the progressive diminution among the permanent teeth.

I have spoken of septic teeth instead of dead teeth. This is because though in fact the infection which culminates in a dental cyst is provided almost entirely by dead teeth, nevertheless the earliest stage—epithelial granuloma—is found commonly in the thickened alveolar-dental ligament of pyorrhœa, and I have seen two cases of dental cyst in connexion with living but pyorrhœic teeth. I have also seen a similar cyst of the gum at the point of exit of a chronic sinus connected with a dead tooth.

As to the actual infecting material, I can only say that I have never yet succeeded in demonstrating microscopically anything except yeast-forms, or in cultivating any organism except in one case in which I grew what I take to have been a yeast. The culture unfortunately died out before I could make any experiment with it. If there are organisms in dental cysts I hope Dr. Drew's method of staining will enable us to demonstrate them. At present it seems as if toxic infiltration were the effective cause. The microscopic appearances of the granulomata—thickly aggregated plasma cells—are such as are always found in cases of chronic infection of low virulence.

Once started, the growth of the epithelium appears to go on independently of external stimulus after a certain point. The originating tooth may be removed and the cyst still continue to enlarge, but up to now I have not demonstrated any persistent organism except yeast already mentioned. Yeast forms are, however, common in all inflammatory tissues about the mouth.

Treatment.

In a general way the treatment consists in extracting the originating tooth, making a large opening into the cyst cavity, peeling off the cyst membrane, and keeping the opening patent and the cavity clean till the cavity heals from the bottom. But in many cases this cannot be done, especially as concerns taking away the whole cyst membrane. When operating on a suppurating cyst it may be impossible or unwise, in view of the septic condition of the parts, to do more than extract the tooth and make a large opening for drainage. In dealing with a large cyst of the mandible peeling away the whole of the cyst-membrane is almost certain to expose the inferior dental nerve in the floor of the cavity; in such a case it is best to cut away the roof and sides of the cyst nearly to the level with the floor, leaving the cyst epithelium of the floor to blend with, or be replaced by, the epithelium of the gum, rather than interfere with the nerve trunk. Once there is free drainage—a big, permanent, opening into the cyst—the remaining cyst-epithelium is no detriment to, or, indeed, may be useful in hastening healing. I have never yet seen the epithelium of a dental cyst become malignant, though I think I have seen the fibrous wall become sarcomatous after the cyst had suppurated and discharged through an insufficient sinus for some years. In this case the microscopic diagnosis was fibrosarcoma, but no recurrence has taken place in some fifteen years though only part of the vertical depth of the jaw was removed. In the maxilla a large cyst may press the antrum upwards till it is a mere slit beneath the orbit. This may be suspected when in the case of a large cyst no blood comes from the nose during operation. In these cases as large an opening as is practicable and drainage for one or two years will be needed. Not uncommonly a cyst projects into the antrum or even fills its cavity. In such cases blood will come from the nose when the deeper part of the cyst membrane is detached. Drainage for a considerable time will then be needed to allow of regeneration of the antral ciliated epithelium and to allow the true cyst cavity to contract. After a time the case should be treated as a healing case of antral suppuration and the mouth wound allowed to close when the whole bone cavity appears healthy.

The wider the bone can be excised and the less cavity there is left the quicker will be the healing, indeed some few saucer-shaped cavities may be left to heal by first intention, but efficient and prolonged drainage will secure success with an opening far less than the ideal. This fact is sometimes of great use in preserving teeth which would have to be extracted to extend the opening, or in avoiding facial deformity. The best means of securing drainage is to make at once a vulcanite plug to fit the opening. This can be moulded in the mouth in soft "Stent's" to the desired shape and size and the "Stent's" plug at once flasked. Till the plug is made the opening must be kept packed —deep plugging is not wanted. For syringing, 10 grm. sod. bicarb. to the ounce of 1-80 carbolic lotion is as good as anything I know of. The sod. bicarb. washes away blood, mucus, &c., and the carbolic acid 1-80 is inhibitant of germ growth.

Mr. Howard Mummery.

As the clinical aspects of the question will, I am sure, be fully presented this evening I propose to confine my remarks chiefly to the microscopic structure of cysts. The predominance of certain cells and the arrangement of the elements of the growth seem to be more varied than we have generally been led to suppose and I would like especially to draw attention to these variations. The essential elements of granulation tissue, of which these growths are built up, are endothelial cells, and fibroblasts or connective tissue cells. In the great majority of cases epithelial cells are present in more or less abundance and inflammatory products are also included. Among the inflammatory products we include leucocytes from the blood, wandering cells (chiefly tissue cells), plasma cells, and degeneration products as fat, cholesterin, and calcified substance.

The Endothelial Cells.—These form the walls of the delicate embryonic blood-vessels found in granulation tissue, but besides forming the walls of blood-vessels they are also found free in the tissue and have been seen to thrust out pseudopodia and seize bacteria and it is even held by Adami, so that although the endothelial cell and the fibroblast are distinct cell species in regard to their origin, in inflammatory tissue the distinction is lost and both may become fully formed fibroblasts and give origin to scar tissue.

The Fibroblast.—This is oval- or spindle-shaped and in addition to the cells there are many strands of fibrous connective tissue which traverse the growth in many directions and have a more or less definite arrangement in many parts, in all cases forming a boundary wall to the cystic growth. We may thus consider that the endothelial cells and the fibroblasts are *the building up cells* of the tissues of these growths and that the other cells involved in them are more or less adventitial.

The leucocytes from the blood are principally of three varieties:—

(1) *The polymorphonuclear leucocyte*, now generally called the neutrophil leucocyte, which is found almost exclusively where bacteria are present and suppuration is in progress. It is an active phagocyte for bacteria.

(2) *The mononuclear leucocyte* which ingests red blood corpuscles, fat and other foreign bodies, and

(3) *The lymphocytes*, from which the mononuclear leucocytes are in all probability derived. These are seen in great abundance, but they are not phagocytic or amœboid, and whether they have any chemical action is still uncertain.

The eosinophil leucocyte appears to be but rarely present in these growths.

The Plasma Cell.—Another cell which forms a very large proportion of the cellular elements of granulomas is the plasma cell. These cells are easily recognized by their form and the appearance of the nucleus. They are larger than lymphocytes but vary considerably in size, and are generally considered to be derived from lymphocytes by a "modification of their nucleus and protoplasm"; they are slightly amœboid and have an excentric nucleus. This nucleus is divided into masses at its periphery, giving it a wheel-like appearance; a clear halo generally surrounds the nucleus and scattered granules, which usually accumulate at the margins of the cell, are found in the cytoplasm. From their great abundance in granulation tissue it seems evident that they must have some important function, and although they cannot take up foreign substances into their interior they probably produce a digestive ferment which acts upon toxins and degeneration products. Some authors consider, in agreement with Unna, that plasma cells are developed from connective tissue cells, and Rumpel describes transition forms between both connective tissue cells and endothelial cells to plasma cells.

Wandering Cells.—Having lately examined a good many dental cysts and other granulation tissue growths, as dental polypus, and scar tissue, by Unna's method of staining, I have been struck with the great abundance in many cases of these wandering cells, which the method brings out with great distinctness. These cells are now generally considered not to be derived directly from the blood but from the tissues. They are by some called mast cells, after Ehrlich, but there appears to be much confusion in the use of this term; it is applied to neutrophil granular cells found in the blood and marrow, as well as to other cells of tissue origin, and it seems probable that these mast cells of the tissues are identical with the clasmatocytes of Ranvier. Both the so-called mast cells and the clasmatocytes are characterized by coarse granules, which take a red stain with polychrome methylene blue and other similar basic dyes, and are therefore neutrophil or basophil in reaction, in distinction to the eosinophils, which are acidophil. Ranvier called these cells in the tissues, clasmatocytes, from the Greek word *κλάσμα*, a fragment, because they are often seen breaking up, and their granules are scattered in the surrounding tissue; in many cases the course of the clasmatocytes may be traced by the trail of granules they leave behind them. These cells are very actively amœboid, and assume the most extraordinary and varied forms. They possess a blue staining nucleus, often lobed, and although amœboid are held not to be phagocytic. They are said to be found in abundance where fatty substances are present, but I have seen them in many places where no trace of fat can be detected by means of fat stains. It is probable that although not phagocytic they act upon toxins by some ferment or antibody to which they give rise. If we call these wandering cells of the tissues which break up into fragments, clasmatocytes, as Ranvier originally called them, then these neutrophil red granule staining cells are not mast cells but clasmatocytes, for in numerous places in the cyst wall they are seen to break up into fragments. As some authorities consider—the mast cells (of the tissues) and the clasmatocytes may be identical.

I have not found in any of the cysts which I have examined the very extended protoplasmic processes and evidences of hurry on the part of the wandering cells shown in the drawing from the pulp polypus and scar tissue, although in the cysts many show changes of form and pseudopodia, and also the scattered granules, as may be seen under the microscope on the table. These wandering cells are found in all kinds of low grade inflammations. They are abundant in scar tissue, and I have found them in absorption of the gums as well as in pulp polypus and dental cysts, and, as described by McCallum, in nasal polypus, in which I examined them from some polypi kindly procured for me by Mr. E. D. D. Davis.

Although so much work has been done in recent years on the cells of the blood and tissues, especially by Pappenheim, Maximow and Marchand, the origin of these wandering cells still remains somewhat obscure. Maximow considers that "the plasma cell is but one phase in the life-history of the *polyblast*, a cell, that is, that may originate either from lymphocytes or from connective tissue cells, which may develop into fibroblasts and so into connective tissue or into clasmatocytes, large cells with pronounced processes which are constantly shed into mast cells of the tissues, or cells filled with large basophil granules, or into cells of the plasma type." Whatever their origin, there are, however, immense numbers of cells in the tissues which hurry to the seat of injury when certain kinds of inflammatory processes are set up. The relationship of clasmatocytes to connective tissue cells is certainly suggested by the appearances in scar tissue.

Römer, in the article on "Granuloma" in Scheff's "Handbuch" (1909), divides these growths into epithelium-containing granulomata and pure connective tissue growths, in which latter, he says, not a trace of epithelium is to be found. In the first class, cysts arise through degeneration of the epithelium, whilst pure connective tissue granulomata, according to Römer, never become cystic. The existence of a dense fibrous connective tissue investment is characteristic of all these growths; it bounds the whole structure and binds it firmly down to the root of the tooth. In some forms, especially the connective tissue forms, a strong development of endothelium is found, and Römer calls these endothelial granulomata, and further he calls myxoid those in which mucoid tissue is found. But endothelial cells and fibroblasts are, as we have seen, the most constant cells in granulation tissue, so that the distinction of a separate class of endothelial granulomata scarcely seems advisable. The myxoid granulomata can also hardly be accepted as a distinct class as they are only probably to be looked upon as a degeneration form. The chief distinction made appears to be into those which contain epithelium and those which contain none. In the cases examined by Römer he found the proportion of growths which contained no epithelium to the epithelial growths to be as 8 to 19.

I have not found a single case in which no epithelium was present, but I have not had the opportunity of examining so many of these growths as the author quoted. It is possible, however, that in an examination of the sections small masses of epithelium may have been missed. In one or two cases I could detect none at first, but further sections revealed epithelium in some abundance. In many cases in which there has been extensive suppuration in the neighbourhood, we can easily understand that the epithelium may have been lost.

Professor Partsch, in a paper read at the Fifth International Dental Congress, states that the cystic degeneration of a granuloma takes place in the connective tissue and not in the epithelium, and that this is the case in the epithelial as well as in the connective tissue granulomata—he says he bases this statement on the examination of over 300 cysts. It certainly seems difficult to understand how this can be the case. The cyst cavity is usually surrounded by epithelium, and epithelial cells are seen lying in the lumen, which have evidently undergone degeneration and vacuolation. The only clefts in the connective tissue portion of the growth have been those produced by the abundant cholesterin crystals, which have been dissolved out in preparing the section, for these cholesterin crystals are often scattered throughout the growth to which they give a very characteristic appearance. The clefts or separations between the connective tissue fibres appear in this case to be due to the crystallization taking place between them.

In an interesting paper on alveolar abscess, by K. H. Thoma, in the *Dental Cosmos* for 1918, the author thus accounts for dental cysts in which no epithelium is found. He says: "The presence of epithelium is not constant in the periodontal membrane, and therefore is not regularly found in the dental granuloma." But, surely, Malassez's rests are always seen in more or less abundance in the membrane, and as the dentine of the root cannot be formed without the epithelial sheath of Hertwig, epithelium must be constantly present in the periodontal membrane.

Dr. Rumpel, of Berlin, in his paper contributed to the Fifth International Dental Congress, described plasma islands in these growths, that is, areas chiefly occupied by plasma cells and surrounded by strands of epithelium. That this arrangement is often seen there is no doubt, but in many others there is no such regular arrangement, and in one of Sir Frank Colyer's cases, which I described in a former communication, the plasma islands are surrounded by connective tissue strands and fibroblasts, and no epithelial cells are seen in the neighbourhood. In this specimen the islands were very numerous and contained many large blood-vessels and much extravasated blood. In other cases I have found a thin layer of connective tissue between the enclosing epithelial strands and the plasma cells.

Time will not allow me to dwell upon the degeneration products found in cysts, but in all of the cysts which I have examined with a special fat stain (Sudan III), I have found fat in places in many parts, as free globules, in others, within mononuclear leucocytes. Under the microscope I have shown several instances of fatty infiltration in pulps and in cysts, and the fat can easily be seen both within and around the cells. In one case given to me by Mr. Dolamore there is a very curious appearance throughout the larger portion of the cyst, suggesting degeneration in the epithelium. It has been suggested to me that this is not an epithelial degeneration, but is some alteration in a cell of the mesoderm. One is able however to trace the gradual change and vacuolation of the epithelial cell from its typical form to this condition, and I think there cannot be much doubt that the tissue is epithelial and is probably the degeneration product described by Römer as a myxoid granuloma.

Calcific degeneration is often met with in cysts, especially around the blood-vessels, and is usually seen in the form of little spherical masses of calcified material, many of them taking the form of typical calcospherites. Dr. Wyatt Wingrave lately showed me a preparation from a dental cyst in which there was a large amount of this spheroidal calcification. As we know that calcification is a common degeneration product in altered and imperfectly nourished tissues, we are not surprised to find it in the walls of old standing cysts.

Cholesterin.—This varies very much in amount. In some cysts, as in one sent to me by Mr. E. D. D. Davis and shown under the microscope, one part of the cyst was cut up into narrow pointed spaces, running in various directions, the crystals having been dissolved out by the alcohol. Such a condition is often seen in these growths.

Mr. HERBERT TILLEY.

As a rule only patients with large dental cysts of the upper jaw consult the nasal surgeons. The smaller ones about the size of a cob nut can be cured by dissecting off a flap of the oral mucous membrane, removing the entire cyst with a sharp spoon and then laying the reflected flap of mucous membrane into the cavity thus produced. Rapid healing occurs. In the case of those large cysts which invaginate the anterior antral wall and often reduce that sinus to small dimensions, it has been my custom to reflect the oral mucous membrane and then to remove all the lining of the cyst and its bony wall. The anterior half of the inferior turbinal is removed by suitable scissors and snare and also the inner antral wall. Finally, the mucous membrane incision which covered the anterior wall of the cyst is sutured and thus the bucco-antral wound is closed. It heals within a few days by immediate union and we have permanent, spontaneous and free drainage into the nasal cavity. The advantages of this method are:—

(1) There is no contamination from food or organisms from the mouth.

(2) There is no prolonged or painful after-treatment which may be entailed if plugging of the cyst cavity is adopted.

(3) The rapid cure, as compared with the "plugging" or "packing" method.

Another form of dental cyst occasionally comes for treatment at the hands of the nasal surgeon because it causes nasal obstruction. I refer to that form which arises from the incisor teeth. The cyst bulges upwards in the floor of the nasal cavity just beyond the vestibule and may extend backwards to the posterior limits of the hard palate. It forms in this way a cyst shaped like the finger-stall of a glove. I have approached these from underneath the upper lip by means of a free incision through the mucous membrane. Having entered the cyst cavity the membrane lining it is thoroughly removed and then its thin bony convex upper border within the nasal cavity is forced downwards so as to obliterate the "finger-stall" and thus leave the nasal mucous membrane intact.

(Some lantern slides were shown illustrating the above methods.)

Mr. F. Coleman.

My practice has been to open a dental cyst in such a way that the cavity is rendered self-cleansing or made accessible to mechanical cleansing. In the maxilla this ideal can usually be attained; thus, the incision for a dental cyst should be made near the alveolar border so that the floor of the cyst is flush with that of the incision and no ledge projects to hinder drainage.

The length of the incision should correspond to that of the cyst so that "pockets" or "dead spaces" difficult to drain are not left at either extremity. Roots of carious teeth must first be removed to allow the incision to be made, but sound teeth need not, as a rule, be sacrificed to this purpose. A portion of the cyst wall above the horizontal incision should then be cut away. A mandibular cyst is less easily converted into a drainable cavity. The indications are to make the incision as low in the cheek sulcus as possible, so that the floor of the cyst is on a level with or approaches the level of the cheek sulcus. Extensive cysts hollowing out the greater part of the mandible might be better treated by an external incision as this would allow of more dependent drainage (*see* fig. 3), but would result in a discharging external wound of many months' duration and be followed by a puckered scar.

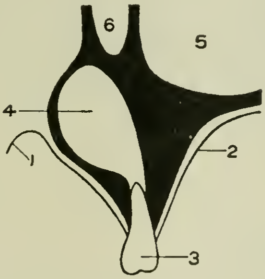

Fig. 1.

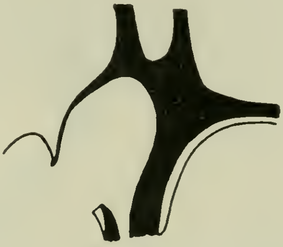

Fig. 2.

Fig. 1.—Showing a maxillary dental cyst arising from a premolar tooth (diagrammatic). 1, mucous membrane of cheek; 2, mucous membrane of palate; 3, premolar tooth; 4, cyst; 5, nasal fossa; 6, antrum.

Fig. 2.—Showing premolar tooth removed and cyst opened. Lettering as in fig. 1.

I have not found it necessary to remove the lining membrane of a dental cyst, in fact, in most cases, there is no distinct membrane to remove, the interior of the cyst being uniformly smooth and glistening; nor do I plug the cavity with gauze unless to arrest hæmorrhage, or to give some comfort to the patient, and, under these conditions, the gauze can usually be removed after a few hours.

The after-treatment consists in syringing or irrigating the cyst cavity with non-irritating antiseptics, in fact, in mechanically cleansing the cavity. I suggested these principles of treating a dental cyst, over ten years ago, and incorporated them in a paper, which appeared in the *Royal Dental Hospital Annual Reports* for 1912.

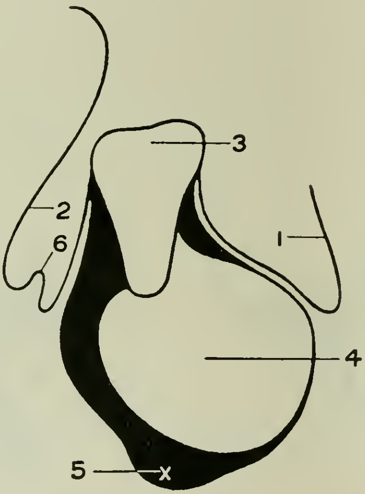

FIG. 3.

Showing a mandibular dental cyst arising from a molar tooth (diagrammatic). 1, Reflection of mucous membrane on to cheek; 2, reflection of mucous membrane on to tongue; 3, molar tooth; 4, cyst; 5, lower margin of jaw; 6, sublingual fold or fringe.

Mr. E. C. HUGHES.

I have recently had under my care two cases of cystic expansion of the ascending ramus of the mandible, due to dental cysts. As I believe such cases to be uncommon, I have taken advantage of this opportunity of recording them.

Case I.—An edentulous woman, aged 55 (approx.) was sent to me on account of a swollen left cheek. In 1911 she underwent an operation on the jaws for the same condition, and was temporarily relieved, but for some time the cheek had been swelling again on the inner side of the cheek; running on to the anterior margin of the ascending ramus of the mandible was a linear

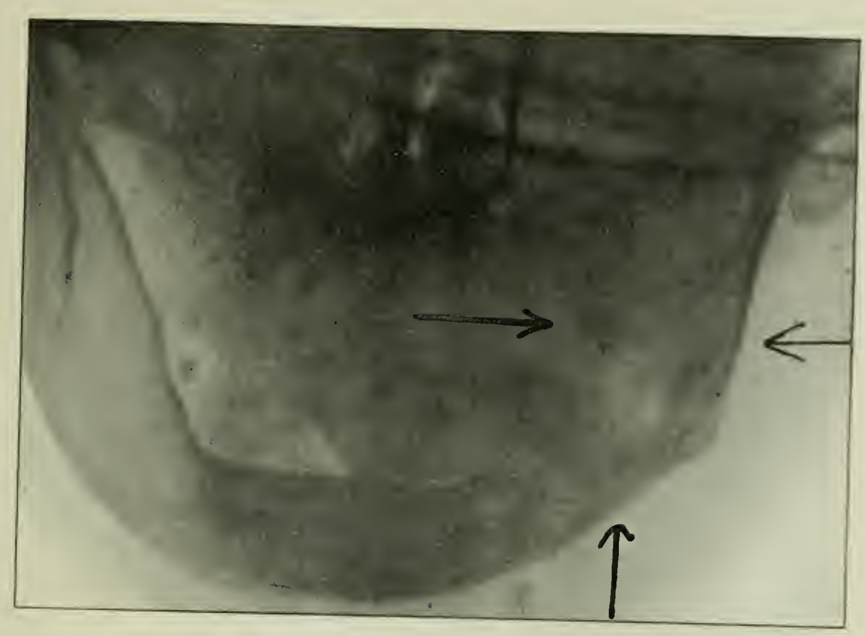

FIG. 1.

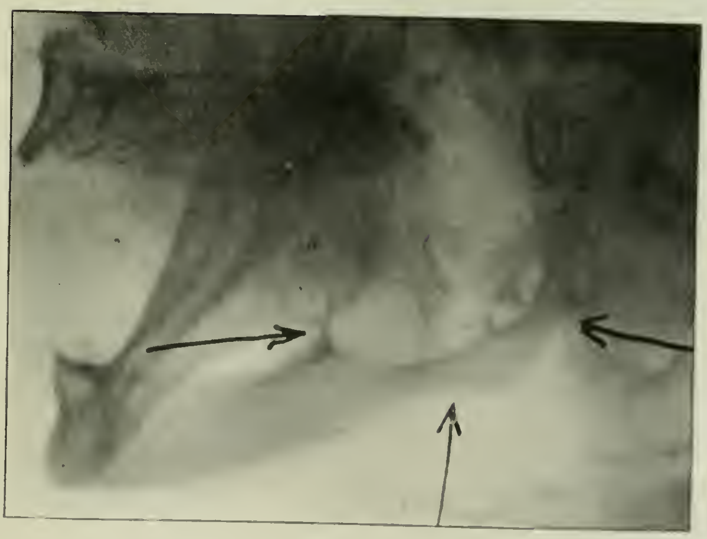

FIG. 2.

scar, while below and above it, the ascending ramus was considerably expanded, the expansion extending into the molar region of the horizontal ramus. The expansion was apparently completely covered by bone, except at two points, where the cyst cavity appeared to be completed by the mucous membrane of the molar region. The two skiagrams (figs. 1 and 2) show the expansion of the entire ascending ramus and of the posterior molar region.

Case II.—An edentulous woman, aged 35, was sent to me on account of a swollen right cheek. The swelling was so conspicuous in this case, that it had been diagnosed as a parotid tumour. The expansion of the ascending ramus and posterior molar region was far greater than in Case I, and its capsule or limiting wall contained very little bone, and it was easy to obtain fluctuation between the oral and cheek portions of the expansion. The skiagram (fig. 3) shows the expansion in the lateral view (the anterior margin of the ascending ramus showed so faintly in the photograph, that I have accentuated it with an ink line).

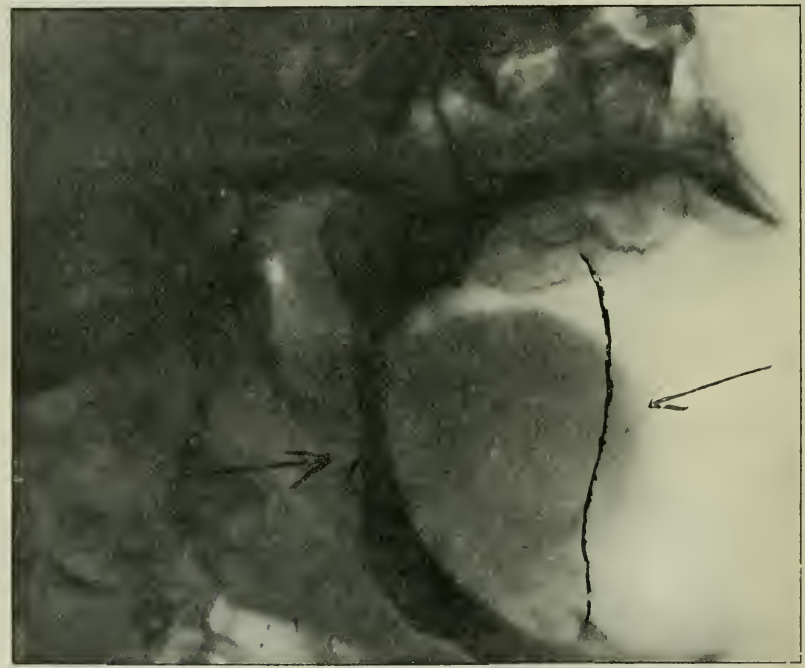

Fig. 3.

I have no doubt that both these cases are examples of dental cysts arising in connexion with a molar tooth: they both gave a history of acute inflammatory trouble in the years preceding the development of the expansion of the bone, the skiagrams show no developmental errors, and their formation is in every way comparable to a dental cyst elsewhere.

Both of these cases were operated upon by an external incision, as it seemed to promise a rapid easy way of removing not only the cyst, but also the external projection of the cyst, i.e., the swelling of the cheek, which led both patients to seek advice. The alternative method of treatment, an incision through the mucous membrane, meant a long and tedious after treatment, for these large cavities would require many weeks' attention of frequent syringings and cleansings, with the possibility of necrosis of bones and no certain promise of removal of the facial deformity.

The operation consisted of an incision under the mandible, parallel with the insertion of the masseter. The position of the cervical branch of the facial nerve must be remembered. In both my cases it was damaged, as evidenced by a drooping of the angle of the mouth. In both cases this weakness has now passed away, so it is probable that the branch was merely bruised. The incision must be well under the jaw to avoid this branch, for the chance of permanent damage to it is the only objection to the external incision. The masseter and periosteum were carefully raised from the ascending ramus until a large area of the cyst wall was exposed: the outer part of this was carefully cut away and the dental cyst lay exposed. As in both cases the cyst wall was completed by or was closely adherent to oral mucous membrane, the cyst could not have been dissected out intact, as the dissection would certainly have opened the mouth cavity. The cyst was then incised and the greater part of the epithelial lining removed, except where it was adherent to oral mucous membrane. In these situations the lining can be destroyed by some such means as carbolic acid. The resulting cavity was filled by a fat graft, as it lessened the chance of a hæmatoma. The subcutaneous tissues and platysma were sutured, after replacement of the reflected flap and the skin incision closed by a subcuticular suture. In both cases recovery was rapid, and healing was complete in ten days.

Mr. Montagu Hopson.

With regard to cysts occurring in the ascending ramus of the mandible to which Mr. Hughes has referred, and in the treatment of which I have been associated with him, I would merely offer this suggestion. They may arise from epithelial tissue derived from an excessive backward prolongation of the primary tooth band, which normally gives off the bud to form the enamel organ of the mandibular third molar about the third year. In some of these cases the third molar was absent.

Some few years ago I saw a boy aged 9, who presented the usual signs of a cyst of the maxilla on the right side. When first he came under observation both the deciduous molars were in place, but in the short time which elapsed before treatment was undertaken the first deciduous molar was shed. A radiogram (fig. 1) confirmed the diagnosis. At the time this was taken the cusps of the first premolar were just appearing through the gum. It will be noticed that the cyst was of considerable size, and that in the course of its growth it had displaced the developing second premolar upwards and backwards. I referred the boy to my colleague, Mr. T. B. Layton, for treatment, and he will describe the operation which enabled us to remove the cyst intact without disturbing its connexion with the first premolar to which it was firmly attached. By a most unfortunate accident the specimen was destroyed in a fire which occurred in the pathological laboratory at the hospital before any sections had been made, but not before it had been photographed (fig. 2). Seeing that the cyst arises from tissues above the neck of the tooth it may probably be described as "dentigerous" rather than "dental" if the latter term is to be restricted to cysts springing from epithelium contained in the wall of the follicle, in close apposition with the root of the tooth. Further it may be interesting to speculate whether the stimulus which led to the proliferation of the epithelial cells was due to toxins emanating from the carious deciduous molar overlying the developing premolar.

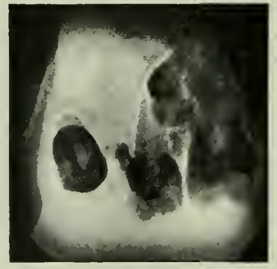

FIG. 1.

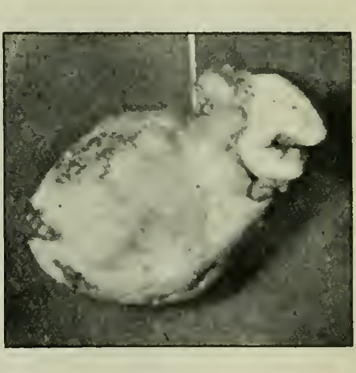

FIG. 2.

Mr. T. B. Layton.

I think that the case which Mr. Hopson has just described to you may afford the clue to the pathology of those cysts of the jaw in which no connexion with any tooth, whole or carious, erupted or unerupted, can be found. Here was a case in which the tooth was seen partially to erupt while under observation. Had the eruption become complete the tooth fang might subsequently have lost all connexion with the cyst. We have been told to-night that the pathology of dental cysts and of dentigerous cysts is the same, and this case also bears that out, for in the carious milk tooth which was removed we have the infective factor which is not usually seen in the dentigerous cyst but which is constantly present in the case of the dental cyst.

These two kinds of cyst may be the same pathologically but they differ clinically in the presence or absence of this infective material. My experience has been mainly limited to the dentigerous cyst. These I have dissected out, and I have packed the cavity with decalcified bone tissue, and have then sewn up the wound. They have healed well. I should not venture to do this in the case of a dental cyst with its carious tooth in relation to it.

I do not agree with Mr. Tilley that it is necessary to open the cyst cavity into the nose. In one case in which I did this I regretted it. It was that of a large cyst cavity which had been drained into the mouth and which I thought was a suppurating maxillary sinus that had been so drained. I performed a radical operation on his maxillary sinus and sewed up the hole through which it had been drained into the patient's mouth. This part of the suturing broke down. As a result of an extensive operation the patient was left no better off than he had been before so far as the object of the operation was concerned. That he was as well off in other respects I cannot say as I have lost sight of him.

Mr. E. D. D. Davis.

I show an X-ray photograph of a dental cyst with its outline marked by a piece of the finest fuse wire which has been introduced into the cyst for the purpose of obtaining a clear X-ray. The injection of a bismuth solution failed in this respect.

I have always completely removed the cyst wall by dissection and have the notes of eighteen cases in which this treatment was most satisfactory. It is not by any means a difficult procedure if a free incision is made and the whole of the outer wall of the bony capsule is removed by nibbling forceps so as to produce a saucer shaped cavity without any overhanging edges, and then with a suitable dissector the cyst wall is easily peeled out. In removing the cyst great care should be taken to avoid opening the antrum or the cavity of the nose, and if either cavity is opened a permanent fistula may result. If the antrum is accidentally opened, then I agree with Mr. Tilley that drainage of the antrum by removing the antro-nasal wall is necessary.

Care should be taken not to remove too much of the lower edge of the cyst and encroach on the palate, and I should like to know the opinion of dental surgeons as to how much of the palate can be removed without disadvantage.

I never pack any cysts, and only in one out of the eighteen cases was it necessary to resort to gauze packing soaked in Whitehead's varnish to control hæmorrhage, and in that case the hæmorrhage was definitely due to faulty operative technique. Packing promotes sepsis, delays healing, and is irksome to the patient.

Mr. J. H. Gibbs, of Edinburgh, gave a clear account of the removal of dental cysts by dissection at the International Dental Congress, 1914, so that further description of the operation is unnecessary.

Mr. J. G. TURNER (in reply).

The endothelial cells described by Mr. Mummery are to be found commonly in the early solid stage of dental cyst formation. The large epithelial cells undergoing (?) colloid degeneration are also to be found on inflammatory gum-tags. In operating I never encroach on the palate or the nose; a perforation in either direction is extremely difficult to close. I do not think the cases of dentigerous cyst of the ascending ramus require an external wound. I have treated three such cases, equally extensive, by internal operation and the use of vulcanite plugs. Mr. Hopson's case smees to have been a dental cyst connected with a temporary tooth—if it can be substantiated it stands as the first on record. I have never yet had to open into the nose as Mr. Tilley suggests and I have treated cases in which the face seemed to be all cyst. I think I can make the mouth as clean a cavity as the nose, and I know I can drain into it more easily. The patient is quite comfortable with the vulcanite plug and daily syringing and is spared an attack of "sinusitis" whenever he gets a "cold in the head."

The differentiation between dental and dentigerous cysts has been disputed. I hold to the distinction between the two conditions because, in point of fact, a dental cyst always arises as a result of chronic sepsis in connexion with an infective tooth-root, while a dentigerous cyst arises as an exaggeration of a process which is part of the normal mechanism of eruption and has always the crown of a tooth projecting into its cavity. Its starting-point appears to be an obstruction to eruption, and its histological origin is the epithelial-lined crypt of the unerupted tooth-crown. The dental cyst is, in one sense, a sequestration cyst. It has its origin in the last possible remnants of the enamel organ, the remains of the epithelial sheath of Hertwig. From the earliest formation of the epithelial tooth-band to this last moment of root-determination by the epithelial sheath of Hertwig there is a continuous sequestration of epithelial masses, and from them are formed supernumerary teeth, solitary cysts, multilocular cysts, various odontomes, even carcinomata. Cysts encroaching on an unerupted tooth from the side or from the apical area seem to have been classed as dentigerous cysts, and hence, I think, has arisen the confusion it is sought to clear up. In that they are sequestration cysts they are allied not to dentigerous but to dental cysts, and the similarity would be all the greater if an infective origin could be proved.

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Section of Ophthalmology.

President—Mr. W. T. HOLMES SPICER, F.R.C.S.

Acute Disseminated Choroido-retinitis of Doubtful Origin.[1]

By RAYNER D. BATTEN, M.D.

THE chief interest of the case lies in the history and in the changes taking place under observation. The patient, aged 27, was first seen at the end of March, 1919. Three weeks previously, whilst returning home from being demobilized, he suddenly—i.e., in the course of a few hours—lost all central vision in his right eye.

On examination, there was a flat or nearly flat subretinal patch towards the temporal side of the optic disk, just below the superior branch of the retinal vessels, about one-third the size of the disk, somewhat triangular in shape, with a small retinal vessel passing over it. It was surrounded by an area of retinal œdema. The rest of the fundus was normal, also the left fundus was normal.

He was seen and examined regularly about once a week, but on examining him on May 23 the whole of the right fundus was found to be thickly strewn with patches of choroiditis in about the same condition that they are now. The central patch was subsiding and his vision improving.

He was seen by our President and Mr. Hudson both in the early and later stages.

The condition was strongly suggestive of a syphilitic choroiditis. The Wassermann reaction was taken twice and on both occasions was negative. There was no other evidence of syphilis. He also strongly denies any possibility of infection.

He was examined by a physician and by a dentist, who both returned negative reports.

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[1] At a meeting of the Section, held November 5, 1919.

In October, 1916, he had a wound in the sole of his foot from barbed wire, which became infected and did not heal till October, 1917. There is still a deep scar under the great toe somewhat tender to pressure.

On Mr. Holmes Spicer's suggestion that the condition might be septic in origin, I referred him to the consulting surgeon at Millbank, who reported that he did not find any evidence of septic trouble in the foot, and declined to open it up in view of the delayed healing of the original wound.

I continued to see the patient up till the beginning of August. The vision in the right eye had improved: Right $\frac{6}{18}$, left $\frac{6}{6}$.

I saw him next on October 2 with the history that the vision in the left eye had failed suddenly two weeks previously, with a central blank spot. On examination of the left fundus there was a condition very similar to that of the right eye. There were three central retinal patches with surrounding œdema and the fundus was thickly strewn with patches of disseminated choroiditis. Vision: Right, with correction, $\frac{6}{9}$, and J.2 easily; Left, $\frac{6}{36}$, J.12 with difficulty.

The question as to the cause is of considerable importance: (1) in view of the possibility of further attacks and further damage to his eyes; (2) if the choroiditis is due to the wound of his foot he is eligible for a considerably increased pension.

As regards treatment also: His vision has improved under anti-syphilitic treatment, but the question of dealing with his foot, if the condition is aseptic, has still to be decided.

The PRESIDENT: I had an opportunity of seeing this case nearly at the beginning of the attack. It seemed to me that the whole of the involved area was slightly raised above the remainder. I took the view that the condition was probably septic, starting from the injured foot. There was considerable tenderness in his foot, and he could scarcely bear pressure on it. Even now the foot is tender: I see no reason to change the opinion I gave at first, that the ocular condition is a septic one.

Unusual Optic Nerve Tumour.

By J. F. CARRUTHERS, Major R.A.M.C.

THIS case presents very unusual features, if, indeed, it is not unique. The man has been under my observation since last July, and the case has been seen by a considerable number of our members to-night.

I shall be glad if some more remarks are made about it than were made about the preceding case shown, because it has puzzled me considerably, and I shall be glad of any light that can be thrown upon it, both for the patient's sake and also to enable me to gain some idea as to the prognosis.

Patient, aged 21, of fifteen months' service in the Royal Field Artillery, complains of pains over the eyes. Apparently totally blind, without light perception in the right eye. Right eye, sight defective since childhood. Eyes rather prominent. Onset about six months ago as far as pain is concerned. Pupils on both sides react to light, very little if at all to accommodation. Tension not increased.

July 4, 1919: Heart, faint systolic murmur at apex, which is about a normal position. Chest, *nil*. First noticed that sight was defective in the right eye when about the age of 14.

July 9, 1919: Right pupil reacts to light very imperfectly with a hippus-like movement (on exposure to direct light); consensually it reacts well. Neither pupil reacts to accommodation. Right eye, no perception of light. Left eye, $\frac{6}{5} + {}^{+1}_{+0\cdot 5}$. Right fundus: A large pearly white mass wholly obscures the disk and extends forward some way in to the vitreous. It is rather larger than a normal disk in diameter—no hæmorrhages on it. It has apparently dragged the surrounding retina along with it as this is detached from the subjacent choroid in a circle surrounding the disk with a radius about two-thirds of the way to the macula. There is slight disturbance of pigment above. The retinal vessels are raised on the detached area but not nearly so much as the white mass.

History: He had slight vision in the eye when at school, as at the age of 14 he first discovered the defect. It must therefore have got worse in the last seven years and if so cannot be a congenital tumour. Tension normal, left fundus normal.

DISCUSSION.

Mr. HUGH THOMPSON: I saw this case at the Brook War Hospital in June. I was extremely puzzled by it, and at first I was inclined to think it was a dangerous tumour. Therefore I took the patient up to the Western Ophthalmic Hospital and consulted some of my colleagues—Dr. Rayner Batten among them—about it. We concluded it was probably the result of a very old injury, probably dating from birth, and that it was not progressive; it is important to know whether it is progressing or not. I cannot definitely say that there is any difference between the appearance this evening and that shown last June when I last saw the case. There may be now a little more pigmentary disturbance. There was some pigmentary disturbance outside the ring in June, in the retina or choroid, or in both. I am afraid I cannot make any more definite statement.

Mr. M. S. Mayou: Is there any history of tubercle and may not this condition be a cicatrizing tubercular nodule in the nerve head? The age of onset would accord with that, and it would account for the rings round the disk and the alteration of pigmentation which has been noticed.

Mr. J. Herbert Parsons: I think the case a member has just referred to was one under the care of the late Mr. Gunn. It was a very interesting case. There was a little mass at the edge of the disk, like that seen in the present patient. The retina was folded up, and there were definite evidences of hæmorrhage under the retina. In the light of Coats's work, there is no doubt that it was of the character of exudative retinitis, in a very early stage. In the present case it is difficult to decide whether it is a birth injury or not: I think this is the most probable explanation.

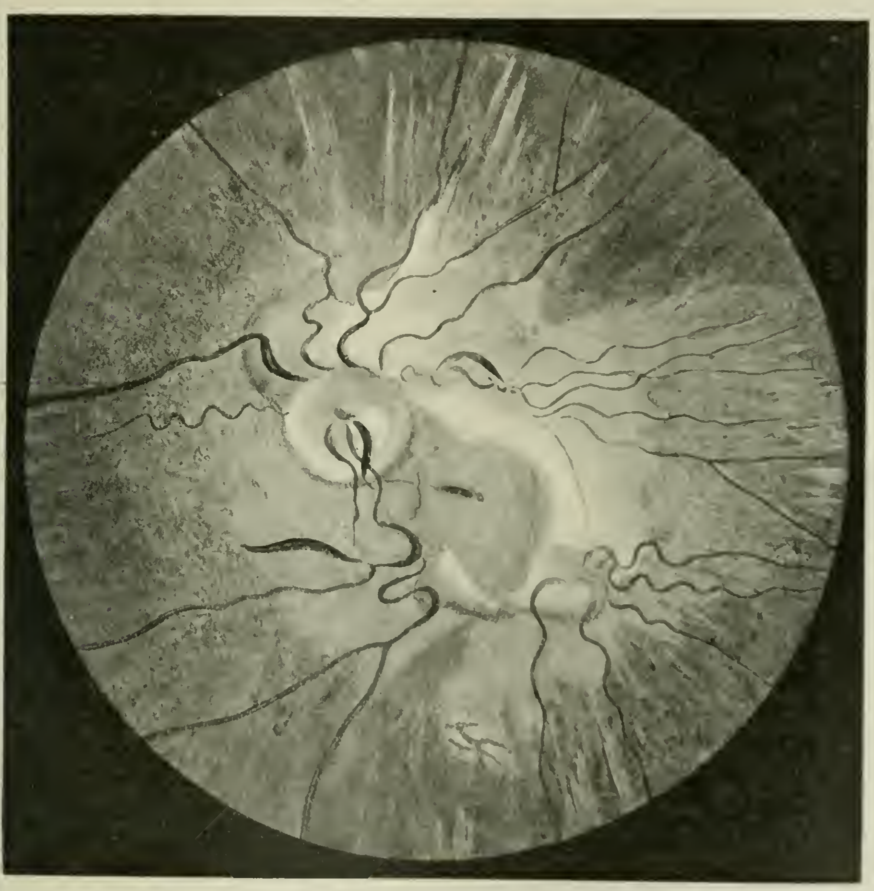
Major Carruthers (in reply): The only reply I can make in answer to Mr. Mayou's question is, that at the medical examination there was no physical evidence of tubercle: but we did not have the von Pirquet test done. Lungs and heart were pronounced sound, and the urine showed no abnormality upon examination. I take it that the prevailing opinion is that the case should be left alone.

Injury to the Fundus Oculi at Birth.

By H. R. Jeremy, F.R.C.S.

The patient is a little girl, aged 7, who attended the London Hospital with her mother in October, 1919. The mother had noticed an external squint of the left eye since the child was 7 months old. The birth of the child was attended with difficulty, and required the assistance of a midwife. The left eye was injured, and the baby was taken to a doctor. The mother states that when she first saw the baby three days after birth the left eye was bandaged.

On admission: There is external strabismus of the left eye, which fixes badly. There is no muscular paresis. Vision consists of perception of light only. Vision in the right eye is $\frac{6}{18}$. The left cornea is clear; pupil equal to right, reacts to light directly and consensually, and to accommodation. The media is clear. The disk is paler than normal, and appears sunken above; the retinal vessels are contracted and pass directly over the lower edge of the disk without distortion. Above, and to the outer side of the disk there is a mass of connective tissue extending outwards to the macula region. Retinal vessels are seen curving over the mass and disappearing below the upper edge of the disk. The disk has a similar appearance to the drawing illustrating avulsion of the optic nerve in Lagrange's book on "Fractures of the Orbit."



In the cases of avulsion of the optic nerve shown by Sir William Lister at this year's meeting of the Ophthalmological Society there was a distinct hole at the disk. There is no hole at the disk in this case, but the appearance suggests a partial avulsion of the nerve in the upper part, and a rupture of the choroid above and to the outer side of the disk, followed by hæmorrhage and a proliferating choroidoretinitis.

I am indebted to Mr. Roxburgh for the beautiful drawing of the fundus and for giving me an opinion on the condition.

Mr. MALCOLM HEPBURN: I think it is most unlikely to be avulsion of the nerve head. In the cases of avulsion which have been published the disk is almost unrecognizable, whereas in this condition, even in a late stage, the disk is plainly and definitely marked. It is probably some kind of exudation into the choroid and round the disk, and there is possibly also some organized hæmorrhage.

Case of Birth Injury.

By W. H. McMULLEN, F.R.C.S.

PATIENT, a girl, aged 7.

History: There was much bruising of left eye at birth. Delivery was carried out under chloroform. The mother does not know whether the forceps were used. The eye has always looked smaller than the other.

Present condition: There is left enophthalmos with slight downward and outward strabismus; the ocular movements are good. Left eyeball smaller than right. Transverse diameters of corneæ: Right 12 mm., left 11 mm. There is a large mass of fibrous tissue in vitreous, of roughly conical shape, extending from broad irregular base, covering disk, to a small irregular head at apex just behind the lens. There is a good deal of pigmentation about the base. Direct pupillary light reflex absent, consensual present. The mass of fibrous tissue in the vitreous is probably the result of hæmorrhage due to birth injury, possibly avulsion of the optic nerve.

Bilateral Changes in the Macula with Good Vision.

By J. HERBERT PARSONS.

THIS is a case of peculiar bilateral changes in the macula, in a young man whose vision is practically normal, when the small degree of mixed astigmatism has been corrected. He got into the Army in 1915, and knew of no visual defect when he did so. After being in the Army three years, he was ordered out to Egypt in a draft. For this he was examined in Leeds by Mr. Secker Walker. He found changes in the maculæ and, quite rightly, had him discharged from the Army. The man has not done any work since, in fact he was advised to abstain from work, to wait and see what would happen. I saw him three months ago, and not being sure of the case, I repeated that advice, adding that he should come back for further examination.

Present condition: vision—

Right eye: $\frac{6}{18} - \bar{c}\,\frac{-1{\cdot}5}{+0{\cdot}5\downarrow} = \frac{6}{6}$ Left eye: $\frac{6}{18} - \bar{c}\,\frac{-1{\cdot}25}{+0{\cdot}25\downarrow} = \frac{6}{6}$.

I believe the changes are of long standing, and that in all probability they are caused by colloid bodies in the choroid. We are familiar with the presence of colloid bodies in the choroid, but that condition is most commonly seen in elderly people. Still, they are met with in young people, particularly in association with retinitis pigmentosa and some other conditions. If they are not colloid bodies, I do not know how he could have such extensive changes associated with such slight visual defect. I think the appearances are consistent with the view I have expressed, because there is a hazy shadow round the smaller spots and there are aggregations of pigment, showing the disturbance of retinal pigment epithelium which is characteristic of colloid bodies. The only difficulty lies in the age of the patient, the extensive nature of the changes, and the larger spots at the periphery, some distance away from the macula. But even those have crenated edges, showing they may be the result of an aggregation of these bodies. Mr. Collins suggested that it might be retinitis circinata, but in that condition I think the spots are usually much whiter, and usually, I believe, there is some disturbance of vision.

DISCUSSION.

Mr. E. TREACHER COLLINS: I think the series of cases we have seen this evening are very interesting, because they exhibit the results of hæmorrhage into the fundus of the eye in different positions with subsequent organization into fibrous tissue. I suggest that in Major Carruthers' case there was a birth injury with some hæmorrhage into the vitreous, and that the white mass in front of the optic disk is organized fibrous tissue. I consider that Mr. Jeremy's case was also a birth injury with subretinal hæmorrhage: the retinal vessels are seen to arch over the white mass in the fundus. It is a case of the nature of "Coats's disease"—i.e., a subretinal hæmorrhage, in the vicinity of the disk. I think Mr. McMullen's case is very likely one of birth injury, with hæmorrhage into the vitreous, similar to Major Carruthers', only coming further forward. It is probable that Mr. Parsons' case is one of retinitis circinata, that is, due to hæmorrhages into the retina itself which have organized into white masses around the macula. These white patches seem to be the most extensive changes, and are too large for nodules of colloid degeneration. I agree that at the macula the smaller dots are of that nature. In retinitis circinata such degenerative changes in the pigment epithelium are not uncommonly met with in the centre of the white ring.

The PRESIDENT: I incline to Mr. Parsons' view. Each one of those masses in the retina you could imagine to be made up of an agglomeration of small masses. I think the objection to the retinitis circinata view is, that in all the cases of retinitis circinata I have seen, the essence has been the degeneration of the macula: there is always loss of vision, and generally the macula is very grey in appearance. Moreover, this fundus does not agree in general appearance with retinitis circinata, which nearly always has that so-called "passover bread" appearance,—solid white masses with black holes in them.

Restoration of Socket.

By R. R. CRUISE, F.R.C.S.

THE patient, Lieutenant W., has undergone an operation for restoration of the inner one-third of the socket by the method I described at the last Ophthalmological Society meeting.[1] I have brought the case to show the way in which the conjunctiva has encroached at the expense of the grafted skin, or else the graft skin has been altered, by the constant moisture in the orbit, into tissue of a nature more nearly approaching to that of conjunctiva. That is a point on which I would like to hear the opinion of members. The case shows that a change does take place, and that provided one can keep the cavity stretched and open, by means of a mould held in position in the manner I have demonstrated this evening, a socket with a very satisfactory lining will be the ultimate result.

DISCUSSION.

The PRESIDENT: I think Mr. Cruise deserves our congratulations in connexion with this case: it is a great success. I do not think he laid enough stress, in his notes, on the care he took to excise all scar tissue before inserting the graft. On that point we have got further than we were before the war. To make a proper conjunctival sac is impossible unless you remove the cicatricial tissue.

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[1] *Trans. Ophth. Soc. U.K.*, 1919, xxxix, pp. 118-128.

Mr. R. R. Cruise (in reply): I did not make a point of the importance of removing all scar tissue, because I fully described the procedure when I dealt with the matter six months ago before the Ophthalmological Society. It is surprising how large a cavity one makes for the mould, but it all contracts down later: in fact I do not think you can very well make it too big.

Partial Avulsion of the Disk, with Ectasia.

By W. H. Simpson.

History: Patient, A. S., male, aged 12, was struck in the right eye by a firework on June 27, 1919. On the same day he was admitted to the Royal London Ophthalmic Hospital.

On admission the right eye showed a small wound of the conjunctiva between the limbus and the outer canthus. The pupil was irregularly dilated and was tucked back on the outer side. The lens was clear and dislocated inwards. There was much vitreous hæmorrhage and commotio retinæ, which obscured details of the fundus. He could perceive light with the eye, projection was defective down and in, and the tension was subnormal. The left eye was normal with a vision of $\frac{6}{6}$.

Patient was kept in bed for three weeks with atropine in the right eye.

On July 23, the vitreous had partially cleared and there was no commotio retinæ. The retina was seen to be in position but the disk was obscured by an unusual appearance, details of which, owing to the state of the vitreous, could not be made out till August 2, when a good view was obtained. The disk was then seen to be included in a horizontally oval ectatic area bounded internally by the nasal margin of the disk and extending outwards towards the macula. The area presented a much paler reflex than the rest of the fundus and was sharply defined, except below, by superficial retinal pigmentation. Round the macula there was dense pigmentation and between the disk and macula there was pigmentation and some cicatricial tissue formation. The retinal vessels, on entering the area, showed a decided bend similar to that seen in early glaucomatous cupping and, once in the ectasia, they were out of focus. The walls of the ectasia were steep at the disk, above and below, but towards the macula the slope was more gradual. The ectasia was rather too shallow for accurate measurement; the disk was the deepest part and the vessels there were brought into focus by − 2 dioptres. Below the area there was much retinal degeneration, and, at the periphery, the only abnormality was guttate pigmentation.

The patient could count fingers at 6 in., projection was defective down and in and the tension was subnormal.

Mr. J. HERBERT PARSONS: This case seems to be a partial avulsion of the nerve, only one side of the disk being affected. It was very difficult to be certain there was ectasia. It is not so obvious now as it was at an earlier stage.

Section of Ophthalmology.

President—Mr. W. T. Holmes Spicer, F.R.C.S.

The Need of Ophthalmic Physicians for the Advancement of Ophthalmology.[1]

By Rayner D. Batten, M.D.

The present paper had its origin when a vacancy recently occurred on the staff of our hospital and I urged that what was really required was an ophthalmic *physician*, not an ophthalmic *surgeon*. My arguments in support of this course were met by the answer that it was "not done" at any other hospital, and, secondly, that there were no men available or educated for such a post. I was then challenged to bring the subject forward for open discussion.

Cases requiring operation or direct surgical treatment form such a small part of the whole of our work that we have to be responsible for a far larger clinic than we can personally attend to if it is to yield sufficient operative material for the study and advancement of that branch of our science. In order to cope with this difficulty large numbers of men are educated, given posts as clinical assistants, and entrusted with a large measure of responsibility. We avail ourselves of their services for years, during which they become able ophthalmic observers, but are given no training in ophthalmic surgery and no chance of learning to operate, while their training in, and knowledge of, medicine is insufficient to make them ophthalmic physicians.

Progress on Medical Lines.

Further progress in ophthalmology, as far as one can see, will be mainly on medical lines, and will require men with a physician's training and experience. Progress on surgical lines is limited and can only result from large experience. There may be another and yet another new operation for glaucoma; we can still discuss the best form of cataract extraction; and someone may some day invent a successful operation to cure detachment of the retina or conical cornea, though I doubt if success is likely to come from the surgical side in the treatment of these diseases.

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[1] At a meeting of the Section, held November 5, 1919.

On the medical side, however, there are endless possibilities which we try to tackle with our second-hand scraps of medical knowledge, picked up from such general medical information as may be current, but with no means of studying the problems from a strictly medical standpoint. Of course, we have physicians attached to our ophthalmic hospitals, but their position is that of consulting referees; they do not have patients of their own. To them we refer cases, but the result is not very satisfactory. There is divided responsibility, or more generally the responsibility rests with the surgeon, even in well-marked medical cases, since the eye-symptom is the most obvious factor. This is specially the case in hospital practice. In private practice the responsibility is more often shared, though even there the need of a physician who can grasp the full importance of the ophthalmic problem is acutely felt. In doubtful cases the physician seldom suggests a cause, and, having returned a negative to the question we have asked, the problem ceases to interest him. Where the diagnosis is obvious he shirks treatment, since the eye-symptom is the guiding factor; and where the case is transferred to the physician he is seldom in a position to study it from the ophthalmic point of view. At present our consulting physicians are not appointed for their knowledge of ophthalmology, but for their knowledge in some branch of medicine, generally with a leaning to neurology. We should still require the consulting physician for special cases, but what we require are *ophthalmic physicians*, who do their own ophthalmic work and have to live, so to speak, with their ophthalmic failures, and to study ophthalmic cases from a physician's point of view and with a physician's experience.

SPECIAL PROBLEMS FOR THE PHYSICIAN.

The need of the physician in ophthalmology seems to me so obvious, that I will not weary you with elaborating the point, but will merely mention some of the most noteworthy instances.

(1) The various vascular conditions; variations in blood pressure, arterio-sclerosis, vascular degeneration, thrombosis, and all the allied conditions associated with renal, hepatic, and cardiac diseases.

(2) The diseases of the central nervous system, not only those of more or less obvious central nerve disease—tabes, disseminated sclerosis, cerebral tumours, ocular paralyses—but the many obscure ocular maladies which fall between the surgical and medical sides of ophthalmology; unexplained optic atrophies, headaches of apparently visual origin, but without ocular symptoms as yet within our range of diagnosis.

(3) The many toxic or septic conditions giving rise to definite ocular disease, such as irido-cyclitis, in which the present position is most unsatisfactory. At present we refer the case to the consulting physician, dentist, pathologist, and all the rest, and get a series of negative reports, and the case comes back to us for empirical treatment; whereas it ought to come back to the physician and live with him until he has found a cause. Our training and means of research are totally inadequate to the scientific investigation of medical cases. Toxic conditions, giving rise to iritis, cataract, retinal changes, in diabetes, albuminuria, syphilis, tubercle, septic and other conditions, and toxæmia from drugs, form a list too long to enumerate.

(4) The various nutritional diseases, giving rise to corneal ulcers. These cases I consider peculiarly disgraceful to us, especially the children with recurrent corneal ulcers, which defy local treatment and result in permanent corneal nebulæ.

This classification by no means exhausts the medical side of ophthalmology, but may be sufficient to show how totally inadequate is our education to deal with a very large proportion of our cases. The fact that we are educated as surgeons tends to dull our keenness in and power of investigating the primary causes of certain ophthalmic problems. We are very loth to stray beyond the orbit, and our education is not wide enough to enable us to do so with profit.

CATARACT AND GLAUCOMA; MYOPIC DETACHMENT OF THE RETINA.

Take the case of cataract. Here we know that, in the ultimate result, we can operate to relieve the worst of the symptoms. We look with suspicion on any non-operative system of treating cataract. Most of these systems depend on some local application to the eye, and are not based on any investigation as to the cause. We know too well the fallacies of the results claimed for this or that system of drops "dissolving" cataracts, and we rightly avoid anything that savours so strongly of quackery.

But if we consider the extraordinary variations in the rapidity of progress in some young or middle-aged cases of cataract—how they will start rapidly, and then become quiescent, or even improve—surely there is sufficient evidence that they are due to some constitutional cause beyond our power of recognition as *surgeons*. The causation of cataract requires a more subtle investigation than we can give it.

Take again the glaucomas. I cannot see how any thoughtful man can be satisfied with either the surgical diagnosis or treatment of many of them. A proportion of them are due to anatomical causes, and are relieved or cured by surgical means. But there are a number which defy surgical treatment, and do not exhibit any anatomical cause; yet they progress, their disks show deep cupping without any evidence of increased tension, and we are driven to the supposition that they have increased tension when we are not looking. Some of the acute glaucomas, too, show a virulence for which no anatomical explanation is sufficient. While the actual treatment of the state of hypertension must remain in surgical hands, yet there are many questions in causation and treatment which require to be investigated by the physician. It is not to our credit that, glaucoma having occurred in one eye, we should be so prolific in theories and so ignorant as to its causation that we are unable to prevent its onset in the other, except by operation. Yet we rarely refer our cases of glaucoma to our physician, probably because we know he is not sufficiently familiar with the condition to be of any real help.

For many years I studied myopias, and convinced myself that they were often due to constitutional causes, taking the view that the sclerotic became soft and yielded to normal pressures. I still think so. But I gave up the subject, because I was not enough of a physician to follow it up. Here, too, the fact that we can relieve the symptoms by correcting the error of refraction lulls us into thinking that we are treating the disease; which, however, is far from the case.

Our attitude towards detachment of the retina is even more hopeless. Even if we leave out of consideration the detachments in highly myopic eyes, which, according to some, have an anatomical cause, yet still we have detachments occurring in emmetropes, or in eyes with only a low degree of myopia. A detachment having occurred in one eye, it is more than probable that it will occur in the other. Yet we are incompetent to give any advice, based on knowledge of causation, to prevent such a calamity. Our surgical treatment of these cases is, on the whole, a failure. One per cent. would be a high average of success. Surely we should do well to hand the lot to the physician, and see what he can make of them.

THE EDUCATION OF THE OPHTHALMOLOGIST.

The education of the ophthalmologist is primarily at fault. We insist in the first place that a man must pass the highest examination in general surgery, which it is true gives us an exceptional type of man, but which has practically no bearing on eyes. He wastes years in acquiring the requisite anatomical and surgical knowledge to pass this examination, when his time would be better spent in acquiring some knowledge of medicine and of his own specialty. It is by no means unusual to find amongst ophthalmic surgeons men who not only confess to ignorance of the medical side of ophthalmology, but who seem to glory in their detachment from medicine. At present we act as eyes to the physician, but I hold that the physician should see for himself, and he requires a larger experience than can be derived from studying only special cases.

I do not expect much support from ophthalmic surgeons on the medical side of the question, for the surgeon is an extraordinarily self-satisfied man, and generally thinks himself at least the equal of the physician on the medical side of his art, a position justified, perhaps, by the small amount of help at present given by the physician. On the surgical side, however, I hope to carry conviction.

The actual surgical work of the ophthalmologist forms only a small proportion—not more than 1 or 2 per cent.—of the whole mass of work to be done. As at present arranged, any man going in for "eyes" is supposed to be an "ophthalmic surgeon," and must assume himself, and be assumed to be, capable of doing any ophthalmic operation. Yet what has his training been, and what opportunity has he had of dealing with operative cases? A man may work ten to fifteen years at our leading ophthalmic hospitals and never do a major ophthalmic operation. Yet he is a highly trained ophthalmologist except in operative skill, which he must gain in the school of experience—expensive alike to himself and his patients—if he is ever to get on the staff of a hospital and become an efficient ophthalmic surgeon. In due course he will *have* to undertake his first major operation. He cannot under the present system decline. He is an ophthalmic surgeon, and he must either undertake the operation or put himself into an inferior rank and pass the case on to another.

The war has brought us many "ophthalmic specialists" who are in no way ophthalmic surgeons. In a case known to me one of them came across a case of cataract. He did not feel himself competent and got another man to "help" him with the operation. Unfortunately, he showed but little wisdom in his choice of a helpmeet. He chose a peculiarly incompetent but confident young man, who had never been trusted to do any but the simplest operations. Between them they operated, with the natural result. I have not heard what happened to the other eye.

DIFFICULTIES OF TEACHING OPERATIVE WORK.

The question of teaching operative work is a very difficult one. It would obviously be impossible and undesirable to teach operative work to the large number of men passing through our hospitals seeking ophthalmic training. They cannot all become operators. It would be unjust to the patients and to the hospital. Ophthalmic surgery differs from all other surgery in that there is no gradual road to competence. You cannot allow a man to begin an operation, say, for extraction of cataract and, if he gets into difficulties, step in and set things right. The mischief is done, and men have only two eyes. Instructing men in eye operations is a most painful proceeding, even when they have watched and helped you at previous operations. From the introduction of the speculum to its removal there is often a series of mistakes and some clumsiness, even in such operations as tenotomy, advancement, or excision.

There are many distinguished ophthalmologists who are in no way fitted to be surgeons. They are either innately clumsy, over-confident, or hopelessly nervous and anxious, so that they are quite incapable of doing justice either to themselves or their patients. One of the best ophthalmologists I have known was constitutionally unfit to operate; over-conscientious, he was always unhappy at his operations, and could never do himself justice; his failures caused him acute distress and sleepless nights. Yet there was no way out for him.

It would, I think, be better for the profession if ophthalmic operations were in fewer hands. Such an arrangement would tend not only to a higher standard of operative skill, but to enterprise and progress in surgical treatment. The surgeon, being no longer required to treat cases essentially medical, would be free to devote himself with a single eye to the advancement and perfection of surgical methods, and would have in addition increased opportunity for their practice. Surgeon and patient alike would benefit. It would then be possible to have private ophthalmic hospitals, accustomed to and equipped for ophthalmic operations, instead of being dependent upon nursing-homes, where eye operations are infrequent and the nursing and equipment unsatisfactory. The present position is not fair to the public, and is not to the honour of ophthalmic surgery, nor to its profit. It would be far better for all concerned if we recognized ophthalmic physicians as well as ophthalmic surgeons.

CONCLUSION.

My remedies for the present conditions would be:—

(1) To improve ophthalmic education.

(2) To appoint ophthalmic physicians to our hospitals.

In place of the Fellowship I would substitute an ophthalmic qualification, and it should be possible to qualify medically or surgically. The surgical qualification would require evidence of operative skill. Even for an ordinary medical qualification, a man is required to have attended his twenty or thirty cases of midwifery. Is it too much to ask that he should show evidence of some operative skill in ophthalmic surgery before he is admitted as an ophthalmic surgeon?

These changes would take time. But the recognition of ophthalmic physicians and their appointment to hospitals could be effected at once. They should attend out-patients and have beds at their disposal. They should see all classes of cases, but should be relieved of all operative work. The surgeons, on the other hand, would refer their medical cases to the physician. This would provide a larger number of ophthalmic men to deal with the ever-increasing mass of ophthalmic work.

This system would, I believe, be of advantage both to the physicians and surgeons, both in hospital and private work, and would redound to the credit and advancement of ophthalmology.

(The discussion was adjourned to the meeting of December 3, 1919.)

Section of Ophthalmology.

President—W. T. Holmes Spicer, F.R.C.S.

Discussion on Mr. Rayner D. Batten's paper, "The Need of Ophthalmic Physicians for the Advancement of Ophthalmology.[1]

Dr. C. O. HAWTHORNE: As the Section has heard what a surgeon has to say, it may be convenient that a physician should now intervene. And let me say, in the first place, I am quite sure that all those members who were present at the November meeting will agree that we had then an opportunity of listening to a paper which was as interesting as it was unconventional, and even those who do not allow Mr. Rayner Batten's conclusions will recognize with me the skill and sincerity of his argument. The occasion presented two other features which, I think, are noteworthy. One of these was provided by the element of surprise or novelty, and the other by the element of courage. It is an unusual thing—in my experience an unprecedented thing—to see a representative of the craft of surgery voluntarily clothing himself in sackcloth, putting ashes on his head, and beating his breast while exclaiming in pathetic accents, "Woe is me, for I am a very much overrated man!" And it is equally unusual to hear, from the surgical quarter, and addressed to the physicians, the plaintive Macedonian appeal, "Come over and help us." I am sure the physicians will appreciate the compliment which is paid to them by Mr. Batten, and they will, so far as possible, give to his paper and to his arguments a most attentive hearing. The element of courage perhaps appeals to me particularly from certain experiences of my own. It happens that, on more than one occasion, I have ventured to say that a considerable part of the field of clinical ophthalmology requires, not the art and craft of the surgeon but the diagnostic and therapeutic methods of the physician; and I have been soundly rated for my pains. I once advanced this proposition at a congress of ophthalmic surgeons assembled at that home of lost causes, the University of Oxford, and in the subsequent discussion I was, metaphorically, torn limb from limb. And still later, I have been publicly rebuked for presumption in a lecture published in a medical journal by an eminent ophthalmic surgeon who occupies a high place in the hierarchy of this Section of Ophthalmology. If these things may happen to a physician, who by the majority of the members of this Section is probably regarded as without the pale, what fate is likely to be reserved for an ophthalmic surgeon who, making similar proposals, is sure to hear from his colleagues the cry "Our foes are they of our own household." It was for these reasons that, when I saw the title of Mr. Batten's paper, though I had not the advantage of knowing him personally, I felt convinced he was a man endowed with an unusual measure of moral courage. And his paper, in my judgment, fully justified this anticipation. With the general spirit and motive of the paper I am in substantial agreement, though I am afraid I cannot say the same of the specific names and titles which Mr. Batten proposes to attach to the officers whom he hopes to call into existence. The true position in this matter may be efficiently illustrated by a remark which is attributed to Mr. Gladstone, who, when he was told that certain members of his party were calling themselves Gladstonian Liberals, replied, "Well, I at least am not one of them." The moral, of course, is that the physician is content with the liberal and catholic title which a long tradition has bestowed upon him, and he does not propose either to allow this title to be limited or qualified in one direction, or to be tricked out with artificial decorations in the other. It is true that there are members of the profession who call themselves, or allow themselves to be called, such names as neurologists, cardiologists, enterologists, endocrinologists, and the rest. But such a proceeding, in my judgment, is neither dignified nor scientific, for the relation of the physician is not to localized disturbances, as these occur in a certain limited and selected portion of the body, but to all the disorders and discomforts of the whole and undivided individual patient. For my own part, therefore, I should object to be called an ophthalmic physician for the same reason that I should object to being styled a non-ophthalmic physician. The one term is a redundancy while the other borders on the grotesque.

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[1] At a meeting of the Section, held December 3, 1919.

Coming now to the essential substance of Mr. Batten's paper, this may, I take it, be described as a critical review of the work of the ophthalmic hospital. Mr. Batten, who is in a position to speak with authority on such a question, has weighed this work in the balance, has in certain respects found it wanting, and he now proposes certain measures with a view to improve and to redress the situation. I say again that, in principle, I am entirely of his opinion. Whether a co-operation of the physician with the surgeon in the investigation of such quasi-surgical problems as the cause and prevention of conical cornea, of detachment of the retina, of cataract, and so on, would be fruitful or not, I cannot pretend to say. But at least it is worth trying; and until the whole body has been thoroughly and repeatedly examined it is anything but scientific to say that the origin of this or that local disturbance cannot be detected. While I speak with hesitation upon such problems as Mr. Batten has proposed for the joint investigation of physician and surgeon, I do not speak with uncertainty about some other conditions which are constantly to be met with at ophthalmic hospitals, and which, though regarded by the patient as having a purely local significance in reference to some defect of sight, are essentially evidences of nervous disease, of cardio-vascular disease, of kidney disease, or of diseases of the blood, that is, these conditions are symptoms of diseases with which the physician, not the surgeon, is constantly occupied. My reference to symptoms includes ocular paralyses, disturbances of the pupil, cycloplegias, optic neuritis, optic atrophy, retinitis, retinal and other intra-ocular hæmorrhages, and disturbances in any form of the field of vision. I say that each one of these symptoms creates an immediate claim for a complete investigation of the patient, that is a demand for the application to this particular individual of the method and practice of the physician. It is a mere accident, due to the natural prominence which ocular or visual symptoms occupy in the consciousness of the patient, that he first seeks aid at the ophthalmic hospital. The true clinical position of such a patient is not determined by the local disturbance in the visual apparatus but by the essential nature of the disease which underlies it. Therefore, quite clearly, such patients fall without the limits of ophthalmic surgery, and this all the more emphatically so, seeing that neither the visual nor the general condition is capable of remedy by mechanical or surgical means. It comes, therefore, to this: that if you are to receive, examine and treat patients of this order at ophthalmic hospitals, it is necessary that the hospitals have towards them some definite policy. If such diseases as I have mentioned are to be treated at an ophthalmic hospital, the hospital obviously must have an appropriately qualified medical staff, nurses in suitable supply and properly trained, the equipment and apparatus needed for medical investigation, and beds for patients who require to be kept under continuous observation and care.

This is what I conceive Mr. Batten is asking for, and I cannot see how his position can possibly be challenged. The question is one which concerns the good repute of the profession, the progress of scientific medicine, and the welfare of the individual patient. The position seems to me a particularly clear and distinct one, and I do not see how it can be adequately and scientifically met except by the adoption of the principle which Mr. Batten has expressed in his paper. There is only one alternative, namely, that patients suffering from nervous, cardiovascular, renal and blood diseases shall not be retained in institutions which profess the practice of ophthalmic surgery, but shall be directed to the medical department of a general hospital.

Mr. CHARLES HIGGENS: Two things struck me about Mr. Batten's paper. He said no word about refraction. After forty years of practice I feel that if it had not been for refraction work I should have had nothing to do. The second point is, that he has left the general practitioner out of consideration. If the general practitioner sends us a case for an opinion we investigate it and tell him what ought to be done, and, if it is necessary for the patient to consult a different specialist, we indicate who he should be, whether for brain or any other disease. And in hospital work, we turn the cases over to the appropriate physician. We do not pretend to know all these things in an expert way, and we send patients to the proper men.

Mr. ERNEST CLARKE: London is the only place in the world where oculists are supposed to be surgeons only, and are styled "Mr." In any other specialty in London—gynæcology, laryngology, dermatology, &c.—the specialist may be either physician, surgeon, or both. How did this custom arise? It is surely not far to seek. Only sixty or seventy years ago, eye work was entirely surgical—there was no ophthalmoscope, practically no refraction work, and the only eye work doing was surgical, and at all our London hospitals one of the surgeons had charge of the ophthalmic department. A little later specialists were appointed, but it is only quite recently that one of the larger hospitals—viz., the London Hospital—started having a special ophthalmic surgeon! I need only refer to such names as Hutchinson, Waren Tay, Eve, Cooper, Hulke, George Lawson, and Silcock, all of them general surgeons practising ophthalmic surgery. The young men aiming at becoming oculists naturally imitated them; they saw it was the custom for *surgeons* to be appointed, and that the possession of the Fellowship of the Royal College of Surgeons was a *sine quâ non*. This meant that the student had to put forth all his energies to pass this difficult examination, and medicine was put on the shelf. Some of us have had the priceless experience of spending some years in general practice, and some have taken the Doctorate of Medicine, and that has enabled us to get out of the narrow groove. I applaud Mr. Batten for having brought forward this subject, and the discussion is most important, but I do not agree with his remedy. I think the remedy is, that the ophthalmic student of the future should be invited, nay urged, to learn more medicine. As Mr. Higgens has said, we do not want to treat cases of general disease, but we do want to be able to diagnose them so that we can transfer them to the physician. I do not understand Dr. Hawthorne's attitude, for he has been a most useful physician to my hospital for many years, and we have always referred our medical cases to him for treatment. The remedy is to get a little more broad-minded in our views, to get away from too much surgery, because nowadays nine-tenths of ophthalmic work is medical, including practically all refraction work. Mr. Batten said only 2 per cent. of the work is surgical. I think it is a little more than that, but certainly the major part of ophthalmic work is medical. We must urge upon students in the future to study medicine more, and to take positions as house-physicians as well as house-surgeons, and I think in this way we shall achieve the remedy.

Mr. E. TREACHER COLLINS: The picture which Mr. Batten has drawn for us of the present position of ophthalmology is not, I think, a very pleasing one or one quite true to nature: I think that in places he has got his colours rather mixed, and in others he has deepened the shadows unduly for the sake of effect. If we want to understand the position we are in at present and realize that to which we are tending in the future we require to consider how we got where we are now. The commencement of British ophthalmology practically dates back to the establishment of Moorfields Hospital in 1804, and Mr. Batten may be interested to hear that when it was originally established the staff consisted of a physician and a surgeon. Dr. Farr was appointed physician, and for fifty-four years he held that post. I have heard Sir Jonathan Hutchinson relate that when he first began to visit Moorfields Hospital Dr. Farr attended and saw cases just as surgeons now see cases. How some patients were allotted to the physician and some to the surgeon, I do not know: probably, in the matter of treatment, the physician superintended the sweating and purging, and the surgeon looked after the bleeding and blistering! At any rate, blood flowed freely at Moorfields in those early days. As time advanced, the surgical side at Moorfields seemed to progress more rapidly than the medical side, for we find that the surgeons increased in number quickly, and the physicians gradually ceased any regular attendance at the hospital. In those days all the surgeons at Moorfields were also general surgeons: they practised ophthalmic surgery as a branch of general surgery. When I first went to Moorfields, half the staff was composed of general surgeons who practised ophthalmic surgery, and half who were ophthalmic specialists. After the introduction of antiseptics, the range of surgery so greatly increased that general surgeons ceased to devote themselves to ophthalmic surgery. After the invention of the ophthalmoscope and the establishment of the correct methods of estimating refractive errors, the realms of ophthalmology were greatly increased, and a new type of man evolved, the ophthalmologist. For him the ophthalmoscope opened up the study of many diseases which had formerly come within the range of the physician. Those who hold posts of ophthalmic surgeons to general hospitals know what a large part of their time is spent in the medical wards helping the pure physician, who has not troubled to learn the use of the ophthalmoscope in the diagnosis of his cases.

This question which we are debating is largely one of names. It reminds me of the story of a man who was a member of the Royal College of Surgeons and a licentiate of the Royal College of Physicians. One of his lady patients said to him: "I never know which to call you, 'Mr.' or 'Doctor,'" and he replied: "It does not matter which you call me, so long as you call me in." So also it does not matter what we are called so long as we are called in whenever a disease of the eye is to be dealt with. In the eighteenth century, the profession was divided into physicians, surgeons and apothecaries. Hospital staffs are still nominally divided into physicians and surgeons, yet we find that such a division hopelessly breaks down. We see on hospital staffs men who are called obstetric physicians who do as many operations as the general surgeons. And in some hospitals in London we find men in charge of nose and throat departments who are called physicians, and in others men who are called surgeons both doing precisely the same kind of work. As knowledge advances, so it tends to be split up into several different departments. In medicine we have departments of ophthalmology, gynæcology, dermatology, rhinology, &c. The men who confine their practice to those departments are regarded as experts in them. But there are border lines to all departments, border lines which are liable to be overlooked by the experts; so it is an advantage to have men who take up two branches, and form a *liaison* between the two, becoming experts in both. They do a lot of useful work, but such men often do not find all the favour they deserve among those who are experts in only one branch. It is related of Sir John Lubbock that when he was in the City he was looked upon as a great politician and a great man of science ; when he was in the House of Commons he was regarded as a great financier and a great scientist : but when he was at the Royal Society he was regarded as a great politician and a great financier. Though I do not agree with all of Mr. Batten's paper, I agree with its title, so far as it goes—" that physicians are necessary for the further advance of ophthalmology "—but I do not think it goes far enough. There is also need for ophthalmic rhinologists, for ophthalmic physiologists, for ophthalmic pathologists, and for ophthalmic mathematicians. All these and other workers on the boundary line of ophthalmology are equally as important as the ophthalmic physician. But what is most needed for the further advancement of ophthalmology is the complete ophthalmologist. By the complete ophthalmologist I mean the man who is greedy to know everything which can be known about any disease in which the eye is affected : and not only that, but eager to practise and carry out any form of treatment which is likely to preserve or restore vision.

Mr. A. W. ORMOND : It appears to me that at the present time one of Mr. Batten's propositions has not received attention, and that is, the qualification which is necessary for the position, in London, of ophthalmic specialist. For a man to hold a post at Moorfields and various other eye hospitals, it is necessary for a man to have a surgical qualification, and that qualification is the Fellowship of the College of Surgeons. I think the contention of Mr. Batten may be put in this way : that the London M.D. is as good a qualification as is the London M.S. for an ophthalmic specialist to possess, and I think that point is one which should not be lost sight of. I take it Mr. Batten is as anxious to push forward the advancement of ophthalmology as Mr. Collins, and he believes we should remove the disability from those who possess the medical qualification only. There is another point in Mr. Batten's paper which I am not clear about. He advocates that there should be on the staff of the London ophthalmic hospitals an ophthalmic physician who shall do no operations at all, and an ophthalmic surgeon who should do all the operations. That would, it seems to me, be a pity, because it would be making a specialty within a specialty, thus further limiting that specialism which has many dangers already. To my mind, the great need at the present time is that the specialist should have a wider, not a narrower view. With regard to Dr. Hawthorne's contention, and his humorous references to want of knowledge of medicine, his remarks remind me of the cases which have been sent to physicians for their help, but help has not been forthcoming. It is not that we doubt physicians know so much more about medicine than we do, but so often, when we are at a loss for information, we gain none from the medical side. I am sure the need at the present time is not so much for an alteration of our organization as for obtaining, as far as possible, a much greater number of men who have far more time to study the medical side of ophthalmology than is possible at present, when most of our junior men are absolutely inundated with refraction work.

Mr. LEIGHTON DAVIES: One of the glories of our specialty is, that it requires such a wide breadth of view. We are both physicians and surgeons, although we may be called surgeons. I do not think it is right to tack on to ophthalmic surgeons the opprobrium that they are incompetent to investigate any branch of scientific work connected with the eye: I fail to see why the ophthalmic surgeon should not be as well able to investigate disease as is the physician. I regard it as a retrograde step to try to divide this specialty into two: it would greatly confuse the issues which lie before it. That brings me to another point in the paper—the training of ophthalmic physicians and surgeons. At present it is a great drawback to all of us in that when we first come into the ophthalmic world we have to learn our work after we become qualified. There is some advantage in that, however, for it is a great mistake for a man to make up his mind at 21 years of age that he is going to be an ophthalmic surgeon and devote himself to it from that time for the rest of his career. It tends to make him narrow-minded. I am really speaking against the granting of diplomas in ophthalmology. The training for this diploma would teach a man all he requires to know about ophthalmology, but it teaches him nothing else about either medicine or surgery. I think the universities should grant their highest degrees, either medical or surgical, in ophthalmology, such as, I am glad to see, the University of London is doing now, because it ensures that the man who is taking his M.D. or M.S. in ophthalmology has had a thorough grounding for his career. Therefore I think the solution is largely in the universities making ophthalmology a special subject for higher degrees. The University of Wales has not yet granted this degree, but I think it will come up for discussion next year.

Mr. HERBERT PARSONS: It is difficult not to agree with a great deal that everybody has said. The chief value of Mr. Batten's paper consists in the stress which he has laid on the feeling many ophthalmologists must have that they are not fully qualified to deal with the medical aspects of their subject in the same degree as the surgical. But I think he might have gone very much further. I think the ophthalmologist ought not only to be able to deal exhaustively with the medical aspect, but with other aspects, such as optics, and physiological optics, and the industrial relationships of ophthalmology; in fact, to deal with ophthalmology from every point of view. I have had the misfortune—it may be—to be much mixed up with various industrial and other aspects of the subject: the fixing of standards for different classes of people, such as soldiers and sailors, the inspection of factories from the lighting point of view, and so forth. And all I can say is, that one feels, as Mr. Balfour said, "like a child in these things," for one feels that the problems are very complex, and very unfamiliar in many of their bearings. The importance to be attached to one's contribution to the discussion of any of these topics depends upon a comprehensive and thorough grasp of the subject. And that brings one round to Mr. Collins's position, which is the nucleus of the whole matter—namely, that we are essentially ophthalmologists, neither specially physicians nor specially surgeons. Therefore the ideal of the ophthalmologist is to make himself *au fait*, as far as possible, with ophthalmology in all its branches. It must be admitted that it is not possible for any of us to reach the highest ideal: some are more suited for surgery and others for medicine, but I think that that sort of thing rights itself as in other walks of life. We should at any rate strive to reach the ideal in regard to our own branch of the profession. The importance of the paper is that it puts the finger on the weak spots, and the lesson of it is to see how we can improve the conditions. I think this reduces itself to the question of the education of the ophthalmologist. What we need in London is a first-class school of ophthalmology: I think that will be the solution of most of our difficulties. And to attain to that better position we shall have to study all the branches of our subject to the fullest degree, and the opportunities for such study must be increased: they are faulty in many directions at the present time. The optician who makes spectacles knows more about optics than we do. But it is not right that it should be so. What the optician knows about it is easily learned. Where we are lacking is, that we content ourselves with getting these things up purely theoretically without handling them practically and seeing exactly how they are done. And the same applies to physiological optics. For instance, at the present time I am on a cinema committee, and one of the important factors there is the question of recurrent images. It is a physiological question which most ophthalmologists do not spend much time in trying to grasp. The only way is deliberately to attack such subjects. At present we have to study them under grave disadvantages. I do not think there is any place in London where a man can get a thorough course of physiological optics, unless it be at the Psychological Laboratory, University College; but we want courses catering specially for the training of ophthalmologists. Associated with the education question is the qualification of ophthalmologists. The Council of British Ophthalmologists have done their best to point out the way in which ophthalmic qualifications may be improved, and, as published in the last number of the *British Journal of Ophthalmology*, they have come to the conclusion that special diplomas in ophthalmology are not altogether satisfactory, but that the qualification should be made part of a general medical qualifying examination of the higher order, such as the M.S. London. Perhaps that does not reach the ideal, but so long as ophthalmic hospitals require surgeons specially that diploma will be primarily a surgical qualification. And if we can get the higher surgical qualifications to include ophthalmologists, so much the better. Probably we can improve matters still more later.

But what I do object to in Mr. Batten's paper are his conclusions. It would be a lamentable thing to split up ophthalmology into two specialties.

All advance in knowledge has been the result of specialization. Every thought we have is a form of specialization, attention itself is a form of specialization; but it must not be forgotten that specialism is responsible for great evils. Specialism has been the ruling tendency of the last century, and the result is that advance in knowledge has been greatest in those sciences which are easiest to grasp. Physical sciences do not appear to be easiest to us, because we are more of a biological turn of mind, but the exactitude of the mathematical and physical sciences renders them easier to certain types of mind. Hence the advance in physical science has been greater than in biological science, and still more than in the higher aspects of mental and moral science. One result has been the engendering of materialism, which has been a prime factor in the causation of the war. Specialism has led to many of the difficulties which have arisen during the war—difficulties of Government departments—and the muddles which have been made have not been because there were not men trained to the different jobs, but because the different men have not been properly co-ordinated. What we want above all now is integration and co-ordination, as opposed to differentiation and increased specialism. We have, therefore, to guard against further dichotomy in ophthalmology, such as the reader of the paper advocates.

Dr. COBBLEDICK: I think Mr. Batten's paper is a move in the right direction, but he should be prepared to go further. He wishes to divide ophthalmic staffs into surgeons and physicians but I think there should also be elected a special refractionist. The question then arises, who is going to apportion the cases to these different sections? This is work that will have to be fairly, accurately and rapidly carried out. The best scheme is undoubtedly a thoroughgoing team work which proved to be such a success in the Army during the war and was practised in some parts of America before 1914. Under this system the ophthalmic surgeon must sink his preconceived ideas, every section of the body must be examined and reported on, and these reports must receive the considered judgment of the ophthalmic surgeon. I am inclined to sympathize with the suggested ophthalmic physician, for how can he report on the condition, e.g., of the post-nasal space or the vesiculæ seminales. The only type of medical man who could report—from practical experience—on the various parts of the body is the general practitioner: he is in the habit of examining the rectum, throat, vagina, chest; has a wide knowledge of children's diseases, but unfortunately his knowledge of eye work is very limited. In this connexion it is interesting to note that the Medical Research Committee, at the instigation of such men as Sir James Mackenzie and Sir Clifford Allbutt, has enlisted the services of medical practitioners to investigate on the early symptoms and diagnosis of disease. All this tends to show there is a movement to obtain in all medical circles a greater general knowledge. I have also noted that the remarks of previous speakers that the F.R.C.S.Eng. which we have all so much worshipped is tottering on its pedestal; it should be conjoined and should work with the M.D., then will the maximum benefit accrue to ophthalmology.

Mr. RAYNER BATTEN (in reply): A point which has not been touched on in the discussion is what I have called the "dilution" of the operating surgeons. With the large number of men necessary to do the ophthalmic work of the country, it is impossible for all to be sufficiently trained to operate. It is lamentable that eyes should be so badly operated on as they must be when a man has only an occasional operation to do. He cannot do an *occasional* operation successfully. The physician need not operate. I do not want to introduce any specialism between physicians and surgeons. Both are ophthalmologists. Both should undertake refractions. But there should be a recognized position in the hospital for the man who does not operate, and yet is qualified in every other respect to be an ophthalmologist. I agree that education is the key to the position, and that is the real point of my paper. But, in the meantime, the opening of ophthalmic hospital appointments to physicians would be a step in the right direction. My desire is merely to insist that men who are not competent to operate should not operate, and that medical work should be in the hands of competent physicians.

Case of Keratitis following Gunpowder Explosion.

By GEORGE POTTS, F.R.C.S.Ed.

T. B., AGED 14½. On September 5, 1919, he met with an accident from the explosion of gunpowder and the same evening was brought to hospital.

On examination it was found that his face was literally covered with charred powder and was scorched about the lips, nose, cheeks, eyebrows and lids. The whole of the palpebral fissures were full of half-exploded gunpowder. It was impossible to distinguish any conjunctiva or cornea. The lids and eyes were absolutely covered by a dense layer of semi-exploded gunpowder which was adherent to the conjunctivæ and corneal epithelium, the left being more severe than the right.

His eyes were cocainized and the powder removed from the corneæ, conjunctivæ, lids and portion of the face. It was found to have burnt and penetrated the superficial layers of the cornea and Bowman's membrane on the left side, and on the right side the corneal epithelium and Bowman's membrane only. There was very little clear cornea on either side remaining and iritis had already commenced. Both eyes were well bathed with boracic lotion and atropine drops instilled, also ung. iodof. and cocaine applied.

He has continued to improve steadily from the first.

Present condition: Right eye—There is a very slight nebula of the cornea which amounts to a very fine film mostly to the nasal side, but with little or no interference with vision. Right vision, $\frac{6}{6}$. There has been some slight injection of the conjunctiva due in all probability to the condition of the weather and desire to use the eye. Left eye—There is a denser and thicker nebulous opacity of the cornea amounting to a leucoma. This is clearing under treatment with dionin thrice weekly. Left vision, $\frac{1}{60}$. The vision of this eye was defective prior to the accident. The face, nose, lips and lids—It will be seen that there is practically little or no evidence of the accident remaining in the structures.

Present treatment: Right eye—Lotio ac. boracis, ung. flav. dil., massage. Left eye—Ung. flav. co. dil., dionin thrice weekly.

Suggestions as to any further treatment to clear the left corneal opacities are invited. Also as regards the prospect of their clearing altogether, having in view the patient's age—viz., 14½ years.

Detachment of the Retina with unusual condition of the Conjunctiva.

By George Potts, F.R.C.S.Ed.

J. P., aged 47 years.

History: Contracted rheumatism in the spring of 1900, whilst on service in South African Campaign. Has had rheumatic attacks frequently since then. His eye trouble dates from April, 1914, when he first complained of symptoms of iritis in the left eye. He has had attacks of iritis on the following occasions: Left eye, April to June, 1914; right eye, November to December, 1916; right eye, April 14 to 29, 1917; right eye, January 16 to 23, 1918; right eye, November 13 to 23, 1918; left eye, June 23 to July 29, 1919.

Patient was admitted to the Kent County Ophthalmic Hospital with iritis in the left eye on June 23, 1919. The right conjunctiva was thickened and swollen, and the sclerotic œdematous, thickened and had a fatty appearance. The subconjunctival veins were somewhat dilated. The right pupil was tied down by posterior synechiæ but dilated irregularly under atropine. There was a detachment of the lower portion of the right retina. The left pupil was also tied down by posterior synechiæ but dilated irregularly under atropine.

Right vision = $\frac{3}{60}$ – 2·5D. sph., – 1D. cyl. ax. horiz. = $\frac{6}{18}$. Left vision = $\frac{6}{12}$ – 0·5D. sph., – 1D. sph. ax. horiz. = $\frac{6}{9}$.

He was placed on pot. iod. 10 gr. t.d.s. This caused some colic with symptoms of neuritis in the calves of the legs.

On November 13, 1919, iritis again appeared in the right eye, and the fundus was most difficult to be made out. There was a distinct hazy appearance of the lens. The pupil was contracted although atropine had been instilled thrice daily.

Family history: Good. There were ten in the family, eight living. One brother died of pleurisy, aged 16. One sister died at child-birth. Mother healthy but had an attack of sciatica many years ago. Father died of valvular disease of the heart.

States that he did not notice his vision failing until 1918, when the first symptoms were floating opacities in the right eye. These then developed into the appearance of a detachment. He noticed that he was not able to observe objects in the upper portion of the right field. The thickening of the conjunctiva was first noticed about eighteen months ago. This commenced as a few "bubbles" which coalesced and subsequently surrounded the cornea.

Opinions as to the nature of the conjunctival condition and treatment are invited.

The patient is a schoolmaster and there is no history of any injury, nor of specific disease.

DISCUSSION.

Mr. A. HUGH THOMPSON: I think that in Mr. Potts's second case there was a distinct rise of tension, also proptosis, and, taken in conjunction with the detachment of the retina, this seems to point to the presence of a new growth. The iritis and the keratitis punctata suggest that its nature is tubercular.

The PRESIDENT: Has Mr. Potts had a Wassermann test done? This case reminds me of some which were shown here a long time ago by Mr. Donald Gunn. They were called syphilitic infiltration of the conjunctiva: it would be interesting to ascertain whether there is syphilis in this patient.

Mr. POTTS: The suggestion is a very good one, but I have not had the Wassermann done.

Postscript.—Harrison's Wassermann test was done on December 9, 1919. Result, doses of complement and deviation 3, 6, 9, negative.

Case of "Gassing" followed by Tubercular Iritis.

By George Potts, F.R.C.S.Ed.

The patient, E. R. D., is aged 23, and has been in the Army since he was aged 16. On November 4, 1918, he was "gassed" and reported sick and was admitted to hospital (No. 5 General Hospital, Rouen) on November 28, 1918. He first noticed his eye trouble about this time, and three weeks after he states he became almost totally blind in the left eye. He had been in France for four years and his Medical Officer stated he was run down and ordered a month's rest in bed. Was transferred to No. 4 General Hospital, London, where he had an operation on the nose (?). States he had something removed from nose and washed out with needle and Higginson's syringe.

Family history: There is a family of six all alive and well. Father and mother alive and quite healthy.

On June 7, 1918, he first came under my care. There was definite evidence of kerato-iritis in the left eye and he was immediately placed under atropine. On June 14 the left eye showed keratitis punctata and synechiæ (posterior). He was admitted to the Kent County Ophthalmic Hospital and treated with pot. iod., ung. hydrarg., gutt. atrop. and dionin.

Discharged June 26, eye quieter.

August 7, 1919: There had appeared on the left iris, outer pupillary margin, two small nodules brown in colour and flocculent in appearance. The right iris also had one nodule similar in appearance but on the inner pupillary margin. These have continued and do not appear to be affected by antispecific remedies. Four Wassermann tests were done, one at Rouen and three in hospital in London which proved negative, also two tubercle bacilli tests negative.

On August 15, 1919, he was again admitted to hospital and Harrison's Wassermann test done; result, doses of complement 2, deviation 3, 6, 9, negative. A tubercle bacilli test was done also which proved negative.

Present condition: Right eye—Some keratitis punctata and posterior synechiæ are present. On the pupillary margin of the iris about "4 o'clock" there is a raised brownish nodule. There is no pain in the eye but he has some photophobia. Vision $\frac{6}{9}$. Left eye—Cornea in the lower quadrant is hazy and the epithelium slightly stripped. Keratitis punctata, iris muddy, posterior synechiæ. On the pupillary margin of the iris at about "4 o'clock" there are two brownish nodules which have the appearance of breaking down. There is some evidence of irido-cyclitis. The eye has been very painful during the past few weeks, particularly over the left eyebrow. Vision bare P.L. General health: There is a slight accentuation of the second sound over the aortic area, otherwise the heart is normal. Auscultation reveals no evidence of lung trouble. X-ray shows some slight enlargement of the mediastinal glands. There is a discrete papular rash on chest and back which he states was present before being gassed. The arms are not affected. This rash appears and disappears without apparent cause.

Suggestions as to further treatment are invited.

Present treatment: Atropine, ung. guaiacol to temples, mist. pot. iod. gr. 10, t.d.s.

I am indebted to Dr. D. M. Mackenzie, House Surgeon K.C.O.H., for the notes and X-ray photograph.

Aneurismal Varix of Cavernous Sinus.

By M. L. Hepburn, M.D.

History of case: C. P., aged 25, while on active service in August, 1915, was kicked by a horse on the right side of the face, this fracturing the jaw and causing an extensive laceration of the skin over it. He was unconscious for twenty-four hours afterwards. In November, 1915, the right eye began to protrude, the protrusion being accompanied by a noise in the head, and the vision in this eye became defective. In January, 1916, the common carotid was tied, and as a result of this operation the sight in the eye returned.

At the present time: Right vision, $\frac{6}{6}$ Hm. + 0·5 sph. Left vision, $\frac{6}{6}$ Hm. + 0·5 sph. He has proptosis of the right eye directly forwards with marked dilatation of the vessels of the conjunctiva, and also of the supra-orbital vein. There is no chemosis, and no limitation of movement. A well marked bruit is heard on application of stethoscope to the forehead. The retinal vessels on the right side are also dilated.

I have brought forward this case in order to learn whether I should do anything more in the way of treatment. He has had his common carotid tied. At present his vision is $\frac{6}{6}$ in the proptosed eye, but he is troubled with noises in the head, and this is the only inconvenience he suffers. Cases of this kind have been shown here on previous occasions, and some have expressed the opinion that tying the angular vein, or other prominent vein, does as much good as tying the common carotid. I feel sceptical about that but this man has a dilated supra-orbital vein, and I wondered whether I ought to do anything to that. My own opinion is that I should advise against any further operative procedure, but I shall be guided by what is said here.

DISCUSSION.

Mr. CHARLES HIGGENS: These cases are very rare, and they are interesting. In the course of my experience I have come across only two of them. They are what Mr. Nunneley used to call vascular protrusion of the eyeball. Over thirty years ago I saw the case of a young fellow who had been to a picnic, and a soda-water bottle burst and some of the glass entered the inner angle of the orbit. He had every symptom of varix-pulsation in his head, and so on. I did not do anything to it myself, but wrote to Mr. Lansdowne, who was in practice in the West of England, and he reported later that he had cut down and found a lump at the seat of injury, that he tied certain vessels which were communicating with it, and the patient was completely cured. I think this case must be unique. I have myself tied the carotid in a case of vascular protrusion. The woman got hemiplegia, but recovered entirely, and never had a recurrence of the head noises.

Mr. PERCY FLEMMING: I have seen five or six of these cases, and perhaps one's experience of them may help a little in regard to this one. In connexion with the last case I had, the suggestion was made that the venous mass—it is not a single vein—should be dissected out, and this Mr. Raymond Johnson undertook for me. He first exposed the common carotid, and put a ligature on it without tying it, and then tried to dissect out the venous mass. The hæmorrhage was very severe. The carotid was then tied, and, with great difficulty, the hæmorrhage was controlled without further attempting to get rid of the mass. The patient had a severe time immediately afterwards and we were anxious about his life. He eventually left the hospital much in the same condition as before the operation. Subsequently he went to South Africa, and recovered completely. From what I saw of that operation, I should be chary about attacking a like mass in the orbit. Spontaneous cure may result in these cases. An old lady patient of mine with this condition was desperately ill. She had pelvic trouble, and operation was out of the question. She could have every comfort and was well taken care of. All her symptoms subsided in eighteen months. Lately, however, she has become hemiplegic. There is always, in these cases, a history of a fall or other injury; possibly the rupture of a vessel in the first event, the fall being the sequel. Another case I remember was that of a man who came to Moorfields with hemianopia due to a fall on the back of his head. At the time he had paralysis of one hypoglossal. That was in September. He came again in the following March with the symptoms of proptosis, pulsation, and a murmur. That is the longest interval I know between the fall and the onset of the symptoms. He was about 60 years of age. His common carotid was tied, and he was nursed very carefully, as there was the possibility of brain trouble afterwards. Notwithstanding this he had an attack of right hemiplegia with aphasia two days after being allowed out of bed. That was five or six years ago. All the ocular symptoms have now subsided, and he can make himself understood. In this present case I would advise Mr. Hepburn to wait, and do no more at present.

Mr. J. H. FISHER : Mr. Flemming's experiences are of considerable interest and importance. We need, in these instances, to differentiate between two kinds of cases: those in which a fracture of the base lacerates the common carotid artery and opens directly into the cavernous sinus, and those other cases in which there is rupture of an atheromatous artery in that situation, causing the patient to fall in a state of insensibility. Mr. Hepburn's case, I think, belongs to the class of direct injury from fracture of the base, the man having received a kick from a horse. It might make a difference to the line of treatment followed as to which category a given case belongs to. If it is a spontaneous rupture of an artery in an old patient, the artery will have atheromatous walls, and the corresponding arteries on the other side will probably be atheromatous too, or in some degenerative condition, and treatment by ligature would probably be attended by some risk. I have had one or two of these cases of ligature applied to the common carotid, without any hemiplegic misfortune. One point which Mr. Flemming's case of cure emphasizes is, that if one decides to ligature the common carotid artery in a case of arteriovenous communication in the cavernous sinus, it is important that the patient should be kept at rest for a long period after the ligation. In one of my cases the bruit and symptoms ceased after the tying, but some liberty having been given the patient, in thirty-six hours pulsation returned and the bruit recommenced, but both subsided in a day or two when complete rest was rigidly enforced. I think surgeons at the present day are a little too ready to discharge patients after operation, and this is an instance. The recumbent position and considerable immobility should be maintained for a period of weeks after the ligaturing has been done.

Mr. R. R. CRUISE : I have at the present moment a case with a similar condition following a gunshot injury, on the left side, with pulsating exophthalmos, and an easily audible bruit. In consequence the patient is in a very distressed condition, as he is prevented by it from sleeping. Speaking from memory I think he has had five operations during the past fifteen months. At first the external carotid was tied, then the internal carotid was similarly treated. As he was no better, the jugular vein was tied on the same side. Still there was no improvement, although obliteration of the lumen of the carotid by pressure had succeeded in stopping the noise. The condition was so distressing, that the man said he would end his life if he was not relieved. Six weeks ago the common carotid on the other side was tied, so that he now has very little left to be tied. All the operations were done by skilled surgeons, so there can be no doubt about the efficiency of the procedures. The man now wants me to tie the veins in the apex of the orbit, which I do not feel inclined to do. I thought the case would be instructive to those who propose ligation as a cure of these conditions.

Gunshot Wound of Face; Loss of Left Eye.

By M. W. B. Oliver, M.B.

Patient, a male, aged 33, leading signaller, R.N. Wounded, May 31, 1916.

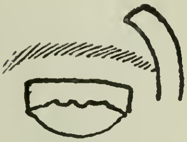
On admission, May 21, 1919: Left eye excised. Loss of large part of upper lid. Sunken socket. Upper fornix completely obliterated.

May 30, 1919: Cartilage implantation from seventh costal cartilage. Shaped into oval and implanted into deep tissues of orbit. Catgut sutures.

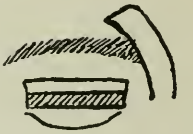
June 13, 1919: Adhesions on inner side of left socket divided, and epithelial inlay inserted.

August 21, 1919: Lining of new lid formed by inverting the remains of the skin of the remnants of the upper lid. Temporal flap containing the superficial temporal artery brought down on pedicle and sutured in position to form outer layer of lid. Tarsal cartilage replaced by piece of cartilage from left ear.

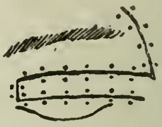
Operation, August 21, 1919: Incisions; lid of skin inverted to form lining of new lid; temporal flap brought down to form outer layer of lid.



Incisions.

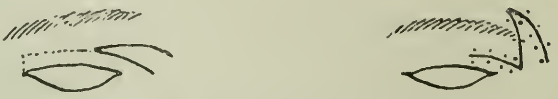


Lid skin inverted to form lining of new lid.



Temporal flap brought down to form outer layer of lid.

October 16, 1919: Pedicle returned from upper lid to forehead, and eyebrow brought back to its normal position.



Pedicle returned and eyebrow lowered.

Section of Ophthalmology.

President—Mr. W. T. Holmes Spicer, F.R.C.S.

Vessels on Iris.[1]

By G. Winfield Roll.

T. B., female, aged 27. Duration of disease from infancy and probably from birth. History of case: Right eye always partly blind; vision, perception of light. Pupil inactive, iris atrophied. Radial arrangement of vessels seen on front of iris. Latterly some circumcorneal injection with pain and intolerance of light; some ptosis. The arrangement of the vessels is perfectly regular, suggesting anatomical arrangement.

Points of special interest for which the case is exhibited: Right, vessels on iris and within pupil, around edge of pupil, encircling shrunken opaque lens; vessels radiating from periphery to pupil; pupil oval, shallow; slight haze of cornea. Left: Coloboma below optic disc; one disc diameter below area equals two discs in size; large atrophic area above optic disc; left optic disc shows indications of a congenital crescent vitreous. Vision $\frac{6}{12}$ c̄ + 2 cyl., down and in. Left, also long riband opacity free end forward, attached end backward, situate in axis of eyeball; forward end floats near back of lens.

I brought forward the case in the hope of ascertaining to what the condition is due. The arrangement of the vessels was so regular that I wondered whether the atrophic condition of the iris would enable some of the normal circulation of the iris to be visible from the front. But possibly the vessels are too large for that to be the explanation.

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[1] At a meeting of the Section, held February 4, 1920.

DISCUSSION.

Mr. E. TREACHER COLLINS: This is a very interesting case. Some of the vessels from the surface of the iris turn round the pupillary border, others pass in front of the lens capsule. Probably there is persistence of the fœtal vascular system, and when Mr. Roll removes the eye he will most likely find there is a patent central hyaloid artery.

The PRESIDENT: I take it there is no evidence that this patient could ever see with that eye, she has never had in it more than perception of light. I showed a case a good many years ago, and a drawing of it appears in the *Transactions of the Ophthalmological Society*, with new vessels on the front of the iris. Since then I have seen many other somewhat similar cases; I do not think they are very uncommon. In the case I refer to, the vessels were of inflammatory origin, and were larger than those in front of the iris in Mr. Roll's case. And they did not run the ordinary course of iris vessels, but went across from one side of the anterior chamber to the other. It was an old case of iritis and cyclitis, followed by increased tension, and I was not sure, at that time, how far the prolonged use of eserine had increased the growth of vessels. The examination of that eye was made by Mr. Parsons: perhaps he can throw some light on it for us.

Mr. J. H. PARSONS: I remember very little about the case referred to, but there is a picture in the Ophthalmological Society's *Transactions*, showing one vessel going across the iris, obviously a newly-formed vessel; certainly it is not a dilatation of a normal vessel. In the present case the vessels are of ordinary type of distribution with the exception of those which have run round the pupillary margin. If it is decided to remove the eye I hope it will be very carefully examined.

Mr. ROLL (in reply): The patient is now aged 27, and she has attended at the Royal Westminster Ophthalmic Hospital from the age of 3 years; I think she was then under Mr. Donald Gunn. The eye appears to have been the same during all that time, but latterly it has become inflamed and intolerant of light, therefore my intention was to remove it. She has never had any more than mere perception of light in it.

Macular Detachment of Retina; ? New Growth.

By A. HUGH THOMPSON.

H. B., MALE, aged 65. Duration of disease sixteen months. Sight of left eye began to fail in October, 1918. In October, 1919, vision was fingers at 3 ft. The macular area was apparently pushed forward about 2 D. or 3 D., and the red reflex was blurred. Tension normal. Slight opacity in lower part of lens. Right eye normal H. + 3 and vision $\frac{6}{6}$.

On October 30 there was a definite detachment at the left macula, the vessels being raised 8 D. above level of disc; red reflex still retained. Tension normal.

On January 22, 1920, the detachment had increased, and the red reflex was no longer there. Below it has a well-defined margin. Elsewhere it rises gradually from normal retina. There is still no peripheral detachment and tension is normal. No hooklet seen.

There is no doubt that this is a new growth, and I am fortified in that view by opinions expressed by several members to-night. That being so, the sooner the eye is excised the better for the patient.

The interest of the case lies in the fact that when I first saw the patient, about four months ago, very little could be seen: the macula itself was raised not more than 2 D., and the diagnosis was by no means plain. Lately there has been a regular grey detachment, which is increasing in size. From first to last there has been no increase in tension. The growth, if such it be, is exactly at the macula.

Postscript.—The eye was excised. The section showed a sarcoma of the choroid at the macula.

Pulsating Exophthalmos.

By Harold Grimsdale, F.R.C.S.

T. L., AGED 75.[1] Summary of history: In 1855 he was run over by an empty wagon which passed over the left side of his head; he was unconscious for three weeks. On recovery he noticed a drumming sound in his head; the left eye became prominent and rotated inwards; he never noticed diplopia.

When seen by Mr. Frost, in 1883, the left eye was convergent about 25° and could not be moved beyond the middle line. The movements of the right eye were normal. Right vision, $\frac{20}{70}$; left vision, $\frac{20}{50}$; 1 : $\frac{20}{30}$. There was a small pulsating swelling in each orbit. Ophthalmoscopic appearances normal in each eye. Right vision field slightly contracted; left vision field contracted on nasal side.

No surgical treatment was undertaken for the exophthalmos, but the left internal rectus was divided, and for a time the eyes were parallel.

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[1] Vide *Trans. Ophth. Soc. of the United Kingdom*, 1883, iii, p. 9, and 1891, xi, p. 35.

In 1893 Mr. Frost published a further note to the effect that the pulsation in the right orbit had gradually lessened and finally disappeared.

In April, 1896, "the left eye became bad," and he came to St. George's again. There was enormous chemosis of the conjunctiva, which protruded outside the lids, and the lids were very swollen and dusky.

On May 6 there was well-marked pulsation in the left orbit, but it had ceased on May 9. The vision at this time in both eyes was $\frac{5}{12}$. In 1901 it is noted that he had drumming in the head, but no pulsation. In 1905 he suffered from herpes frontalis. In 1911 there was still a blowing murmur, but no pulsation. Now right vision is $\frac{6}{24}$; he thinks it failed slowly. The optic disc is definitely pale; but the visual field is roughly normal. The left eye is so convergent that he does not use it, and I have no recent note of its acuity. I cannot tell from my notes when the right vision failed; there is a note in 1883 that both eyes move outward badly, but the note in the *Transactions* (vol. iii) is that right movements are normal. It has been paretic for twenty years.

The fact that this condition has persisted for sixty years seemed to me to warrant my asking him to come up, but I am sorry he has been unable to do so on account of illness.

I want to ask the views of members of the Section on one point. On looking up ophthalmic literature, I find it is said that, often, a prominent feature of these cases is paralysis of the third nerve; but in the few cases of the condition I have seen it has always been the sixth nerve which has been most clearly paralysed. This man had complete movement of his right eye, with the third nerve, but complete paresis of both sixths. In a case I recently saw, in which rupture occurred without any traceable exciting cause, the first symptom was diplopia, due to paresis of the external rectus. And that is what one would rather expect, because, of course, as soon as rupture occurs, pressure increases on the sixth nerve, which is lying inside the sinus—i.e., the pressure surrounds the sixth nerve, therefore, one would think, the conductivity would be comparatively easily interfered with, whereas some of the excess of pressure is taken up by the dura mater before it comes on to the third and fourth nerves on the outside of the sinus.

DISCUSSION.

Mr. CHARLES HIGGENS: I remember two cases of the kind in which the patients got well spontaneously. In both instances the condition made its appearance during labour. There were all the symptoms present: proptosis, pulsations, buzzing in the head, and in a few months both patients recovered without treatment. The eyes were more or less fixed during the time the trouble lasted.

Mr. HERBERT FISHER: The cases of the kind I have seen have not been notable for definite paralysis of any of the extra-ocular muscles, but I agree with Mr. Grimsdale as to the probability of the sixth nerve being the one most frequently involved. And that would apply to two classes of cases of pulsating exophthalmos, which I think exist, with arterio-venous communication in the cavernous sinus. In the first class of case, those definitely due to fractured base, it lacerates the carotid artery, and obviously the sixth nerve, which is lying on the floor of the cavernous sinus, would be the one most likely to be implicated, whereas the third nerve, in the lateral wall of the sinus, would almost certainly escape. In the other class of case, in which, I think, there is usually a history of a fall, rupture of a diseased artery is the first trouble. The disturbance in the circulation results in a faint, and the falling of the patient is a secondary phenomenon. And there, too, I think it is the sixth nerve which would be implicated, owing to the immediate contiguity of the sixth nerve below the wall of the diseased artery. There are some cases of extreme atheroma of the cavernous sinus which produce paralysis of the sixth nerve, without a rupture of an artery taking place at all, merely by the pressure of a diseased arterial wall on the subjacent nerve.

Mr. GRIMSDALE (in reply): In the case I saw recently in which the internal carotid was tied, the paresis of the rectus disappeared in a short time. It was tied within ten days of the onset of the symptoms.

Gunshot Wound in Occipital Lobe.

By NORMAN B. B. FLEMING.

F. P., AGED 37, was wounded by shell fire in August, 1918, and states that he was subsequently blind for four days. No notes taken at the time are available. Examination shows a deficiency of the vault of the skull over an area roughly circular in outline and 2¼ in. in diameter, situated in the middle line, the lower part of the aperture being 1 in. above the inion. He was operated on in a casualty clearing station, when fragments of bone and nickel were removed, and he has had no operation since. He complains of head-

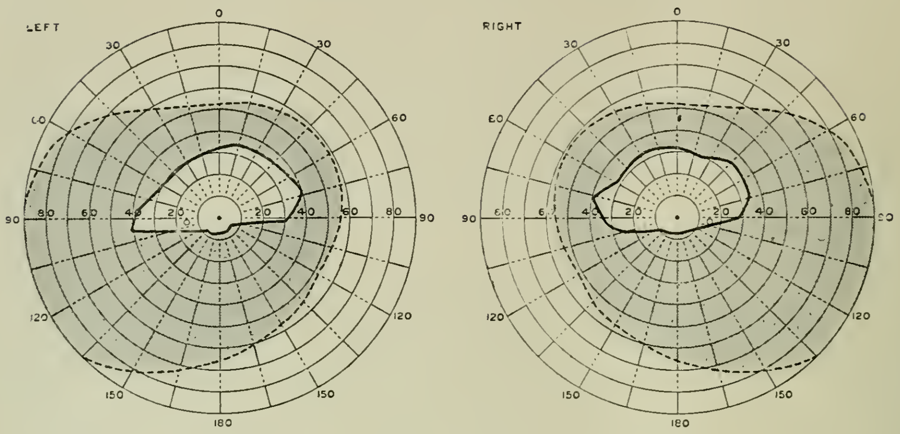

Fig. 1 (Blue).

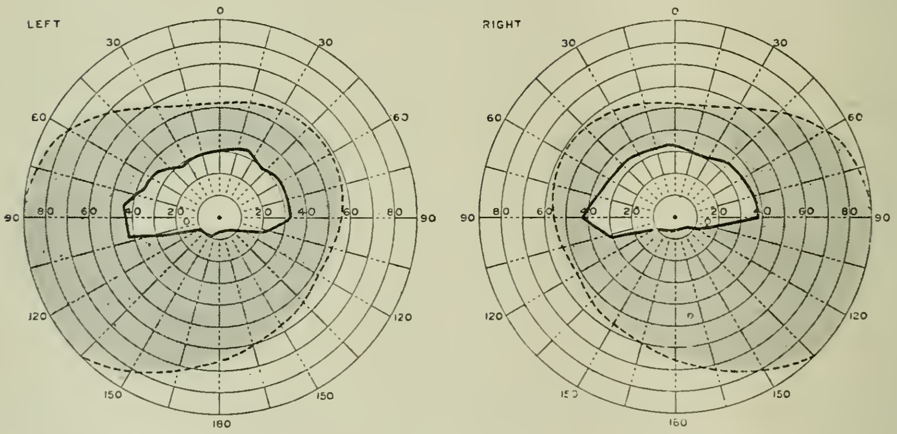

Fig. 2 (Red).

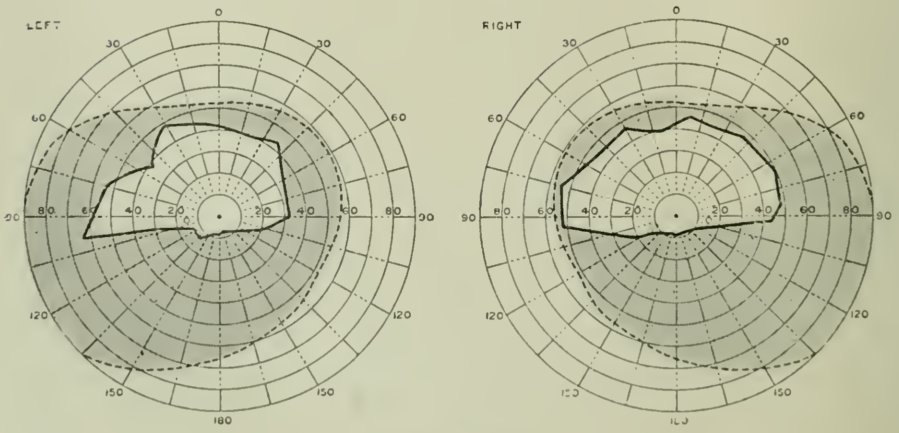

Fig. 3 (White).

aches, giddiness, slight deafness of the right ear, and loss of sight. The eyes present no objective abnormality; central vision is acute, being $\frac{6}{5}$ partly in right eye and $\frac{6}{5}$ fully in left eye. His fields show a sharply defined hemianopia below in each eye with a considerable reduction of the peripheral part of the upper field. This suggests a complete destruction of the dorsal part of each visual cortex, the injury extending into the anterior portion of each ventral part also; it is difficult to see how this could take place without injuring the extreme posterior part where the macular area or the radiations from it are located, unless it be that the fibres pass in a lateral direction for fully 1 in. before turning forwards. Pressure from exudation would be more likely to affect the macular area, being nearer, and the path of a shell fragment to the peripheral cortex would be through the macular radiation.

DISCUSSION.

Mr. HERBERT FISHER: I saw a case almost identical with this after the Boer War. It was sent to me by Sir George Makins, and I reported on it to him, and I think it is included in the book he published on the surgery of the Boer War. The only difference was, that the first field this man gave me was an altitudinal hemianopic field, with some peripheral loss above, and a horizontal lower margin. The man, however, had become very mistrustful of my purposes in making the examination: he had secured a post in civil life as a letter-carrier, and his suspicion was quite definite. He gave me a spiral field in his second eye, though I have no doubt that really it was comparable to the condition in the first eye. It was a mid-line occipital lesion, high up. From memory I cannot say whether both maculæ were left, but he evidently had enough vision to enable him to carry on as a postman.

Mr. NORMAN FLEMING (in reply): I cannot find any case exactly similar to mine in Dr. Gordon Holmes's paper in the *British Journal of Ophthalmology*, 1918. There is one in which part of the upper field has been destroyed, in addition to the lower, not one in which reduction in the upper field goes all the way round.

Mr. HERBERT FISHER: The reduction in the upper part of the field went all the way round in my case. I do not think there is much difficulty in explaining the peripheral loss. These men, after being hit in the occipital region, even when left with lateral hemianopia, are, frequently, totally blind for days, and in the lateral hemianopic cases there is a direct laceration and damage of one half-vision centre, and a concussion effect on the other, in the form of œdema of the nerve tissues with punctiform hæmorrhages, &c.: from some of these conditions recovery is possible. But, I take it, the whole of the half-vision centre does not always recover. In the case before us the lower portions of the half-vision centres below the calcarine fissures, recovered, but not completely.

New Growth of the Retina.

By N. Bishop Harman.

E. S., AGED 4, was seen late in October, 1919. His doctor thought there was cataract in the left eye. The boy is a little fellow, but his parents, too, are unusually small. He is an only child. His health is good. There is no defect other than that of the eye. The Wassermann reaction is negative. The mother states that she noticed something wrong with the left eye when he was 14 months old. When she was teaching him to walk the light came from behind her and made his eye shine; she saw the reflection several times but thought nothing of it until her doctor observed it.

The right eye is healthy; there is nothing out of the natural order except 1 D. of hypermetropia. The left eye is immediately noticeable, owing to the presence of a large white oval body behind the lower part of the iris; it looks just as if the lens were dislocated and opaque. Focal illumination brings out this oval mass in strong relief and illuminates the whole of the interior of the eye. Examination with the ophthalmoscope shows the whole of the fundus to be covered to a fairly uniform depth from ciliary region to centre and in almost every part with masses of white material; the masses are all more or less spherical; they vary greatly in size, some are but little larger than the diameter of a vein at the disc, others are as big as a dislocated lens. Most of the masses are separate from each other, that is, they have no visible connexion, but close to the central region there is a pair attached, shaped like a cottage loaf of bread. Most of the masses are immobile, but two or three in the centre float with the movement of the eye; they do not leave their positions, but sway so freely that their attachment (whatever that may be) must be very slender, and the vitreous must be fluid to allow of the movement. The masses are white, with a suggestion of transparency; they have a nap or slightly fluffy surface that suggests solidity. With one exception, no blood-vessels can be seen in connexion with the masses; the sole exception is a fine vessel curling round a mass in the upper and outer part of the fundus. In a very few spots the colour of the fundus can be seen between the masses; in these small areas the vessels seen have an irregular distribution. Transillumination gives an appearance which is difficult to make out; light enters the eye between and around the masses, but it also appears to pass through the masses and to be inversely proportional to their size.

Until fourteen days ago the eye had caused no sense of discomfort, and there was a total absence of symptoms of irritation. The cornea, lens and iris were normal, there was a consensual pupillary reaction, and the vitreous was free from opacity where there were no white masses. In the last fortnight there has been pain in the eye, the vessels of the conjunctiva and ciliary region have been enlarged and the iris discoloured; the tension of the eye has increased. Until these signs appeared I hesitated to advise the removal of the eye. The long period of the known presence of a reflecting mass in the eye (three years), the total absence of any sign of irritation, the absence of change during repeated examination over three months, the unlikeness of the appearance of the masses to any usual form of glioma, left room for uncertainty as to the danger of the eye. The change in the last fortnight leaves none. But the parents refuse to allow the removal of the eye. But I anticipate that the remarks which they have heard from those who have examined the eye this evening will be calculated to persuade them.

The appearance of the fundus is remarkable. I do not remember having seen anything at all approaching this character before, and I can find no description of a similar growth.

DISCUSSION.

Mr. ELMORE BREWERTON: Any child under 6 years of age who has a white mass in the vitreous, with increased tension, should have the eye removed. In this case the tension is undoubtedly increased, and there are new vessels on the iris. I had a very bad view of the fundus, but what I saw made me suspect an endophytic glioma. The eye should be excised as soon as possible.

The PRESIDENT: I have seen a case of glioma with separate proliferations seeming to come forward into the vitreous, and even having no connexion with the mass behind. I have seen a few cases in which there has been what one called a hypopyon. It was not that, because the material of the supposed hypopyon was like white paper and consisted of débris of the glioma that must have somehow originated from the growth behind. I think the present case is one of glioma.

Addendum.—June 22: As a result of the statement made to the mother of the child by various members at the meeting, she agreed to the removal of the eye. The operation was performed two days later. A long length of nerve was removed with the eye. The free end was found to be healthy, but close to the globe there was a suspicious knuckle. After hardening, this was cut and found to be part of the new growth, which was a glioma. The orbit was watched carefully; one month later the socket was seen to be bulging suspiciously. The contents of the orbit were therefore exenterated, including the stripping of the periosteum. After hardening, the contents were cut across and new growth was found in the centre. The outside of the excised mass was free from growth. The cavity is now clean and will be shortly grafted. The boy will be kept under regular observation, and report made later if there are any further signs of recurrence. His general health remains good.

The Radical Cure of Gonorrhœal Iritis.

By S. H. Browning, M.R.C.S., L.R.C.P.

Gonorrhœal iritis being a late sequela of gonorrhœa, is not as a rule seen first hand by a specialist in venereal diseases, with the result that it is more often than not treated as a local affection. In this way it has become to be talked of as "recurrent iritis," whereas few, if any, cases of gonorrhœal iritis ought to occur in the first place, and certainly ought not to recur if properly treated.

The object of this paper is to try to show that by proper treatment of the genito-urinary tract and the cure of the disease existing therein, a permanent cure of the gonorrhœal iritis will follow; also to impress upon ophthalmic surgeons their responsibility for seeing that their cases of gonorrhœal iritis are adequately treated in the way I shall indicate.

Gonorrhœal iritis is, I think, a toxic condition, and is not due to the presence of the gonococcus in the eye, for this organism has only been isolated from the eye in one case (Sidler-Huguenin), and then only from the blood-stained exudate from the anterior chamber of a patient suffering from acute gonorrhœal septicæmia. I have examined the gelatinous exudate from cases of gonorrhœal iritis under the care of Mr. Lang, but have not been able to cultivate the gonococcus or find the organism in direct smears. These experiments were carried out under the most favourable conditions, as the exudate was in all cases planted out on to suitable media within a few seconds of being drawn off. Then, again, the rapid recoveries I have sometimes experienced in patients after vaccine treatment more closely resemble recovery from a toxic than from a microbic condition.

That the iritis is gonorrhœal in nature is suggested by the following observations:—

(1) There is generally a definite history of gonorrhœa some years previously. I have never seen a case of iritis during the acute stage of a urethritis; an observation confirmed by Mr. Canny Ryall, of All Saints' Hospital, and also by Mr. C. H. Mills, who has been at the Military Hospital, Rochester Row, for the last five years. The observations of these two surgeons must run into many thousands of cases.

(2) The specific reaction to gonorrhœal vaccines.

(3) Its association, though not invariably, with gonorrhœal rheumatism.

(4) The recurrence of iritis or exacerbation of symptoms after prostatic massage and vesicular massage, probably due to the liberation of toxins and bacteria—dead or alive—into the blood-stream.

(5) By negative evidence, that is to say, eliminating as far as possible all other sources of infection, such as pyorrhœa, septic tonsils and ears, cystitis, alimentary infections, &c.

I have not mentioned the complement-fixation test in the above list as I did not do it in any of the cases I am going to record, and also because in my hands it has not yet proved reliable and I do not yet know of a method which gives consistently true results.

TREATMENT.

Treatment must be considered under two headings, preventive and radical.

The preventive treatment of gonorrhœal iritis really rests with those who see the gonorrhœa in its early stages, as no case of gonorrhœa can be said to be cured till the prostate and vesicles have been carefully examined, and proved by repeated examination of their contents expressed by massage, to be free from infection. Examination with the urethroscope should also be carried out. I believe that if the above examination were done by a specialist in genito-urinary work and a bacteriologist familiar with the examination of urines after massage, there would be no such eye condition as "gonorrhœal" iritis. I think that it is now generally recognized that all cases of gonorrhœa should be treated with vaccines, for although they do not materially shorten the acute stage of the disease, there is considerable evidence to show that complications and sequelæ are to a large extent prevented by their use.

The radical treatment I would suggest to be as follows:—

(1) The immediate treatment by the ophthalmic surgeon.

(2) The immediate treatment by vaccines.

(3) Continued treatment by vaccines and prostatic and vesicular massage with treatment of the urethra if necessary.

Of the immediate and continued treatment by the ophthalmic surgeon I, of course, can have nothing to say, but the responsibility generally rests with him as to whether the subsequent treatment is advised or not. I am convinced that the immediate treatment by gonococcal vaccines is essential if the best is to be done for the patient. The acute attack is often cut short and the relief to the patient as regards pain is definite. An autogenous vaccine should be used if possible, but while this is being prepared suitable doses of a reliable mixed vaccine may be given.

Vaccine therapy has rather gone out of fashion and repute the last few years, and this, I think, is due to the haphazard use of commercial vaccines by persons unskilled in vaccine therapy. Vaccines freshly prepared from many strains are necessary for stock use, and these are seldom obtainable in a chemist's shop. However, I have found the French preparation called "Dmégon," and Mulford's vaccine to be really good and reliable. The dose of vaccine varies with each patient, and I have used initial doses of from 5 to 500 million with success. I would here remind you that vaccine therapy is an aid to general medical and surgical treatment and is in no way intended as a substitute.

I now come to an important part of the treatment, and that is the examination of the genito-urinary tract. Practically every case of gonorrhœal iritis I have examined, or have had examined, has shown some disease of the prostate or vesicles, and massage of these organs has shown the presence of pus cells and often of gonococci. The cure of the iritis depends on the cure of the accompanying prostatitis or vesiculitis, and this is generally brought about by massage. Massage of the prostate and vesicles is a difficult and arduous operation, and unless done by an expert is useless, as much judgment is needed in estimating the amount of massage any one prostate will stand. Only those thoroughly familiar with the normal and diseased organs can carry out the treatment successfully.

The method usually adopted is as follows: The patient is told to come with a full bladder, and before massage a little urine is passed into a specimen glass in order to test for threads, pus or micro-organisms.

He then gets up on to a couch or table in the knee-elbow position, and the well-lubricated forefinger is passed into the rectum. The prostate is then massaged with a sweeping motion round the edges of the lobes, first on one side and then on the other, firm pressure being applied all the time, and working towards the centre: this is continued for two or three minutes. The finger is then passed over the prostate and the vesicles are massaged and emptied of their contents, pressure is applied to the abdomen with the left hand at the same time in order to push the bladder back and thus enable the vesicles to be more easily reached. This is an important point. The patient then passes his water into a glass, and the centrifugalized deposit is examined for pus, gonococci or other organisms.

Gonococci are not found in all cases of iritis, but this is probably due to the difficulty of detecting the organism, which is often swollen and stained badly, or lost in a mass of spermatozoa, pus cells, epithelium and other organisms, but the fact remains that even when the gonococcus is not found the iritis is cured when the urine examined after massage repeatedly gives a normal deposit.

I do not think that iritis is often due to the other bacteria found after massage, such as staphylococci, *Bacillus coli*, diphtheroids, &c., though Mr. Canny Ryall has told me of a case of iritis in which a pure culture of the *Bacillus coli* was isolated from the urine after massage, and which recovered without recurrence after suitable treatment.

I have not yet seen a case of gonorrhœal iritis in a female.

The following is a list of some of the cases I have treated, or had treated by Mr. Canny Ryall or by Mr. C. H. Mills, and thanks are due to them for the great trouble and interest they have taken with the cases, also to Mr. Lang, Mr. Worth, Mr. Hudson, Mr. Greeves, and Mr. Whiting for permission to publish their cases. I have followed up these cases as far as possible, and with one exception there has not been a recurrence for at least the last five years. The recurrence was in L——, a case of Mr. Worth's, and he came to see me last month with an acute iritis, and begged for vaccine treatment and a letter to All Saints' Hospital. He had been to this hospital before, but for certain reasons had been unable to continue treatment although he was told that he was not cured. He had been four and a half years without a recurrence. Mr. Ryall saw the case and demonstrated the enlarged prostate and matted vesicles to be felt in the rectum. Examination of the urine after massage showed the presence of many pus cells and a considerable number of gonococci. He is now undergoing a thorough course of massage and vaccine treatment.

Case I.—Dr. A., married. History of gonorrhœa eighteen years ago; a little rheumatism; recurrent iritis; massage and vaccines. Cured.

Case II.—Dr. B. History of gonorrhœa six years ago; sixth attack; massage and vaccines. Cured. No rheumatism.

Case III.—Dr. C. Many attacks of gonorrhœa, also rheumatism; cannot remember the first one, wife has chronic "tubes." No children. A difficult case; another attack developed during treatment, but eventually cleared up completely, and there has been no recurrence for the last five years.

Case IV.—Dr. D. History five years. Cured. No rheumatism.

Case V.—Dr. E. History six or seven years. Cured.

Case VI.—Dr. F. History ten years; did not improve much with vaccine treatment alone, but got quite well with massage and vaccines.

Case VII.—W. P. History of gonorrhœa ten years ago, mentioned above.

Case VIII.—O. R. First attack; pus and gonococci after massage. Cured.

Case IX.—J. History two years; third attack in five months. Cured.

Case X.—I. M. S. History of many attacks of gonorrhœa with acute epididymitis and prostatitis; one eye practically blind as the result of many attacks of iritis. Cured.

When I wrote the above I was not aware that Mr. A. S. Cobbledick had published an excellent paper on the same subject in the *Lancet* in 1918,[1] otherwise I should probably not have gone to the trouble of writing mine. However, I notice that he leaves out a most important point—namely, the necessity of examining the seminal vesicles as well as the prostate, and I rather think that the vesicles are as important, if not more so, than the prostate. Mr. Rolf Creasy also brings out this point in his letter to the *Lancet* on March 25, 1918 (p. 447). Mr. Cobbledick in his paper quotes me as having said that I had rarely seen a case of eye syphilis properly treated—a statement which still stands—and he goes on to state that he had "never seen a case of recurrent gonorrhœal irido-cyclitis in which any attempt had been made to obtain a cure." He could not have been familiar with the practice of at least four of the surgeons at the Royal London Ophthalmic Hospital, who were in the habit of sending me cases for vaccine treatment early in 1912, cases which I sent on for further treatment to Mr. Mills and to All Saints' Hospital. Mr. Creasy no doubt refers to some of these cases in the letter I have mentioned above. Altogether, I have records—many of them rather scanty—of some forty-seven cases of recurrent gonorrhœal iritis treated in the way I have described.

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[1] *Lancet*, 1918, i, p. 335.

DISCUSSION.

Mr. C. H. MILLS: I propose to commence with a few remarks under the heading of "Cautions."

First.—I am of the opinion that it is better to withhold the local treatment in the form of massage to the prostate or seminal vesicles, should a residual focus be detected in the same, until the attack of iritis has reached its zenith. Up to this stage it is wise to rest content with treatment of the iris itself with mydriatics, &c., the reason for this being that I have noticed a severe exacerbation in the iritis on the day following massage to a residual focus; if undertaken during the acute stages, this is sufficient to discourage the patient. I have seen the same reaction in an acutely inflamed iris following the first vaccine injection.

Second.—Never give a vaccine injection simultaneously with massage to the residual focus. During massage a large dose of toxin is put into circulation, to which one would be adding the toxin in the vaccine, unless the vaccine is detoxicated. I have seen an attack of iritis follow prolonged massage to a gonococcal prostate at the first sitting.

Third.—Thoroughly examine the patient for any co-existing sources of toxic absorption. I have frequently been struck with the high incidence of metastatic complications in cases of gonorrhœa when such are present. For instance, should a patient with severe oral sepsis contract gonorrhœa, he is a very likely candidate for arthritic involvement. Therefore in dealing with a case of gonorrhœal iritis, do not be obsessed with treatment of the focus in the prostate, vesicles, &c., to the degree of overlooking say pyorrhœa, otorrhœa, suppuration in an antrum, frontal sinusitis, or intestinal auto-intoxication, &c. The absorption from the genito-urinary focus, whether merely of the toxins, or of the gonococci themselves may be the deciding factor in causing a chronically irritated iris to flare up. Gonococci will always tend to attack a "damaged" spot of lowered resistance.

Conclusive evidence is still wanted before we are justified in classifying all these cases as gonococcal. In such a case—say where the patient definitely contracted gonorrhœa complicated by a prostatitis ten years previously, which has remained chronic, and is to-day suffering from iritis, we yet require further evidence before we dogmatically classify the iritis as gonococcal in the absence of gonococci in the specimens obtained from the prostatic secretion. When the complement-deviation test to the gonococcus has been rendered as reliable as the Wassermann test is in syphilis, then we shall have made great strides towards an absolute diagnosis in these cases. I have met with a case of recurrent iritis with gelatinous exudation clinically typical of gonorrhœal iritis in which we were never able to detect gonococci in the accompanying prostatitis, and the complement deviation to the gonococcus remained negative (by Dr. David Thomson's technique). The eye condition cleared up with that of the prostate, and has not recurred in the past eighteen months. The organisms in this case were a staphylococcus and strepto-pneumococcus.

The strepto-pneumococcus is a very frequent organism in chronic prostatitis and vesiculitis. There certainly must be a definite group of cases in which the gonococci have died out in the residuum from an original gonococcal focus, such focus remaining chronically infected by other organisms, the commonest of which are staphylococci, diphtheroids and strepto-pneumococci (Gram-positive). We know that in the urethra itself, a chronic gleet frequently persists, due to a secondary infection when all gonococci have disappeared. An American surgeon, Colonel Young, if I remember rightly, who was investigating the connexion between chronic vesiculitis and rheumatoid arthritis, informed me that he had isolated a streptococcus in the great majority of a series of 300 cases in which he had removed the vesicles. This might possibly have been the strepto-pneumococcus mentioned above. On the sociological side, with regard to the incidence of gonorrhœa — a very difficult estimate to arrive at with accuracy—it has been stated that in cities and densely populated areas 70 to 80 per cent. of the male population contract gonorrhœa sooner or later. One will therefore recognize that when a patient presents himself with an attack of iritis the mere fact that he admits to have suffered from gonorrhœa in the past is in itself but slender positive evidence in the classification of the iritis as gonococcal. One should also recognize the existence of the hundreds of thousands of gonorrhœal patients who never develop iritis.

Mr. CHARLES HIGGENS: I can relate two cases which bear on this paper. The first, that of a gentleman aged 43, I saw first on September 18, 1893, and between that date and 1910 I attended him for sixteen attacks of iritis, more or less severe. He went backwards and forwards to New Zealand, and had many other attacks in which I did not see him. He had many treatments, including visits to Aix, Wiesbaden and La Bourboule. In 1910 I had him treated with a vaccine, and after that he had no more attacks. He had been seventeen years under my treatment. He lived until 1919, when he died of Bright's disease. Before he had the vaccine I do not think he went more than three years without an attack of iritis. But I think there may be a fallacy here and that the vaccine may have had nothing to do with the cure. The late Mr. Critchett saw this man and another who was under my care. He told them both: "You will grow out of it." The second man, of the same age as the other, 43, came to me in 1884; he had had many attacks of iritis before I saw him, and I attended him in a great many. In 1903 he had recovered, for he had no more recurrences; he had attacks of iritis to my knowledge for nineteen years, and then got well. I say there is a source of fallacy here, because both these men were of the same age when they came to me, and one had vaccine, the other did not, and both ceased to have iritis at about 60, one having been subject to attacks for at least seventeen years, the other for nineteen. It may be that at about 60 a man has used up his stock of toxin, and has not been in the way of getting a fresh dose. I may say that both these men had stricture. I was only groping in the dark in those days, but I associated the stricture with the attacks of iritis, and I told the patients to keep the strictures open, or they would have trouble with their eyes.

Mr. N. BISHOP HARMAN: I should be glad if Mr. Browning could say whether the cases in which gonococci were demonstrated after prostatic massage are likely to be carriers of the disease. I would point the question by an example: There was a man I saw in 1911, who had had gonorrhœa shortly before, and who then had episcleritis. He was under the care of a very good practitioner, who treated him in the most approved style. I treated the eye, and suggested a vaccine. Later he had prostatic massage. After five years he had another attack of episcleritis. A fortnight ago I saw him with a third attack of episcleritis, and he said that after the second attack he had been examined by a good genito-urinary surgeon and was found to be free from organisms. I told him the repetition of attacks made this doubtful, and that he had better have his prostate again massaged; and I added "You may want to get married and therefore must be safe." He replied, "I got married a year ago, and my wife is quite right." Does the satisfactory marriage prove that his prostate is healthy, or that any organisms that might be obtained after massage are sterile—dead, in fact? Or does the occurrence of the episcleritis indicate that this man has got the organism lurking about, which may at any time do harm?

Mr. GOULDEN: I disagree with Mr. Browning's statement that it is not proved that an iritis may be due to the presence of gonococci because the patient has had septicæmia and Mr. Browning has not been able to cultivate the gonococcus from the exudate. It seems to me that if you find the gonorrhœal organism in the exudate, or even in the blood from the iris when there is iritis, the point is proved. I do not say that every case of iritis which comes on after gonorrhœa is due to the presence of the organisms, but surely in that type which comes on after gonorrhœa the point is proved. The earliest time at which I have seen iritis come on after gonorrhœa is six weeks. A question which perhaps Mr. Mills can answer is this: How long does it take the prostate or the posterior part of the urethra to be involved in a case of urethritis?

Mr. WILLIAM LANG: Will Mr. Mills say what more there is to be done in a case of recurrent iritis of twenty years' duration where massage of the prostate and a course of vaccines has failed to effect a cure? The genito-urinary surgeon who saw the case found one of the vesicula seminales enlarged and would not empty it by massage. He proposed to inject the vesicule with argyrol through the vas deferens. This was not done and the attacks continue.

Mr. WHITING: There are one or two points about the clinical application of vaccine therapy in gonococcal iritis which may be of interest. In the majority of cases, after a dose of the vaccine there is a very marked negative phase, and it is usually not only local, but general. It varies according to the dose of the vaccine, and to the individual case. Sometimes this negative phase is extremely alarming; but I have seen a considerable number of cases—mostly Mr. Lang's at the Middlesex and the Royal London Ophthalmic Hospitals—treated in this way, and I do not think that in any single case the negative phase proved disastrous, as sometimes happens in tuberculous infection after the use of tuberculin. The negative phase is always followed by a well marked positive phase, and if the use of vaccines is combined with the ordinary eye treatment for iritis, the case progresses satisfactorily. But they get an extremely bad exacerbation of the condition, and if they have not had a gelatinous exudation in the pupil before, they generally get it after the first dose of vaccine: I am speaking of acute cases. Mr. Mills seemed rather to deprecate giving vaccine in this stage, but I have not seen any harm from this, except in making the case more acute for twenty-four hours or so. With regard to the diagnosis and treatment of gonococcal irido-cyclitis, there are several factors which prevent these cases being diagnosed and treated as successfully as they might be, and I do not think that is altogether the fault of the ophthalmic surgeon. One is the history. A negative history is not of value in venereal cases, but a positive one is, especially if one finds the case has been either treated inadequately or not at all. I recently saw a case in which every other cause had been excluded except gonococcal infection. He was suitable for treatment by the methods Mr. Browning has suggested. Another point is, that it is not always easy to get hospital patients seen by the genito-urinary surgeon for diagnosis or treatment for a cure. I recently had one case, which I sent up to see if there was chronic prostatitis. It was confirmed, and he was given one application of prostatic massage. The iritis got well, but I think it will almost certainly recur. If one could ensure greater persistence in getting out histories, and greater facilities for diagnosis and treatment by the genito-urinary surgeon, we might make considerable advances in the treatment of these cases.

Mr. HERBERT FISHER: I am not clear as to the relative parts played by the gonococcus vaccine and the local treatment of the prostate and the vesiculæ seminales. I have had cases treated by surgeons for recurring gonorrhœal iritis, and I am not sure whether urologists consider it necessary to supplement their mechanical treatment of prostate and vesicles by giving a vaccine, or whether they claim to be able to eliminate the disease from the deeper portions of the urinary tract by local massage alone. If they claim that, I do not see where the field for the vaccine exists. If both are required, then the two methods must go hand-in-hand to complete a cure for our iritis cases. The important point, I take it, is that the local treatment shall be carried out by a man who is thoroughly competent to use the most approved methods in the right way, and that his work must be supplemented by that of the bacteriologist, who must be a thoroughly trained man: the duty of the bacteriologist would be to discover whether, in the detritus examined after prostatic massage, there is evidence of the gonococcus or other organisms. And later he should be competent to say that there is no longer any organism in the centrifugalized urine, and hence that a cure has been brought about and may be expected to be permanent.

Mr. C. H. MILLS (in reply): (1) One may say that six weeks after a gonococcal infection is by no means an impossible date at which iritis may develop. It is certainly infrequent so early. The posterior urethra is affected by the twelfth day in nearly 100 per cent. of cases. This is especially so when the out-patient is injecting with a small syringe. One frequently sees an acute prostatitis, epididymitis, &c., within a month of the disease having been acquired. I believe that every case for a period becomes septicæmic, some mildly so, some severely, according to individual resistance. Cases of septicæmia with a fatal termination within the first week are on record.

(2) With regard to the infectivity of these cases of recurring iritis. One meets with cases in which an interval of ten years has elapsed since the gonococcal infection, within which period the patient has married and produced healthy children, with no evidence of the wife having been infected. Others, of course, are a family tragedy. In the former the gonococci may have died out in the genito-urinary focus or possibly become encysted. In some cases the condition of the vesicles would suggest this, from the hard, discrete, movable, cystic nodules that can be palpated. It is in such cases in which one cannot isolate the gonococcus from the expressed secretion, or from the gelatinous exudate, as Dr. Browning has stated, that the complement-fixation test when perfected will be so valuable in accurate diagnosis. Chronically inflamed vesicles vary enormously. After excision some can be stretched out almost like a tape-worm. Others resemble a small bunch of loculi, some of which are "shut-off" and cystic. It may be possible for gonococci to remain trapped in such recesses, and so exist for years without their coming down the urethra.

(3) With regard to treating these cases by injections up the vas, I once had a case to treat in which this had been attempted: the patient also was a medical man. Together with his original gonorrhœa he had had left-sided epididymitis, and both vesicles were affected. The surgeon cut down on to the left vas first, and endeavoured to inject argyrol up the vas. It was found that the vas was occluded, due to the old vasitis. He went across to the other vas, and was successful in getting into it and injecting. The introduction of the argyrol was followed by intense epididymitis on the right side, which was hitherto functional. He had a sinus at the site where the vas was exposed, and the iritis kept recurring. There is a saying "Honour among thieves," but such treatment is pretty drastic on a medical man! Injecting 10 c.c. of argyrol is not going to reach one-twentieth part of the lining epithelium of a vesicle. One can wash quarts of it over a urethra and yet find gonococci next morning. It is very drastic treatment to excise chronic vesicles. One has to go through the perineum to expose the posterior aspect of the bladder, and I think it is never necessary for this condition. Usually the vesicles can be massaged comfortably, but it is difficult in very fat subjects. One first distends the bladder with an antiseptic, such as 1 in 4,000 permanganate, which will render innocuous any cocci which are expressed into it by massage; otherwise such organisms left in the bladder are likely to cause cystitis.

With regard to the delicate question as to what part is played by vaccines, and what part by massage, I am convinced that an autogenous vaccine is a very valuable adjunct. Some may have seen recent discussions on vaccine therapy in connexion with "protein shock." One can often produce good in a case by injecting the vaccine of a germ other than that which is causing the disease. In bad cases of gonorrhœa, with polyarthritis, and even in that rare condition hyperkeratosis blenorrhagica, where one has tried everything, T.A.B. vaccine produces excellent results. Some have tried to explain this on the theory of "protein shock." They maintain that if a "foreign" protein is injected the tissues are thereby roused out of their lethargy to deal with the gonococcus to which they have become, so to speak, "blasé." The vaccines, other than gonococcal, that undoubtedly have a marked beneficial effect on gonorrhœa are those consisting of typhoid bacilli, *Micrococcus catarrhalis*, meningococci and *Bacillus coli*. According to David Thomson these organisms are closely allied to the gonococcus in their biochemical composition, since they are all readily soluble in alkali, and moreover they are rich in proteoses.

Mr. BROWNING (in reply): Mr. Whiting said he had seen acute cases treated with vaccine, that they got their negative phase and a gelatinous exudate, and were no worse; but he did not mention that they get very much better: immediately they get over their negative phase, their iritis rapidly decreases; this Mr. Lang can confirm. I think acute iritis should be treated with gonococcal vaccines, not necessarily to the stage of producing a severe reaction, but sufficiently so to stimulate the body to the production of antibodies to correct this local condition. All the cases I saw in the early days with Mr. Mills were treated with vaccines beforehand, to get their eyes quiet rapidly, before massage was practised. The cases I have seen have done better with vaccines than without. I may remind you I said in the paper that vaccines should not be used as a cure, but as an aid to treatment. Because you put atropine drops into an eye, it does not say you should not do other things. Mr. Mills seems to think that in a case which had gonorrhœa ten years before, the gonococci have died out, but those eyes react to gonococcal vaccine, and with fairly big doses there is an excerbation of the condition. I do not think it is at all comparable to the anaphylactic shock which Mr. Mills talked about. I suggest there are gonococci still lurking in the patient's body. With regard to carriers, I know of one case of a doctor who was sent to me for vaccine treatment of his gonorrhœal iritis. I asked him—as I always do—when he had his gonorrhœa, and he said that was ten years ago, and that he had forgotten about it, and that there was nothing of that sort the matter with him. He had his vesicles massaged, and we found gonococci there. He subsequently infected his wife. She never had an acute discharge, but her tubes were blocked. I took notes as to whether my patients were married, and I always ask about their wives. Most of them have got healthy children, and so far as we know their wives were healthy, yet we found gonococci in the men after massage. A woman may be infected with gonococci for years without knowing it. I do not think the case of septicæmia mentioned by Mr. Goulden was comparable. In septicæmic conditions there are liable to be hæmorrhages anywhere, and the gonococcus was recovered from the patient's blood. This man had hæmorrhage into his anterior chamber, and gonococci were found, but I do not think that is sufficient ground on which to found proof that these old-standing cases are subject to a condition with actual gonococci in the eye.

Section of Ophthalmology.

President—Mr. W. T. Holmes Spicer, F.R.C.S.

Extraction with Peripheral Iridectomy, Endophthalmitis, Central Iridectomy ("Iridotomy") with Hook.[1]

By J. Herbert Parsons, F.R.C.S.

F. D., aged 60, attended at Moorfields Eye Hospital on May 3, 1916, with diffuse posterior cortical opacities in both lenses, the vision being $\frac{6}{18}$ in each eye. She was kept under weak atropine (gr. $\frac{1}{16}$ ad ℥i), semel, until June, 1917, when the vision was: Right, $\frac{3}{60}$; left, $\frac{6}{12}$. In July, 1919, the vision was: Right, counts fingers at 2 metres, projection good; left, $\frac{6}{24}$. Right eye slightly divergent.

On July 13, 1919, the lens was extracted from the right eye by a simple extraction, followed by a button-hole iridectomy at the periphery. The cataract was immature, and some soft lens matter remained in the pupil. The eye quieted down and the patient was discharged July 23.

A week later she came to the hospital with much injection of the eye, no keratitis punctata, small hypopyon; tension full; iris greenish and muddy.

August 6.—Right paracentesis.

The iritis gradually subsided under hot bathings and atropine, but the pupil became blocked and shrunken. The patient was discharged in August, 1919. The eye gradually became quite quiet, retaining good projection of light.

On January 17, 1920, the patient was readmitted. A central iridectomy was performed, and I carried out the method I have used, off and on, for a number of years as follows (you will find it described in the *British Journal of Ophthalmology*, March, 1920, p. 124, in an article by Mr. Mayou): A keratome incision was made in the lower part of the cornea, piercing the iris. A Tyrrel's hook was passed up behind the iris, and hooked into the small peripheral coloboma above. The pupil and iris were drawn out of the wound and cut off with de Wecker's scissors. An almost perfectly circular gap was made in the iris. Only a small amount of vitreous was lost. There was very little reaction, the eye being quiet three days later. The pupil now looks like an ordinary pupil. There is a bridge of iris above, and the artificial pupil below is circular. There are some vitreous opacities. The vision is now, with +11 D. sph. +1·5 D. cyl. axis 20° down and out, $\frac{6}{18}$, which I think is good, considering the severity of the operations the patient has gone through.

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[1] At a meeting of the Section, held March 3, 1920.

DISCUSSION.

Mr. M. S. Mayou: I do not know who originated the operation. It needs a very long, thin keratome, which you put in at the corneo-scleral margin, through the iris, and bring the point out either through the coloboma or through the iris itself. You then lateralize the keratome, so as to cut a band of iris, put a Tyrrel's hook behind, and pull the iris out, and cut it off.

The President: I have done this operation for years. I learned it from Nettleship. It is an extraordinarily good operation when it comes off. I have used it less often lately because I have got equally good results, on the whole with less risk, by cutting a hole with a Ziegler's knife.

Mr. Treacher Collins: I have often performed this operation, but I have always put the iris hook in front of the iris, first making an incision through the membrane, filling the coloboma above, and then passing the hook in through it, drawing the iris down, and cutting it off. It has been done at the Royal London Ophthalmic Hospital in that way as long as I remember.

Extraction of Right Dislocated Lens; Subluxation of Left Lens, which shows Suspensory Ligament.

By J. Herbert Parsons, F.R.C.S.

D. P., aged 24, attended Moorfields Eye Hospital on May 21, 1919; admitted the same day.

History: Patient had worn glasses since the age of 12, being "short-sighted." Three weeks before admission the right eye became inflamed, and patient had pains in forehead.

Examination: Right eye inflamed, central opacity of cornea. Clear lens dislocated into anterior chamber; tension full. Vision: Finger counting at 1 metre. Left eye quiet, cornea clear; iris tremulous. Lens displaced down and out, the edge crossing the upper part of the pupil; tension full. Vision: Finger counting at 1 metre.

Treatment: Complete rest, hot bathings. No mydriatic nor miotic.

Operation: On May 28, under chloroform, the lens was transfixed with a needle passed through the limbus. A corneal section was made with a Graefe knife, and the lens removed with a scoop. Some vitreous lost.

Subsequent course: The patient developed erythema of the chest and arms, and had a slight temperature for a few days. The wound healed well.

June 18: Vision, with + 8 D. sph. + 5 D. cyl. 20° down and out $= \frac{6}{18}$, with + 12 D. sph. and same cylinder = J 6.

February 11: Iris of left eye more tremulous, and lens dislocated farther downwards. The suspensory ligament is seen as a fringe along the upper border of the lens both by oblique illumination and with the mirror.

Remarks.—The patient came with a clear lens dislocated into the right anterior chamber last May. I thought it was an abnormally small lens, so I was not surprised to find there was ectopia lentis in the other eye, the lens being partially dislocated downwards, which is not the common displacement of lens in congenital ectopia lentis. The eye had *plus* tension, so something had to be done. I kept the patient on her back in bed for a week, but the lens did not return to its normal position. Therefore I had to extract it, which I did by fixing with a needle passed through the cornea, and extracting with a scoop. Some vitreous was lost, but not much. She has now $\frac{6}{18}$ vision in that eye, with glasses, and she says she sees better than she ever did before. That confirms the view that she had ectopia lentis in the right eye as well, before the dislocation occurred. At that time, the iris in the left eye was tremulous, so the slightly displaced lens did not satisfactorily support the iris. Two or three weeks ago I thought the iris was more tremulous than before, and the lens was displaced still farther downwards. I think there is a further dislocation of an already displaced lens in the left eye. It was only then that I observed what I have shown the case for, viz., a fringe of suspensory ligament round the edge of the displaced lens. The appearance, different from that of the fine fibre of the suspensory ligament, when seen in its normal condition, I attribute to the retraction of the fibres when they became separated from the ciliary body.

Vestibular Nystagmus.

By Leslie Paton, F.R.C.S.

I have brought this up as a case of extremely easily excited nystagmus, of vestibular origin. In cases of vestibular disease nystagmus can be easily produced by syringing the ear with water and even in healthy subjects this can produce the condition, and the direction of the nystagmus can be altered by substituting hot for cold water or vice versa. In the present case, however, a violent nystagmus is produced by simply pressing gently with the finger in the meatus of the right ear. Mr. Sydney Scott reports that the case is one of vestibular fistula.

Blue-dotted Cataract.

By Leslie Paton, F.R.C.S.

I have brought this patient up because punctate cataracts are not very common. I only remember having seen one other, and that I operated upon. Vision is very little interfered with, and the ophthalmoscope reveals very little in the form of opacity. With correction, this patient has $\frac{6}{12}$ in one eye, $\frac{6}{9}$ in the other. Probably the blue appearance is not due to pigment at all but to a refractive peculiarity. Lord Rayleigh's explanation of the phenomenon is adopted by Hess, and may be briefly paraphrased: In an opalescent medium containing innumerable fine particles with a refractive index differing from that of the medium, the dispersion of light is inversely proportioned to the fourth power of its wave length. The parts of the crystalline lens presenting such irregularities of refractive index cause the dispersion chiefly of waves of short wave length, green, blue and purple. By diffuse light they are rendered visible, and as seen by daylight they appear blue and by lamp-light they appear green.

I removed one such cataract from a boy, aged 19, and it supported what Hess says, that the lens comes out easily and no soft material is left behind. This boy had no secondary cataract. It has been said to be a characteristic of these cases that they develop full opacity of the lens later in life. This boy had the same type of cataract in both eyes. I removed it from the eye in which vision was interfered with, leaving the other. I saw him recently, and he is using the eye from which the cataract was removed in preference to the other, as he sees better with that.

DISCUSSION.

Mr. CHARLES WRAY: Have any other members of the family that condition? I have seen several examples, and in nearly all the cases in children it has occurred in the parents. I have regarded these dots as analogous to the white spots on finger nails. These opacities do not increase: I have seen them in people up to 72 years of age.

Mr. BISHOP HARMAN: Are there any signs of "night-blindness" in the case shown? In two cases I have had under observation there was this symptom, apparently due to obstruction of vision by the blocking of the periphery of the lens, so that a large pupil did not permit of the entry of what light there might be. One was an elderly lady, in whose lenses there were both the blue dots and grey striæ of probably senile origin. One of her sons had congenital cataract; two others were healthy. The second case, also in a lady, had rings of the blue dots, and some "night-blindness." Her two daughters had normal eyes.

Mr. ERNEST CLARKE: I saw an old lady some years ago whose cataract presented a beautiful appearance, a sort of combination between mother-of-pearl and opal. When the cataract was removed, however, there was no sign of any colour. Probably in this case, as always, the colour was due to diffraction of light.

Mr. LESLIE PATON (in reply): I cannot answer about "night-blindness" in this case because the patient has other conditions which might cause it. She has disseminated sclerosis, and giddiness, and changes in the nerves. There is a history of heredity in some of the cases. I only saw this patient on Monday, and then could not get a family history.

Mass of Connective Tissue Covering the Disc.

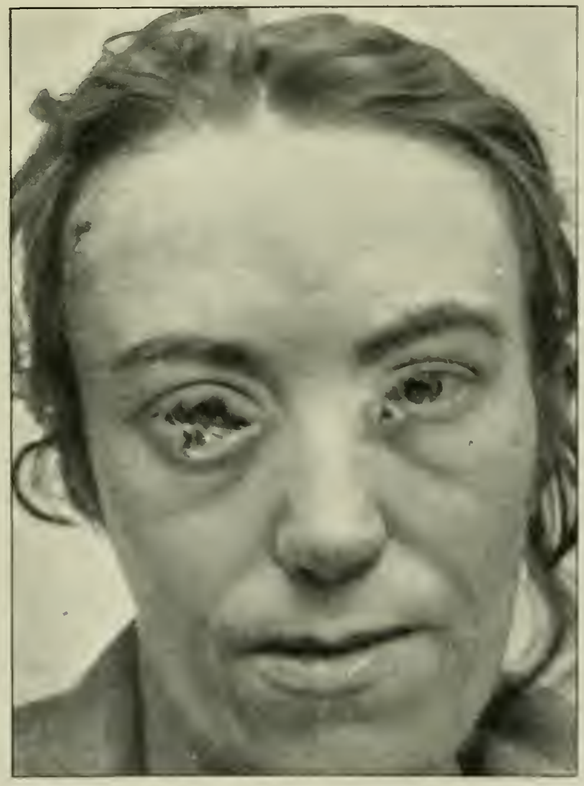
By LESLIE PATON, F.R.C.S.

WE have had one or two cases shown at the Section, especially that which Major Carruthers brought up two or three months ago, in which there was an extraordinary connective tissue mass covering the disc. The mass in this case raises the retina, and through it one can see choroidal disturbance. In the other eye, at the macula, there is a healed area of old choroiditis. There is a very definite history of tubercle. In early life bones in her right arm were scraped, probably for tubercle, and she is now in Queen Square Hospital with caries of the spine and compression paraplegia. The question is as to whether the mass at the head of the right disc is tubercular, and whether this may not throw some light on the nature of the other cases with a similar appearance.

Gummata of the Eyelid.

By M. S. Mayou, F.R.C.S.

The salvarsan treatment the patient has had has altered the appearance, but I show a photograph of it taken ten days ago, when she first came to see me. It shows the lid nearly eaten away. During the three days she was waiting for the Wassermann reaction, a large piece of the lid went: the process of destruction was very rapid. She had



salvarsan a week ago, and you will note the rapid improvement. Formerly it was a deep ulcer, with undermined edges: now it has become quiet and is rapidly healing.

DISCUSSION.

The PRESIDENT: We used to call these cases gummatous tarsitis. They have to be distinguished from multiple inflamed Meibomian cysts and from a chancre: the induration is much less than in a chancre.

Mr. M. S. MAYOU (in reply): I think it probably began as a gumma of the tarsus. When I first saw the patient there was nothing to see that was wrong with the left eye. I thought it was a primary chancre, except that there was no glandular infiltration. The Wassermann reaction was very strongly positive, which was also against a primary syphilitic lesion in the early stages. Then the other eye began to develop, and then we recognized its true nature.

Card Test for Colour-blindness.

By F. W. EDRIDGE-GREEN, C.B.E., M.D., F.R.C.S.

THE principle involved is the perception of difference between two colours presented in a special diagram of spots of irregular shape and various tones. On a ground of separate spots of one colour a letter is formed in spots of another colour. The test consists in discriminating between the colours, and hence recognizing the letters.

Before discussing in detail the use of the test, it should be remarked that a person of very acute colour vision can distinguish seven distinct colours (the so-called primary colours) in the solar spectrum—viz., red, orange, yellow, green, blue, indigo, violet—a person of normal colour vision can distinguish six (failing to discriminate between blue and indigo), and after this in descending order of acuteness of colour vision, there are those who can distinguish five, four, three, two colours, and finally the totally colour-blind, who distinguish no colours and see the whole spectrum as a shaded grey band. I call those who can distinguish seven colours, heptachromic, and those of less acute perception, in descending order, hexa-, penta-, tetra-, tri-, and dichromic. The cards are devised to enable the examiner to decide to which class the examinee belongs.

The new features of this test are: (*a*) The construction of a special diagram so arranged that there is no indication by way of form or tone given of any letter which is formed upon it. It will be seen that the diagram—i.e., the shape, relative position, and number of the spots is the same on all twenty-two cards, the only method of distinguishing the spots which form the letter being a difference of colour from the other spots. The examinee is forced to judge by colour perception alone.

(*b*) On a given card the colours exhibited are those which appear to be the same colour to the particular class of colour-blind which that card is designed to detect, each card having a special purpose. The colours correspond to portions of the spectrum which, when isolated, all appear of a uniform colour to a class of colour-blind.

(*c*) There are special cards for the detection of cases of shortening of the spectrum both at the red and the violet end — i.e., to detect inability to distinguish certain reds or violets from black, and also special cards to detect shortening of the spectrum combined with defective colour differentiation.

DISCUSSION.

Mr. J. HERBERT PARSONS: It is scarcely fair to criticize these colour tests under the conditions under which they have to be shown to-night. The principle is the same as Stilling's. I think they are a most useful subsidiary test for colour-blindness. Like the Holmgren wools, only more so, they give a very definite indication of the defect in a large number of cases. But probably Dr. Edridge-Green would not commit himself even to these for an absolute decision in a case of colour-blindness. I think he will find extraordinary difficulty in getting exactly the colours he wants reproduced for a number of sets. These we have seen now may be very excellent, but if you look through different editions of Stilling's work or even different copies of the same edition, you will see how the colours vary, probably due to the greater penetration of the colour into the lithographic stone in the later impressions, also to the fading of the aniline dyes.

Dr. EDRIDGE-GREEN (in reply): The essential point in any test for colour-blindness is that the colours should be exactly correct. One test may be quite efficient and a similar one, as for instance a copy of my lantern, quite useless. The originals of the diagrams were all checked by being used to examine special cases of colour-blindness. This process was repeated with the finished copies, then with the proofs, and finally with the finished tests. Tests in which a letter is drawn first and camouflaged after may be read by a clever colour-blind. Pseudo-isochromatic tests were used by Donders; many of these tests are so inefficient that the large majority of colour-blind will pass them.

Cyst of Orbital Portion of Lacrymal Gland.

By W. H. McMullen.

C. S., male, aged 60. Blow on left brow when aged 9, followed by slight drooping of lid which persisted. A swelling has been noticed for five or six years, gradually increasing; patient states it has nearly doubled in size during the past two and a half years.

Present condition: There is a rather irregular scar about 3½ cm. in length, extending obliquely down and out from just above brow across left external angular process. There is a regular ovoid swelling beneath the outer part of the left brow, measuring about 25 mm. by 16 mm. It lies immediately below the outer part of the upper orbital margin, the inner extremity being about 4 mm. external to the supra-orbital notch; the outer extremity reaches the outer margin of the orbit; the outer part of the lid is pushed down so that little more than half the cornea is exposed in the primary position. When the patient looks up only a quarter of the cornea remains visible. The skin moves freely over the swelling, and the lower part of it lies in front of the tarsus. It is adherent to the roof of the orbit. Fluctuation is easily obtainable and the swelling is quite translucent.

This is a case of cyst beneath the upper and outer part of the left orbital margin, which I believe to be a cyst of the orbital portion of the lacrymal gland. It is firmly adherent to the roof of the orbit, and is loosely attached to the tarsus. It does not present behind the lid, as the ordinary lacrymal cyst does, and it is very translucent. It appears that cysts of the orbital part of the lacrymal gland are rarities. Parsons, in his "Pathology of the Eye," states they probably only occur in association with new growths of the gland.

DISCUSSION.

Mr. Treacher Collins: There is one point which helps to confirm the diagnosis of cyst of the lacrymal gland in this case, which Mr. McMullen has not mentioned, namely, that the cyst begins to get bigger when the man goes out in a cold wind. I have noted that before in other cases.

Sir William Lister: This is evidently a case in which the cyst has come forward. I had an interesting case in the London Hospital some years ago, in which there was a cyst of the lacrymal gland which went behind the globe. The boy came with proptosis of his left eye, and I took him in and gave him treatment to see what would happen. The proptosis got worse, and the eye was pushed downwards and inwards, this showing there was something in the orbit upwards and outwards. It seemed to be a case for Krönlein's operation, which I performed on him. Having removed a triangular portion of bone and pushed that backwards, I made a horizontal incision through the periosteum, and came across orbital fat. Separation of that revealed a very black tumour. I was in the act of separating this tumour, quite easily, when suddenly the whole thing exploded in my face, and practically disappeared. It was mixed up with the lacrymal gland and the external rectus, and I was rather anxious about the patient at the time, because he had very marked acne of the face, and I feared the orbit might be infected from the skin. But the wound healed up by first intention. The vision had gone down to $\frac{6}{24}$, with marked papillœdema, but it rose to $\frac{6}{6}$, with only a slight restriction of the visual field. It was the only case of cyst of the lacrymal gland I have come across, and evidently it had gone backwards.

Mr. CHARLES WRAY: Would it not add very much to the ease of removal of these cysts if some of the contents were first let out by puncture, and then a suture passed round?

Mr. JEREMY: I removed a cyst similar to this three or four years ago, from the inner side of the upper lid, and on examining it with the microscope it was found to be lined with squamous epithelium, containing mucus. I regarded it as an implantation cyst. Does Mr. McMullen regard the cyst in his case as of that nature?

Mr. LESLIE PATON: I think the last speaker removed a cyst of Krause's gland, and my experience of dissecting out cysts of that gland is, that if you can get it away without puncturing, it is easy, but if you puncture, it becomes very difficult. Therefore I do not advise puncture before dissecting out.

Cobalt Glass as an Aid to Refraction.

By LESLIE PATON, F.R.C.S.

THE use of cobalt glass in subjective testing of errors of refraction is no new thing and I read the following note in a duly apologetic mood; but I have found that many of my friends have never used this aid at all and I personally find it frequently of value in testing intelligent patients. It is of special value in showing the axis of astigmatism rapidly. The principle on which the test is based is a simple one. Cobalt glass of a good quality cuts out all the rays of the spectrum between the red and the blue. Most of the cobalt glasses in the market are valueless, but Messrs. Hamblin have managed to secure an extremely good melting and have made up some specimens for members to try. The red rays are of low refrangibility, the blue rays of high refrangibility, consequently the blue rays are brought to a focus before the red rays. In an emmetropic eye the retina is placed at the focus of the white rays, and the blue rays by themselves would be focused in front of the retina and the red rays behind the retina, both sets intersecting on the retina and producing a uniformly cobalt-coloured image. In the hypermetropic eye the red rays form a halo round a blue centre; in the myopic eye the reverse is the case. In a case of mixed astigmatism a blue streak with red dots on each side is produced. In hypermetropic astigmatism the blue is not extended into a streak but remains as a blue dot between two red dots, and in myopic astigmatism we see a red centre with blue tips extending from it. It may be said that the method is of more scientific interest than practical value but I find it occasionally of distinct use.

DISCUSSION.

Mr. ERNEST CLARKE: Seventeen years ago I worked at this subject, and I referred to it in the first edition of my book on "Refraction." There I say, and I have not since had any reason to alter my view, that it is a pretty scientific toy, or experiment. It shows that the blue rays are refracted more than the red rays in passing through the dioptic media of the eye. I do not think it is likely to be of any practical use. First because it is a subjective test, and requires an extra amount of intelligence on the part of the patient, and secondly, because other tests—viz., retinoscopy and the ophthalmometer are so much more reliable. I think we all agree that the more objective our testing in refraction is, and the less subjective, the better. I have found that the cobalt glass gave very definite results with hypermetropia, in fact $\frac{1}{8}$ D. of hypermetropia and hypermetropic astigmatism is revealed; the blue centre and the red margin being very distinct, if a cobalt glass of the proper density is employed; but I have not obtained the same result with myopia or myopic astigmatism. Has Mr. Paton had the same experience?

Mr. N. BISHOP HARMAN: There is one reason why this glass should not be used habitually. There are certain intelligent patients who are very keen on small refinements; if once they had demonstrated to them a minimal defect which could not be corrected they would be insatiable. For example, I have some astigmatism, a small part of which cannot be corrected; if I wear 0·125 D. more than my present correction it is uncomfortable, and yet without that extra fraction I never fail to see the spread of the rays with all these light tests. If I were one of these "intelligent patients," I should be a bugbear to the examining ophthalmologist who proposed to correct my astigmatism. These subjective tests are, I think, best kept for our own instruction.

Mr. LESLIE PATON (in reply): It is not so easy to use in myopia. The red dots come out clearly in hypermetropic astigmatism, but when I test for astigmatism in a myope, I find it advisable to over-correct so as to produce hypermetropic astigmatism. The distance should be 5 to 6 metres. I worked away at one patient for two minutes, and then he told me he was colour-blind!

The Treatment of Prolapse of the Iris following Accidental Perforating Wounds, with a Note on the Removal of Non-magnetic Foreign Bodies from the Anterior Chamber.

By CHARLES GOULDEN, F.R.C.S.

PROLAPSE of the iris in connexion with accidental wounds of the eye is a fairly frequent experience. Many cases are not difficult to deal with, but on the other hand there are cases, especially when the wound in the cornea or corneo-sclera is an extensive or irregular one, which are difficult to manage. Individual experience even over a long period is never very extensive, but the numerous eye injuries that occurred during the war gave to those who were in charge of ophthalmic wards overseas an extraordinary opportunity of dealing with not only great numbers but an infinite variety of wounds of the eye associated with prolapse of the uvea.

Attempts at reposition of prolapsed iris should be avoided on account of the risk of infection of the interior of the eye, and because such a procedure is useless, as the reposed iris will again prolapse. Every attempt should be made not only to remove the piece of iris prolapsed, but the wound should be cleared of uvea; if left with iris incarcerated one or other of the following undesirable conditions will ensue:—

(*a*) Delayed healing of the wound.

(*b*) A weak scar which may bulge before the intra-ocular pressure.

(*c*) Defective vision due to a displacement of the pupil and irregular refraction of the cornea.

(*d*) Iridocyclitis.

(*e*) Sympathetic iridocyclitis.

In any case the appearance of the eye is much better with a well made iridectomy than with a distorted pupil and a leukoma adhærens.

When undertaking the removal of a prolapse of the iris a great deal depends upon the length of time that has passed since the accident, and no doubt cases that come into the hands of the surgeon several days after the iris first prolapsed are very much more difficult to deal with. It is impossible to lay down a definite time after which it is impossible or not advisable to operate. Cases can certainly be operated upon and the wound freed of uvea as long as ten days after the accident. A method of operating which makes these cases more easy to deal with will be mentioned a little later on.

It will be convenient to divide the cases into groups as follows :—

(I) Cases in which the wound is purely corneal.

(II) Cases in which the wound is at the corneo-scleral margin.

(III) Cases in which the wound extends past the limbus into the sclera.

If the prolapse of iris is complicated by the presence of a magnetic foreign body, this should receive attention and be removed from the eye through a separate incision before the prolapse is dealt with.

As a general rule the patient requires a general anæsthetic. This will allow deliberate operating, and the surgeon will be able to pull the iris in any direction he may consider necessary without feeling anxious lest the patient make some sudden movement which may lead to extensive tearing of the iris and bleeding that will hide everything that it is necessary to see so as to complete the operation properly.

(I) Cases in which the Wound is purely Corneal.

If the wound be small these cases are the easiest to treat. The prolapsed knuckle of iris should be seized in toothed iris forceps held in the right hand and the iris gently pulled in each direction, from side to side, to and from the ciliary attachment, the iris within the eye being observed all the time. It is important to make certain that there is perfect freedom of the iris where it is grasped by the edges of the corneal wound. Whilst drawing gently upon the iris seize, in the plane of the anterior surface of the cornea along the whole length of the prolapse with Couper's capsule forceps, release the hold with the iris forceps and drawing gently with the Couper's forceps cut off the iris with de Wecker's scissors pressing them gently upon the cornea whilst cutting. This cut must be done with one snip of the scissors, as the hæmorrhage which follows will most likely obscure the contents of the anterior chamber and will not allow a second attempt to be made with safety to the lens capsule. Usually the iris springs back into the anterior chamber leaving the pillars of the consequent coloboma quite free.

What has been done is, in effect, this : A portion of the iris not previously prolapsed has been drawn outside the eye, the iris has been put on the stretch, and the cut with the scissors has been made through healthy iris drawn outside the eye by the operator.

Now wounds of the cornea are usually radial, the perforating instrument piercing the eye directly from before backwards. It is in such cases that treatment is not difficult, but when the wound of the cornea is ragged, flap-like or tangential (like the wound made with a keratome) the problem of freeing the wound of the cornea completely is much more difficult. It is not difficult to free that portion of the iris that followed the path of withdrawal of the perforating instrument, as the repositor can be used passing it into the eye in the same plane and direction in which the perforating instrument entered the eye. But the repositor, however bent or manipulated, cannot be made to engage that portion of the iris which lies under the lower lip of the valve-shaped wound, and consequently that pillar of the coloboma is often left still engaged in or adherent to the wound in the cornea.

The plan that has been used with success in such cases is as follows : Before attempting to deal with the prolapse of iris, and whilst there is still an anterior chamber, make a small incision in the cornea at a point just opposite the accidental wound and 3 mm. within the limbus ; the wound need not be more than 4 mm. long. Now deal with the prolapse and free it as well as possible. After this the repositor may be used by inserting it within the eye through the incision made with the keratome and the pillars of the coloboma replaced. If a foreign body has been removed previously, the incision made for the removal may be used for inserting the repositor.

The above remarks apply to a case in which operation is undertaken within twenty-four hours of the accident, and before any firm adhesions have formed between the prolapsed iris and the edges of the corneal wound. If more than a day has passed, and especially if several days have passed, since the accident the operation is much more difficult. The iris will be covered with greyish lymph and sodden with exudate where it is prolapsed and the edges of the corneal wound softened and grey with infiltration. The lymph should be gently teased away from the iris with a repositor under a stream of warm saline solution, which will cause the prolapsed iris to be seen more easily as it is washed here and there by the stream of lotion, for it must be remembered that the iris is often almost completely bleached and much attenuated. It is now seized and carefully freed. This is best done by paying attention to each part of the corneal wound in turn. Draw the prolapse away from one side almost in the plane of the cornea, and whilst doing this, gently insert the iris repositor between the lip of the corneal wound and the iris, breaking down adhesions all the time until it is possible to pull uninjured iris from within the eye all along the edge of the wound in the cornea. This must be repeated in all directions, and then the iris cut off as described above.

(II) CASES IN WHICH THE WOUND IS AT THE CORNEO-SCLERAL MARGIN.

In these cases the wound is not infrequently punctiform, and, although the wound will allow the iris to prolapse, it is not sufficiently large to admit the iris repositor after the iris has been removed. Here again the advantage of the preliminary corneal incision will be seen.

(III) CASES IN WHICH THE WOUND EXTENDS PAST THE LIMBUS, THUS INVOLVING BOTH CORNEA AND SCLERA.

Here the corneo-scleral wound and subsequent prolapse of iris is complicated by a wound in the conjunctiva. As the conjunctiva, lacerated or cut, falls together and soon becomes adherent by its cut edges, it is difficult to judge the extent of the wound and the amount of uvea prolapsed; hence the importance of exposing freely the corneo-scleral wound and the inadvisability of attempting an operation until the conjunctiva has been drawn quite freely to one side. With an ordinary strabismus scissors extend the conjunctival wound, undermining if necessary, and then, having passed a suture through each edge, let your assistant draw the edges apart or attach a small artery forceps to the end of each suture and allow them to retract the wound by their own weight. The corneo-scleral is then beautifully exposed and the prolapsed iris and ciliary body can be seen along its whole length and deliberately dealt with. It is important that such wound in the sclera should be completely covered with conjunctiva, and unless the edges of the conjunctiva fall together easily and without tension, the conjunctiva should be undermined around the scleral wound and detached from the limbus on each side by cutting round the edge of the cornea. To ensure immediate closure of the wound the conjunctiva should not be sutured edge to edge, but by two or more U-shaped sutures passed a little distance from the edge of the conjunctival wound. The question of the use of scleral sutures will admit of discussion, but they are difficult to pass even when each is passed from the edge of the wound to the exterior, and the probability of bruising the eye severely and losing vitreous is very great. The conjunctival suture passed in the way described causes good adaptation of the edges of the scleral wound and braces them up quite satisfactorily.

A very troublesome type of case is one in which the wound has passed through the limbus on one side across the cornea and through the limbus on the other side. These cases will only permit of conservative treatment when the wound is just within the limbus; wounds which pass across the middle of the cornea will cause so much harm to the eye that it will have to be excised at once.

In those cases which allow an attempt to save the eye the wound usually passes across the lower part of the cornea, and it will be found that, after the prolapsed iris has been removed, the edges of the cornea do not fall into apposition and that any chance of healing is very small and the risk of intra-ocular equally great. Much more is this the case when the prolapse has been present several days, and the lower corneal flap accustomed, as it were, to its vicious position.

These wounds must therefore be liberally covered with conjunctiva with the sutures so placed in the conjunctiva as to help to approximate the edges of the corneal wound. The conjunctiva should be detached from the periphery of the cornea in about its lower third or over one-half and drawn straight across the cornea and sutured at the opposite limbus. Such sutures will hold from five to seven days, and when they have broken loose the conjunctiva will have become firmly adherent to the site of the injury.

A NOTE ON THE REMOVAL OF NON-MAGNETIC FOREIGN BODIES FROM THE ANTERIOR CHAMBER.

The removal of non-magnetic foreign bodies from the anterior chamber is usually not only very difficult but of extreme danger to the lens. It has been usual when a foreign body is lying on the iris (often in the lower part of the anterior chamber) to make an incision at the limbus with a keratome at the site of the foreign body and then introducing a curved iris forceps to try to seize the foreign body and so remove it. This often fails, and then an iridectomy is made in the hope of including the foreign body in the piece of iris removed.

The reason why it is so difficult or impossible to remove the foreign body in this way is largely due to the fact that the incision at the limbus is not on the same plane as the iris, and therefore not on the same plane as the foreign body, and consequently it is not possible in so small a wound sufficiently to depress the points of the forceps used so as to hold the foreign body securely. If the foreign body be at the angle of the anterior chamber there is a lip of the sclera between the forceps and the foreign body round which it is not possible to manœuvre the point of the forceps, and the attempt becomes an impossibility. Again, if the first attempt fail, the iris will, in all probability, be injured and blood will cover the field of operation. The following plan simplifies operation: Make a keratome incision in the cornea half way between the centre and the limbus (i.e., 3 mm. inside the limbus) in the quadrant of the cornea lying over the foreign body and with the point of the keratome driven towards the foreign body. If the point of the keratome seems to drive too near the angle of the chamber the wound may be enlarged by side-to-side movements instead of by an onward thrust. The foreign body may now be easily seized with toothless iris forceps and withdrawn. The risk of prolapse of iris is also avoided, as with corneal incisions in this situation the iris does not prolapse as is so often the case in incisions at the limbus.

In a case in which an eyelash was lying on the lens and no part lying on the iris a similar procedure was adopted, but the lash was first disengaged and gently coaxed over the iris by a stream of saline solution through the silver nozzle of a M'Keown set of irrigators devised for the treatment of immature cataract.

DISCUSSION.

The PRESIDENT: This is an exceedingly valuable and very suggestive paper. Most of us have been confronted with these difficulties, and we have always felt the great responsibility in deciding what is the best course to take. This paper makes some of the difficult matters clearer.

Sir WILLIAM LISTER: Mr. Goulden's paper is most illuminating. I was not very clear about the very difficult situation when there is a wound across the cornea, well away from the margin, and the iris well caught in. How would Mr. Goulden propose to free the peripheral portion of the iris from the lip of the wound? It is extraordinarily difficult to have the iris hanging out. If it is pulled on it may cause a tremendous amount of irido-dialysis. I do not know whether he suggests making a further incision from the opposite side of the wound and pass a repositor behind the iris. I saw Mr. Goulden do several of these procedures of which he speaks, and they came off most beautifully.

(Mr. GOULDEN described the method by the aid of the blackboard).

Section of Ophthalmology.

President—Mr. W. T. Holmes Spicer, F.R.C.S.

Perithelioma of the Lid.[1]

By R. S. Charsley.

Patient a male, aged 60. He is now dead, but I have brought specimens, as I thought it was an unusual kind of case. He was brought to me by his medical attendant at the end of last December, in perfect health. The doctor had mistaken this tumour for an ordinary Meibomian cyst: he had incised it and tried to scrape it out. Under cocaine I immediately removed the tumour, which was 25 mm. long and 15 mm. wide. The man continued in good health for two months, and then the pre-auricular gland began to enlarge, and there was some loss of vigour. Two months subsequently to that he developed weakness in the spine, and ultimately curvature, and the opinion was that recurrence had taken place in the vertebræ. The man died six months after the tumour first became noticeable. I particularly asked the medical attendant to let me know when the end came, as I wanted to get the gland to examine: but I did not know of his death until a fortnight after it had occurred.

As far as I have been able to gather from the literature to which I have access, perithelioma appears to be exceptionally uncommon, only three or four instances having, I think, been seen. Dr. F. E. Taylor, pathologist at Charing Cross Hospital, to whom I showed the specimen, named it perithelioma, and I would particularly like to hear the views of any pathologists present. Peritheliomata are described as being of only slight malignancy, but the opposite was the case here. The tumour was encapsuled and easily removed, being closely adherent only to the edge of the lid, 5 mm. of which it was necessary to remove with the growth. There was no local recurrence.

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[1] At a meeting of the Section, held June 9, 1920.

DISCUSSION.

Mr. TREACHER COLLINS: I do not see that there is any definite relationship between the cells of the growth and the vessel walls. It has an alveolar structure: I should describe it as an alveolar sarcoma.

Mr. LESLIE PATON: It may be of interest if I refer to the history of a case of my own, one of endothelioma of the orbit. The case was shown at the Ophthalmological Society in 1904, and specimens of the growth were shown the same year. The interesting feature in that case was the method of transmission. The growth was freely removed, but recurred locally, and the contents of the orbit were removed. Within three months the patient had an enlargement of the pre-auricular gland, and of a submaxillary gland. Mr. Pepper himself removed them, and they also were examined, and were found to be of the same nature as the original orbital growth. There was a gradual spread until the axillary glands became involved, and a subsequent spread to the mediastinum. The patient (a female) ultimately died within two years of the removal of the original growth. There was a huge mediastinal growth, which, again, was of the same nature as the first tumour. The interest of the case lies in the transmission of a growth of this nature by lymphatic channels. The early history of the case is given in the *Transactions of the Ophthalmological Society*, 1905, xxv, p. 240.

Mr. M. S. MAYOU: The cells of this tumour are particularly large, and I think the diagnosis of perithelioma is the most likely one. It is difficult to separate some of the endothelial tumours into the various groups: they often take an areolar arrangement. I have seen several endotheliomata of the large celled type, such as this, and they seemed to be very malignant indeed, as this was. The tumours when large in size broke down into soft caseous-looking material. I have one specimen of endothelioma of the lid, but it is composed of round cells.

Case of von Recklinghausen's Disease.

By ARTHUR W. ORMOND, F.R.C.S.

E. J., AGED 28, gardener, came to Guy's Hospital on April 26, 1920, complaining of headache and failure of sight. Vision: Right eye, $\frac{6}{18}$ some; left eye, $\frac{6}{18}$. Post-neuritic optic atrophy in both eyes; rather more marked in right eye. Colour vision defective. Fields of vision very contracted. Wassermann reaction negative. Hands and feet are larger than normal. Supra-orbital ridges prominent. Kyphosis, slight degree. The headache is referred to the right side and also the occipital region. The history of failing sight dates back vaguely to the time when he was in the Labour Corps in France, and he attributes it to the uncongenial task of acting as grave-digger. He is mentally slow, being unable to read or write or to do more than the simplest arithmetic.

Over the covered parts of the trunk, both front and back, extending from the neck to the thighs, are extensive areas of pigmentation with freckle-like blotches and some areas of light brown extending to 2 or 3 in. in diameter. This pigmentation has been noticed from childhood, but some of the areas patient thinks have increased within his memory and become darker. In the skin and subcutaneous tissues are some soft non-fluctuating masses, which feel like fibro-lipomata, and are not painful to touch. These tumours vary in size from that of a pea to a mass 1½ in. across. There is one large one in his scalp, another over the left scapula; others of smaller size can be seen in various places, and on looking along the surface of the skin in a good light a large number of undulations too small and smooth to be felt with the finger can be observed. The heart is normal. Systolic blood pressure, 105; diastolic, 80. Respiratory system normal, except that the chest is irregularly shaped.

Patient is one of a family of twelve children, six boys and six girls. He has always been over-developed, and was in 1915 much bigger and stouter than at present; the last two years he has been losing flesh. His appetite sometimes fails for days together, his mother says, and his sight varies considerably at different times. The stoop and general change was noticed in 1917 first of all, but the changes must be very gradual, as the dates given by his mother are quite vague. His previous maladies seem to have been the usual exanthemata and three or four attacks of quinsy. There is a history of fits: one which took place in 1919, and nearly resulted in the death of the patient by being run over.

The interesting point is the association between the two classes of symptoms; those associated with the neurofibromatosis, and those which are referable to some increased action in the pituitary body. I think the symptoms rather suggest that at some time the patient has had hyperpituitarism, but in the last three or four years the symptoms have rather suggested hypopituitarism. One would like to be able to attribute all the symptoms to one cause: and to my mind it would be possible—though

I agree it would be impossible to prove it—that he may have some sort of condition affecting the fibrous tissue in the base of his brain, which might explain the pituitary trouble, also the optic neuritis and his general symptoms. Although it is true that the optic nerve is not a peripheral nerve—that it is part of the brain—yet Alexis Thomson has quoted four cases in which the optic nerve was involved, and that authority clearly states that in different cases the whole of the cranial nerves have been implicated. I show you the skiagram, which indicates that the sella turcica is small, but there is some interference with the upper part, as if it were pressed down by the enlarged clinoid processes. The stereoscopic view shows it better: the anterior and posterior clinoid processes meet over the sella turcica.

DISCUSSION.

Mr. J. H. FISHER: I have nothing useful to say about this case, but it is a very interesting one. A fibrosis implicating the pituitary body is a feasible proposition. The difficulty is that the main pituitary symptoms here appear to be those of hyperpituitarism, inducing the acromegalic features. If Mr. Ormond's view is correct, one has to imagine that in the early fibrous developments in the neighbourhood of the pituitary body there was produced some irritation of the pituitary gland, which led to an increase in its secretion, and to the acromegaly which still persists. There seems to be no enlargement of the sella turcica, and that lends support to the idea that Mr. Ormond holds. I do not know enough about the pathology of von Recklinghausen's disease to carry the matter any further than that. I do not suppose, for example, that there is any association between that disease and the condition of any other of the ductless glands.

Mr. A. W. ORMOND (in reply): I suppose the pigmentation may have a relationship to some involvement of other glands, such as the suprarenals: but here there is no pigmentation of mucous membranes, such as is found in Addison's disease. In this patient the pigmentation is rather that described by von Recklinghausen as enlarged freckles, and he considered the pigmentation was most marked over various prominences, where pressure is borne. This man has one over the scapula, for instance, where, when lying in bed, there would be pressure: also a kind of mole on the right hip. Pain does not seem to have generally been a marked feature in these cases. This man says he gets pain of a shooting character in the lesions sometimes, and that they ache, but he has never complained that they are tender when they have been manipulated and examined. Therefore I imagine the nerves themselves are well protected: the change is in the fibrous tissue of the nerve sheaths. Still, with such a name as neurofibromatosis attached to a condition one would expect there to be pain. There have been no nerve symptoms in this case.

Cyst of the Iris.

By W. H. McMullen, F.R.C.S.

F. M., a girl, aged 17. Nothing wrong was noticed until patient was a year old, when a small spot was observed in the left eye. This increased gradually in size. She attended at the out-patients' department under Mr. Hartridge at the Westminster Hospital for six months. He operated on her at the Royal Westminster Ophthalmic Hospital on January 19, 1905. The note on her out-patient letter reads: "Small iridectomy with removal of the cyst." Condition before operation is not described. She came to the Royal Westminster Ophthalmic Hospital on May 21, 1920, complaining of slight soreness of the left eye and pain in the right brow. She also "thought the film was growing slightly over the eye."

Present condition: Coloboma of iris in left eye, extending from "7.30 to 11 o'clock." Occupying the periphery of the anterior chamber in the region of the coloboma with the exception of the extreme lower part is a greyish thin-walled translucent cyst. It consists of two parts, a rounded head above, 3 mm. in diameter, and a fusiform body below, 5·5 mm. in length with a maximum width of 2 mm. The head overlaps the upper inner part of the body of the cyst, the two parts being divided by a very marked constriction. The appearance at first sight suggests the presence of two contiguous cysts, but on close examination no septum can be made out between the two parts, and the cavity appears to be continuous. The whole of the anterior surface of the head of the cyst is in contact with the cornea. Only the outer part of the lower segment appears to touch the cornea. Attenuated iris tissue can be seen on both the anterior and posterior surfaces of the upper part. There is a small deep-seated opacity in the cornea over the lower part of the cyst. On the surface of the lens capsule there is a fine deposit of pigment. A small opacity is present deep in the lower outer segment of the lens, and a few peripheral cortical striæ in the posterior layers below. The fundus is normal. Tension normal. Left vision, $\frac{6}{9}$ part; $\bar{c}$ − 1·5 cyl. 20°/ = $\frac{6}{6}$ part. Right vision, $\frac{6}{5}$ part. Right eye normal.

The commonest cause of cyst in the anterior part of the iris, I suppose, is injury, with the implantation of epithelium. There is no history of injury in this case. The child was born in natural, though prolonged, labour; no instruments were used. The mother says there were no signs of bruising or injury about the eye at the time of birth, and has no recollection of any subsequent injury. As a spot was noticed when the child was almost a year old, it must be regarded either as congenital, or as coming on at a very early age. As there was no injury leading to implantation of epithelium, it must be a serous or endothelial cyst. The vision is good and the eye is giving no trouble, therefore I propose to leave the eye alone. Assuming it to be an endothelial cyst, Mr. Collins has suggested that simple transfixion should suffice to bring about its disappearance.

DISCUSSION.

Mr. J. H. Fisher: This case reminds me very much of one under my charge which was undoubtedly an implantation cyst, the result of injury. Sir William Lister will probably recollect it. The patient was an officer who was serving in the war, and I came across him first when he was a schoolboy and had an injury, a puncture wound with prolapsed iris. My operation left him with a coloboma beneath the upper lid; he had full vision. Then he served in the war, and developed a cyst in the lowest part of the angle of the anterior chamber. The appearance was exactly as in this present case. He came under my care again, and I dealt with that cyst first of all by simple puncture, and subsequently by more complete transfixion. That temporarily improved matters, but the cyst refilled. Eventually, I was compelled to do iridectomy in the downward situation, so that he now has a coloboma upwards, and another downwards. It is three or four years since the final operation, and there has been no recurrence. While accepting Mr. McMullen's history as he gleaned it in this case, I think there must have been some injury, a small puncture, which was unobserved at the time.

Mr. Treacher Collins: Cysts of the iris do sometimes arise without injury, and it is very important to differentiate between endothelial and epithelial iris cysts. The latter are always secondary to perforation injury, and are due to the implantation of surface epithelium. The endothelial cysts arise spontaneously as the result of closure of the crypts on the surface of the iris; they are lymphatic cysts. The differentiation is important in regard to the treatment which should be adopted. It is obviously of no use simply to puncture an epithelial cyst, because as long as the epithelial lining remains, it may spread to other parts of the anterior chamber. An endothelial iris cyst may be cured by transfixion; in one such case which I treated in this way the cyst collapsed, and never re-formed. I have treated an epithelial cyst successfully by doing a wide iridectomy and getting the whole lining membrane of the cyst away so that no recurrence occurred.

Mr. J. H. FISHER (in further comment): Is it not against the idea of it being endothelial that the primary operation done at Westminster Ophthalmic Hospital was for a cyst? If it were endothelial, the wide iridectomy should have cured it, but it is not finally cured, and perhaps we now see it in a larger form than before. It seems likely it is a cyst which has been caused by some unnoticed perforating injury.

Sir WILLIAM LISTER: I had a case, abroad, that of a Frenchman who had a cyst of the iris. As it was non-translucent, the condition was thought to be a growth and the eye was removed. Sections were made of it, and this definitely proved that the cyst was lined by a number of layers of epithelial cells. There seemed therefore no doubt that it was a case of epithelial implantation cyst. There was, however, no history of any injury. It has always seemed to me that a possible explanation of implantation epithelial cysts arising without history of injury, is, that owing to the habit so common among women of casually putting needles and pins into pillows, or other places which may be handy, such as the front of their dress, a baby's cornea might easily be punctured when the child is put down hurriedly on a pillow or held against a woman's dress in which a needle or pin had been deposited, and no one would be aware of the injury which had been done.

Mr. M. S. MAYOU: I do not think that all the cases of lymphatic cyst of the iris end so happily after puncture as Mr. Collins's did, because I remember one case which I examined pathologically in which a piece of cyst wall was excised; it was lined by endothelium, and the patient got a recurrence just at the site of the original cyst. The surgeon again excised the cyst, and it was followed by sympathetic ophthalmia.

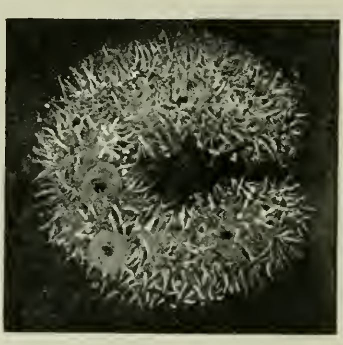
Mr. A. W. ORMOND: I have seen four cases in the last three or four years in children who have been brought to me with perforating wound, with a little tag of iris involved, cases in which nobody had the slightest idea as to how it happened. I think Sir William Lister's suggestion is not so far-fetched as it might at first appear to be. The wound would heal up, and nobody would know anything about it. In those four cases there was either actual prolapse of the iris, or adhesion of the iris to the posterior surface of the cornea.

Mr. GREEVES: I remember seeing a case of the kind when I was curator at Moorfields, a child with a cyst of the iris. An iridectomy was done, and the cyst removed. The cyst was lined by stratified epithelium, and we did not obtain any history of injury. I do not know what the subsequent course of that case was.

Symmetrical Crystal-like Deposits in the Region of Bowman's Membrane.

By W. T. HOLMES SPICER, F.R.C.S. (President).

I FIRST saw this patient a week ago; she is a healthy young woman, aged about 28. The only history obtained is that of gradual failure of sight for the last three or four years; there is no failure of sight in other members of the family. Her vision is $\frac{6}{12}$ in each eye. With the exception of the cornea the eyes are quite normal. There is a horseshoe shaped deposit in the centre of the cornea, occupying about one-fifth of the corneal diameter, the open part of the horseshoe being towards the nose. On magnification the surface of the cornea is quite bright. A little beneath the surface, in the region of Bowman's membrane, there is a fairly thick deposit of little golden-coloured lines, some of which end in minute spear-heads. They seem to be grouped round separate centres, and in two places there are little rosettes of radiating lines; where the deposits are densest there is quite a thick felt of lines, but at the edge the lines stand out against the background of perfectly clear cornea. The lines from their shape and brilliant appearance suggest organic crystals of some kind. The condition is a rare one; I do not think I have ever seen anything like it.



Since the case was shown at the meeting of the Section, I have found the record of another case from the late Mr. Devereux Marshall's clinic, which was brought to me by Mr. Hudson in December, 1915. The patient was a male, aged 30, sent to Moorfields by the military authorities for an opinion. There was no history of defective sight or of any trouble with his eyes; he thought his sight was perfect. He was a good shot at a miniature range. His vision was $\frac{6}{6}$ in each eye. In each eye there was a broad corneal arcus, and the centre of the cornea was occupied by a small grey disc of opacity. It appeared to be just below the surface, which was quite smooth. It was entirely made up of minute spikelets and dots suggesting crystals and identical in appearance with the first case. Mr. Hudson has also shown me corneal drawings of a whole family, some of whom had the crystal-like spikes, and others an appearance like that seen in the more usual nodular deposits. These are interesting as showing the probable affinity of the spikes.

Herpes Ophthalmicus in a Child, aged 3.

By G. W. Roll, F.R.C.S.

I saw this child a few days ago, and as it seemed an unusually early age for such a condition, I brought the patient here. The attack occurred in February, simultaneously with chicken-pox, the latter affecting the whole body, and the eruption remained on the brow. It has the typical distribution of herpes ophthalmicus, and the scarring is very definite. Some of it travels down the nasal nerve to the tip of the nose, and the cornea is affected too. There is no surface lesion and no involvement of the iris. The intimate connexion between chicken-pox and herpes ophthalmicus has been noted by many observers recently, and this appears to be a case in point. There was a case of a child, 6 years of age described by Dr. Barber, and that child had herpes ophthalmicus and herpes zoster at the same time. The mother of this child herself suffered from herpes zoster, some time previously I believe, so that it could not be a case of infection—if there be such—from the mother. Several epidemics of chicken-pox have been described in which there were a number of cases of herpes ophthalmicus: as many as forty-one cases were, I believe, observed at one time. There is no oculo-motor paralysis in this case. There is a point of surgical importance: I have noticed that in adults affected with cataract that if they show scars of herpes, it may be of years before, they recover very slowly. I believe the condition has been observed even in infancy—i.e., at a few months of age.

DISCUSSION.

The PRESIDENT: I suppose everyone has noticed the association between herpes ophthalmicus and chicken-pox. I have certainly had several cases in which there has been this association. The other point of interest about this case is the youth of the patient. I remember seeing some years ago a child with a corneal ulcer; it was part of a herpes ophthalmicus; there was no chicken-pox. The baby was under a year old, and the father had himself recently had herpes. These facts point to a rather low degree of infectivity, and to its possible conveyance from one person to another by herpes direct or by means of chicken-pox.

Mr. J. H. FISHER: With regard to the infectivity of this condition, I recall the case of a patient with herpes ophthalmicus whose wife had suffered from the same condition a few weeks earlier. I myself had a mild attack of herpes intercostalis, and at a short date afterwards my wife developed vesicles of the skin, which a physician to the London Fever Hospital pronounced to be chicken-pox, and she was isolated. I remember a family in which two members in the same household were patients—one had chicken-pox, the other herpes ophthalmicus. I have seen an infant under 12 months of age with herpes ophthalmicus, and I was struck by the fact that the baby was not inconvenienced by the attack, although it was a severe one from the cutaneous and ocular point of view; it sat up in its cot and was quite happy. And that has been pointed out in regard to herpes zoster generally: that the older the patient the more severe is the pain, hence, I presume, the younger the patient the less the pain. The oldest case of herpes ophthalmicus which has come under my observation was in a man 96 years of age, and a fatal result followed in that case in a few days.

Mr. ROLL (in reply): I think that whether there is pain or not depends on the involvement of the posterior columns of the grey matter of the cord; that is as far as I have been able to make out from a study of the matter. Unless those columns are involved, the hyperæsthesia or actual pain is not great.

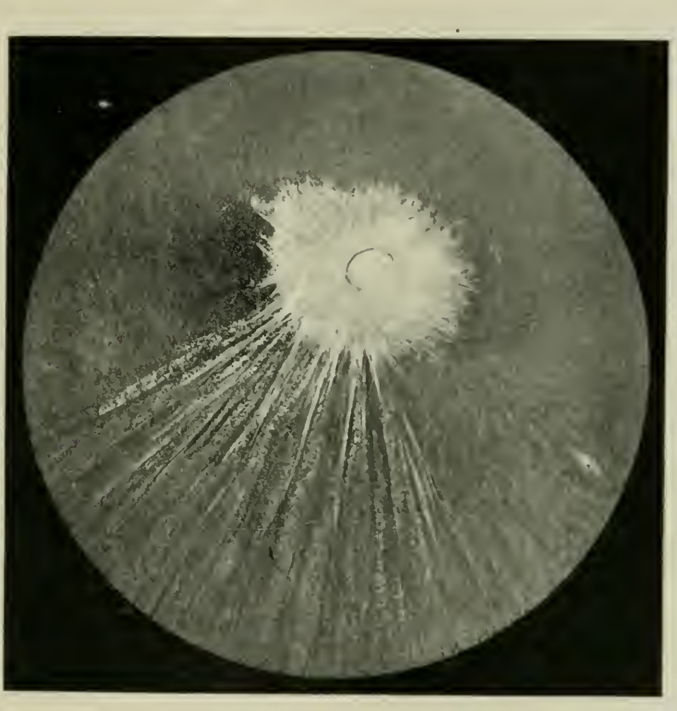
Mass Obscuring the Optic Disc.

By J. F. CUNNINGHAM, F.R.C.S.

PATIENT, a boy, aged 5. He was first seen on May 10, 1920, at the Central London Ophthalmic Hospital. The left eye had been found to be defective at a school inspection shortly before. He was struck in the left eye in August, 1919, with the handle of a scythe and had "a bad black eye" after it. Right vision: $\frac{6}{9}$. Left vision: No perception of light, tension normal.

An eye of a brother of the patient was excised for glioma of the retina in 1905, the child's age being 1½. He was under the care of Mr. Percy Flemming at the Royal London Ophthalmic Hospital (this statement has been verified). A maternal uncle lost an eye and later his life from the same disease fifty years ago.

There is a mass in the position of the nerve entrance coming far forward into the vitreous, and numerous folds are seen extending down and in from it. There is some choroidal disturbance in the region of the posterior pole.



During the month the condition has been under observation it has not altered in appearance. I consider it to be traumatic in origin. It comes soon after the interesting cases shown by Mr. Paton and Major Carruthers, in which swellings involved the optic nerve head.

Superficial Punctate Keratitis.

By Rayner D. Batten, M.D.

T. S., aged 33. Both corneæ are dotted over, especially in the central portion, with fine superficial depressions in the epithelium. The depressions or ulcers stain slightly with fluorescin, but the base of the depressions is clear and transparent. The condition causes pain and photophobia and considerable reduction in vision. The trouble began in August, 1918, after an operation for the removal of a piece of shrapnel from the kidney. I have attended him since June, 1919, but no permanent improvement has taken place under any form of treatment. He obtained temporary relief after having a peritomy done and the conjunctiva stitched over his cornea. His general health is very good, and no cause for the condition can be found. His teeth, nose, throat and blood have all been examined and the reports are all negative. There is no iritis. The pupils are equal and active and the fundus normal.

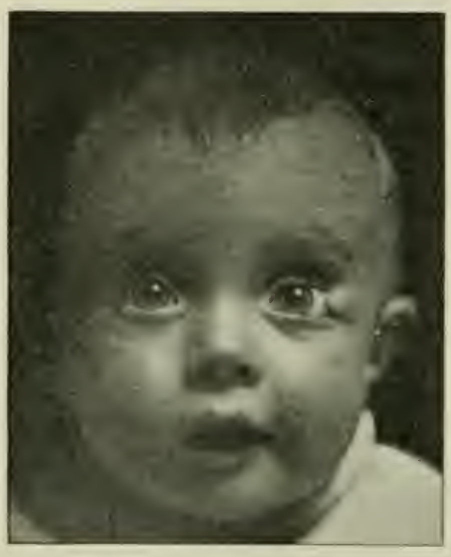
I think this condition is somewhat rare, and I have brought the case because of the extraordinary length of time it has been going on, certainly two years. No treatment I could think of, or which has been suggested to me, has had any effect on it. The man states it always abates in the evenings. I have always seen it in the early afternoon, which may account for the fact that no depressions, such as I describe in my account, can be detected this evening. Possibly the depressions heal over at night and become vesicles. I suppose it belongs to the herpes class. I shall be very glad of suggestions as to treatment.

The President: My experience is that there are two classes of these cases: one is a transitory kind, in which the condition lasts only a few days, and the other which lasts a number of years. Sometimes there is an invasion of the whole epithelium of the conjunctival sac, and there are the same punctate elevations on the conjunctiva and the upper lid, as on the cornea. I had one man with this condition in hospital for several months and treated him in every kind of way. My attempts varied from carbolizing the whole surface to stitching the conjunctiva over it. In the end the disease wore itself out.

Congenital Malformation in the Outer Canthus.

By W. H. McMullen, F.R.C.S.

This is quite different from anything I have ever seen before, and I cannot hear that other members have seen anything quite like it. The outer extremities of the left lids are separated by a low pad or cushion, covered with skin, which passes on to the conjunctiva. Various suggestions have been made to me this evening as to its nature; one is, that there is not really any tissue interposed between the lids, but that there is a notch in the upper lid, with displacement of the outer part. Some suggest that there is a dermoid there. I do not think there is a dermoid: there is only a thin cushion of subcutaneous tissue; there is no deformity of the outer margin of the orbit, and the limits of the subcutaneous swelling are not well defined. Many congenital defects of the lids have been attributed to adhesions of the amnion or pressure of amniotic bands. This theory as to their causation appears to be generally regarded as far-fetched and improbable. It would, however, explain this case rather nicely, if one assumed such an adhesion occurred



at the outer margin of the orbit before the development of the lids, preventing their union at the outer canthus.

Mr. TREACHER COLLINS: This case reminds me of a class of cases, the first of which was described by Sir George Berry in the *Ophthalmic Hospital Reports*, in which there was a notch in the lower lid at the outer canthus. In Berry's case it was symmetrical in two members of the same family. I have shown two cases of similar character: one of them closely resembled Mr. McMullen's shown this evening, though there was not quite such a bulging mass between the two divisions of the eyelids. In the two cases I showed, there was, besides the deformity in the eyelids, a marked deficiency in the development of the malar bones so that the two patients—who came to me independently—resembled one another so closely that they might be taken for brothers. I think the condition is best explained by assuming a delayed closure in the cleft between the fronto-nasal plate and the superior maxillary plate at the outer canthus: it is the position in which we meet with other congenital abnormalities, dermoid cysts, and fibro-fatty tumours of the conjunctiva continuous with the fat of the orbit, and due to some defect in development of the palpebral ligament in that situation.

Sebaceous Horn of Left Upper Lid becoming Malignant.

By M. L. HINE, M.D.

PATIENT, a male, aged 62. I first saw this patient at the end of January last, when he had a definite sebaceous horn in the middle of the left upper lid. He said it had been growing larger for the last two months, though he had had a very small lump on the lid for some two years. I excised the tumour on February 11: it shelled out after the ulcerated skin round the base had been cut away. It was not then examined microscopically. The patient disappeared till March 20, when the growth had extended considerably, the extension having occurred, according to the patient, only during the previous fortnight. The base was a little indurated and the surface fungating. I again excised the growth with some surrounding skin, and sent a portion for examination. The pathological report stated only that the "epithelium was thickened and keratinized." Instead of returning in a week, as requested, the patient again stayed away from observation and did not report till May 19, when a much larger fungating, ulcerating mass was seen covering a large portion of the left upper lid. The base was indurated. There was no glandular involvement. I sent him to the

Radium Institute at once, where he was kindly treated quickly. On May 27 Dr. Lynham wrote: "I inserted a radio-active needle in the growth this morning, and propose to ray him again, by means of a flat external apparatus, applied for several hours, to-night and to-morrow." Five days later a wonderful improvement had taken place; the fungating portion had come away, leaving a clean base, which has rapidly closed in during the past week. The growth now looks like a rodent ulcer in spite of the pathological report.

Postscript.—Unfortunately initial improvement under radium was temporary, and growth shortly afterwards began to extend superficially. The whole upper lid will have to be removed and a new lid fashioned from flap from forehead.—M. L. H.

The Corning Daylight Filter.

By W. Wallace, M.D.

This was the outcome of experiments made to find a light-filter for microscopy which would transmit light having daylight qualities, and enable histologists and bacteriologists to be independent of artificial illuminants the standards of which were not constant. It was suggested by Professor Gage, of Cornell University, and has been adopted in the United States by microscopists as well as by manufacturers into whose ordinary business the comparison of colours enters. By means of this light, the spectrum of which corresponds closely with that of sunlight, faint and delicate stains, which with some illuminants are inappreciable, are given their daylight value. Specimens of washes of water-colours are shown, which—the yellows especially—vanish completely or are altered in tone under the ordinary electric light, but are seen unchanged with the Corning filter. The unit consists of an aluminium reflector, unpainted inside. Into this is fitted a half-watt gas-filled Mazda lamp of 200 c.p. Beneath this is suspended the filter. This is a plate of blue glass, manufactured according to a determined formula. Each pot of the glass from which the plate is to be cast is tested beforehand by means of a photometer to ensure the necessary correction. The pattern shown is of use for colour work when the natural daylight has gone, also for comparing and matching tints.

Case of (?) Furrow Keratitis.

By P. G. Doyne.

Patient, a male, aged 72. He attended hospital on April 26, 1920, complaining that the eyes had been inflamed during the past fortnight. Vision: Right, $\frac{6}{24}$; left, $\frac{6}{18}$. The pupils and tension were normal. There was mild conjunctivitis, which has since largely subsided, and there was also some degree of ectropion of both lower lids. In both corneæ situated halfway between the limbus and the centre, there is a groove, most marked above, but extending all round the central zone of the cornea. The mesial edge of the groove is cliff-like and steep, the peripheral edge shallow. There is no staining of either cornea.

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Section of Otology.

President—Mr. Herbert Tilley, F.R.C.S.

PRESIDENT'S ADDRESS.

A Plea for the Better Teaching of Oto-Rhino-Laryngology.[1]

By Herbert Tilley, F.R.C.S.

(ABSTRACT.)

[This Address is printed *in extenso* in the *Lancet*, December 6, 1919, p. 1014.]

The teaching of otology, rhinology and laryngology in the medical schools of the United Kingdom must be made more efficient if English otology is to hold its own in the world.

The following are some of the principal reasons why students should possess a fuller knowledge of diseases of the ear, nose and throat than is obtainable under present systems of teaching :—

(1) Diseases of the ear, nose and throat are probably the commonest maladies met with in general practice and particularly in children. The regions thus involved cannot be seen and still less treated unless the practitioner has had a certain amount of training in the technique and use of instruments necessary for the examination of his patients. At present such training is not compulsory in many medical schools.

(2) The responsibility of diseased tonsils and adenoids for physical defects and backward mental development in the young.

(3) The frequency with which specific organisms gain access to the body by way of lymphatic structures in the fauces and nasopharynx.

(4) The frequency of acute infections of the ear in childhood, which lay the foundations of deafness in adult life.

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[1] At a meeting of the Section, held November 21, 1919.

(5) The principal cause of septic meningitis being neglect of suppurating lesions in the ear and accessory sinuses of the nose.

(6) The significance of laryngeal lesions in the early stages of tuberculosis, syphilis and cancer.

(7) The clinical significance of certain laryngeal symptoms in the early stages of grave intrathoracic disease—e.g., aneurysm—and in some affections of the central nervous system—e.g., paralyses of the cords in tabes.

A three months' compulsory course of study in an ear and throat clinic supplemented by simple anatomical, pathological and clinical demonstrations would go far to familiarize the student with the commoner affections of these regions which he is likely to meet in general practice.

The direct method of examining the air passages and the gullet has completely revolutionized the treatment of foreign bodies in these localities and is of increasing value in the diagnosis and treatment of pathological lesions situated therein. Many students have passed through their final examinations without ever having seen the direct method employed.

The student should also be made acquainted with the recent work on the physiology, pathology and surgical treatment of the "static" portion of the labyrinth, so important in the elucidation of many obscure cerebral symptoms.

With regard to the regulations in force for teaching the subjects of diseases of the ear, nose and throat in the United Kingdom, in six out of the teaching hospitals of London attendance is not compulsory for the conjoint student, though as a rule it is for those preparing for a University degree. In most of the provincial Universities attendance in the ear and throat departments is compulsory, and oto-rhino-laryngology may be chosen as a special subject for the M.D. or M.Ch. degree. This regulation, however, does not obtain, as it ought to do, in the University of London, but the question of this defect has been brought before the advanced Board of Medical Studies of London University by Mr. Arthur Cheatle and the author. It is much to be hoped that the Senate will permit the subject of diseases of the ear, nose and throat, to be taken as part of the M.S. degree.[1]

Such concessions would not only fit the student for better work in general practice but it would also attract some of the best class of men to devote their whole time to diseases of the ear, nose and throat, and thus evolve the highest type of "specialist"—viz., the man who builds up his special knowledge on a firm basis of general medicine and surgery. Post-graduate teaching in the special hospitals should be of invaluable assistance in imparting and extending further knowledge of our subjects to qualified practitioners. It should also be so organized that our overseas students will find all their needs supplied in the great medical centres of the United Kingdom and not, as heretofore, find their Meccas in Vienna, Berlin or Paris.

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[1] Since the above address was delivered the University of London has made this concession.

Finally, in the newly formed Ministry of Health, it is to be hoped that otologists and laryngologists will see to it that their department plays its very important part in caring for the ears and throats of the children of the Nation.

A Malformation of the Human Stapes.

By S. G. SHATTOCK, F.R.S., and S. R. TATTERSALL.

(Introduced by ARTHUR H. CHEATLE, F.R.C.S.)

CARIES of the ossicles, and ankylosis from disease are well-established conditions, but malformations appear to be of extreme rarity. The stapes here described exhibits a form of hypoplasia, and it is of interest to note that there were other defects of the same class present in the head. The child, a full-term, still-born male, had a cleft palate and bilateral hare-lip. The ex-occipital of the left side moreover was ossified from two centres. The specimen was obtained in the course of maceration of the head. On the right side the ossicles do not show any abnormality, but on the left the stapes is malformed. It is smaller and more slender than normal, and more so than the corresponding bone of the right side. The footplate, or base, of the bone is reduced to an oval process 1·7 mm. by 1·3 mm. in diameter, into which the anterior crus is inserted, the rest of the base being absent and the other crus terminating in a free, slightly bulbous end. On the tympanic aspect the footplate is deeply concave. The anterior crus itself is short, straight, and deeply grooved on the inner aspect. The posterior crus is shorter than the anterior; it is almost straight and diverges from the anterior crus at an angle of about 75°; its free end is smoothly rounded and slightly enlarged. The inner surface is grooved, though not so deeply as is the anterior crus. The long axis of the footplate does not lie in the same plane as the crura. The head and neck of the bone show no departure from the normal. These details will be clear by a reference to the accompanying figures. Fig. 1 shows the bone as viewed from the apex. Fig. 2 is from above, with the bone in its anatomical position. Both figures are twice the natural size.

In conclusion it may be added that in the left temporal bone the fenestra ovalis, although oval in form, with the longer axis directed downwards and forwards, corresponds precisely in form and size with the footplate of the malformed stapes.

The nearest approach to the malformation here recorded is the closure of the crus (columella formation) referred to by Politzer[1] as having been described by the older anatomists.

FIG. 1.

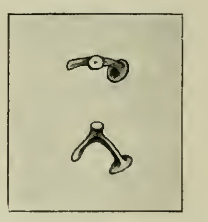

FIG. 2.

Malformation of human stapes.

DISCUSSION.

Dr. ALBERT GRAY : I think this is a peculiarly interesting specimen ; I have never seen anything like it. And it is very difficult to account for it. It may be due to a prenatal injury. On the other hand it may be a developmental error, and even then it is difficult to offer an explanation, because we know so little about the evolution of the oval window and the stapes. If it had been a defect in the round window we might have brought some knowledge to bear upon it. And embryology does not teach us much about the oval window. There is no stapes in any mammal similar to this except in the monotremes, in which the stapes is very much like this one. It is like a miniature columella, but in this case the crus which is left is inserted into one end of the stapes, whereas in monotremes it is inserted in the middle. If one wanted to find out the explanation of this developmental error, I think the animals to examine would be the amphibians ; no reptile has got a columella like that. I remember that in one of the frogs there is an appearance somewhat similar. I would like to know whether there was any stapedius muscle in this specimen.

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[1] "Diseases of the Ear," 5th ed., 1909, p. 27.

Mr. W. STUART-LOW: This appears to be a developmental reversion. Reptiles do not hear at all but have a very delicate sense of feeling through the skin. A snake learns of a person's approach through ground vibrations, not by means of an ear. [Dr. GRAY: I think there is a mistake—reptiles hear, but not fishes.] In answer to the remark of Dr. Gray I beg to say that my information is taken from Hill and Flack's latest edition of their "Physiology," in which it is clearly stated that reptiles and amphibians have very rudimentary hearing apparatus and practically do not hear. I have proved this myself by trying to make them hear by a visit to the reptile house at the Zoological Gardens with negative results.

Mr. SYDNEY SCOTT: The investigation of hearing in animals is very difficult. Some years ago, when testing reptiles in the reptile house of the Royal Zoological Society, I found a lizard which always raised its head and neck in response to a certain note on the Galton-Edelmann whistle, but took no notice of higher or lower tones. The late Dr. Plimmer showed me a bullfrog which evinced no sign of appreciating the sound of a pistol fired close beside it but which would croak in response to the croak of its companions. I could not awaken snakes with any tones of large or small Edelmann whistles, but these reptiles were immediately awakened by the rumbling and shaking of the ground produced by the heavy food trolley which moved on its rails around their house.

Congenital Redundant External Meatus; Repeated Abscess Formation; Excision.

By DAN McKENZIE, M.D.

THE subject of this rare congenital deformity is a little boy aged 7. At birth his doctor noticed a redundant auricle on the *right* side which he attempted to remove. At the age of one year the child developed an abscess behind the *left* auricle, which, apart from its being rather prominent, seemed to be normally formed, and this abscess, which pointed both into the meatus and behind and below the auricle externally, was opened and curetted by Mr. George Wilkinson of Sheffield, to whom I am indebted for these notes dealing with the early history of the case.

From the nature of the contents and the extent of the cavity Mr. Wilkinson suspected that he had to deal with a suppurating dermoid cyst, but nothing further was done at the time, as he considered that the septic condition of the cyst precluded an extensive dissection. Shortly afterwards the parents left Sheffield, and he lost sight of the case. At the operation, however, he removed the redundant auricle on the *right* side.

Since that time abscesses had formed behind and below the left auricle on two separate occasions, each being opened, and, after a time, closing up again.

About a month before I first saw the boy a third recurrence took place, and the abscess was opened for the fourth time in my absence on holiday by Mr. Archer Ryland, who reported that the trouble seemed to be confined to the soft parts, as he could not make out any connexion with bone.

When on August 20 of this year I first saw the patient there was a sinus discharging pus in the lower mastoid region, while another but much smaller opening was present in the postero-inferior wall of the external meatus close to the conchal margin. The tissues around the lower part of the ear were swollen and infiltrated, and pus could be pressed from a deep situation in front of the mastoid process and below the auricle. Temperature and pulse were normal. The tympanic membrane was also normal, and there did not seem to be any deafness.

The likelihood of some dermoid or other congenital malformation occurred to us, and our suspicions were confirmed on hearing from Mr. Wilkinson as above. Accordingly, the affected region was exposed by a post-aural incision, and, the auricle being displaced forward and the parts cleared, what seemed to be an additional external auditory meatus was removed (in two pieces). It consisted of a tubular structure about an inch in length and ending blindly. Cartilage could be felt in its walls, and it was lined with skin. It occupied a position anterior to the mastoid process and immediately below the normal meatus, the bony floor of which seemed to be deficient, but which otherwise seemed to be normal. No difficulty was experienced in effecting the separation of the two canals. The redundant meatus was surrounded, especially towards its outer end, by a collar of thick fibrous tissue, and this part of the tube, together with the tissues around it, was bathed in pus. The opening of the accessory meatus into the external meatus was quite minute, but it was skin-lined, and gave us the impression of its being a permanent fistula. The opening below and behind the auricle led first into a subcutaneous abscess cavity, and through that into the accessory meatus.

A substantial cartilaginous nodule lying close to the posterior surface of the normal auricular cartilage was also found and removed. It lay much higher than the redundant meatus, and seemed to be quite distinct from it, although it probably corresponds to the auricular cartilage of that meatus.

The child rapidly recovered after the removal of the meatus and is now quite well. Its removal, I must add, necessitated the clearing of the fistulous opening into the normal meatus.

The specimen has been examined by Dr. Wyatt Wingrave, who reports: "This specimen shows all the elements of an aural meatus, but distributed most erratically. There are islands of cartilage but not a cartilaginous plate. Glands (sebaceous) and hairs are embedded in a dense mass of fibrous tissue which has evidently formed the greater part of the tube, which is bounded by muscle fibres. Here and there, suppurating tracts or channels can be traced in the fibrous tissue. The lumen contains inflammatory products, e.g., leucocytes, epithelial cells, and many varieties of bacteria. We have then a tube simulating a meatus with suppurating sinuses connected with it. Its eccentric construction may be (1) original, or (2) the result of chronic inflammatory changes subsequent to its formation. There are the materials for a normal meatus, but the construction is imperfect as in other accessory organs."

We may suppose the course of events in this case to be somewhat as follows: The cavity of the redundant, skin-lined meatus filled slowly with secretion like a dermoid. This became infected, probably through the fine fistulous opening into the normal meatus; an abscess formed which discharged partly through the fistulous opening, but, as this was too small for sufficient drainage, also into the soft tissues behind and below the auricle, and thence externally.

This particular congenital malformation seems to be rare. I have not been able to trace any more than, at the most, eight cases recorded; in the majority of these the orifice and the external meatus are described as being divided by a septum or partition into two canals, one of which ends blindly while the other leads to the tympanic membrane. In only one case did the original appearances resemble those of the case just described, but the opening lay in the concha, while in our case the accessory meatus opened into the postero-inferior wall of the normal meatus close to its orifice. Abscess formation does not seem to have been encountered in any previous case.

Cases are recorded by Köhler, Bernard (bilateral), Macauln, Urbantschitsch (with an opening behind the auricle), but these are doubtfully congenital, according to Spira,[1] so that the cases in which the congenital nature is clear amount in all only to four.

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[1] *Mon. f. Ohrenhk.*, 1907, xli, p. 664.

The undoubted cases are Brieger's (1896); Guranowski's (1898);[1] Habermann's; and Spira's.

Hysterical Deaf-mutism of Eighteen Years' Duration.

By A. F. Hurst, M.D., and W. M. Mollison, M.Ch.

G. C., aged 18. His mother states that he heard perfectly well when he was a small baby, but she cannot give a definite account as to when he first became deaf. It appears that the deafness either began after a slight illness or after a fall on his head somewhere between the age of three and nine months. At a later date a number of aurists were consulted; they all, however, stated that this observation must be incorrect, and that he must have been deaf from birth. With great difficulty he was taught to speak, but he speaks like a deaf-mute, extremely indistinctly. He does not remember ever having heard anything at all, except on rare occasions during the last few years, when he thinks he has sometimes heard a loud noise, though possibly he really only felt the vibrations of the sound.

One of us (A. F. H.) saw him at Seale Hayne Hospital at the beginning of May, 1919; he appeared to be completely deaf, but the vestibular reactions were perfectly normal. It was therefore explained to him at the first meeting, by the aid of lip reading, at which he is an expert, that he could not hear because he had never tried to listen, and that if he once made the effort to listen he would begin to hear. On this first occasion he was taught to listen sufficiently to hear his name pronounced; this was the first word he ever remembered having heard. The same day he heard his bicycle bell and a motor horn for the first time. During the course of the next three weeks he learned to hear a number of words; but each word had to be taught separately, as although he could hear the sound, it conveyed nothing to him until he realized what the sound meant by lip reading. When once learned, he could understand it on a future occasion, but found it extremely difficult to continue to listen for more than a few moments, so progress was slow. At his best he could hear a familiar word from the other side of the room without the voice being raised, but frequently it was necessary to shout a word into his ear. At the same time he improved sufficiently to hear all ordinary sounds. He could hear a band

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[1] *Zeitschr. f. Ohrenhk.*, 1899, xxxiv, p. 245.

and he could hear himself playing on the piano, but he had no idea of pitch, and could not distinguish one note from another.

The conclusion was reached that, whatever its original cause, the deafness must have been temporary, but that, having come on at a period when he was just learning to listen, it interrupted his development in this respect, and he never learned to listen again; so that the deafness must really be regarded as hysterical, though of organic origin.

A recent examination by one of us (W. M. M.) shows that there is no evidence of disease of either middle or internal ear; in spite of this, before treatment, the deafness to both ear and bone conduction was absolutely complete, and the auditory motor reflex (dilatation of the pupil and blinking in response to loud sounds) was completely absent. The reflex is now normal.

DISCUSSION.

Dr. DAN McKENZIE: This is very interesting, from several points of view. About a year ago, the test for functional deafness by means of vestibular tests were very severely, even violently, criticized, and, I think, with justice. There are cases of nerve deafness in which the caloric tests are normal, and remain normal for a considerable time, possibly always, and yet there is no reason to suppose that the deafness is other than organic. I have reported a case in which it was due to a poison, probably that of mumps, an organic condition, and the caloric test was positive.[1] There has been recorded at least one case in which, after exposure to shell shock, the caloric tests were negative, while later they became positive. That is to say, had the patient been examined immediately after the accident, and reliance placed on the caloric test, the diagnosis would have been organic deafness, whereas, as time went on, it proved to be functional deafness. And it was shown by the caloric test being positive later on that the vestibular organ had suffered temporarily from the shock, just as the cochlea had suffered. But, allowing for those rare cases, I think we may nevertheless act upon this: that when the caloric test is normal in undoubted severe or absolute nerve deafness, there is a presumption that the case is functional, and that presumption is sufficiently strong to encourage us to persevere in the measures directed towards the treatment of functional deafness. This case brings that out beautifully, because, I take it, Dr. Hurst depended for his diagnosis on the fact that the caloric test was normal; at any rate it encouraged him to proceed with the idea of treating the case as one which was functional. And in this case he was correct. There is another interesting point which has often occurred to me in connexion with functional deafness. I believe functional deafness to be much more common than some people think. When people are seriously deaf, I think there enters into their case a functional element: that in addition to the withdrawal of the sense of hearing which the organic lesion induces, there is a dulling of the nerve centres for hearing, so that they gradually become functionally inert and cease to react to sound. It is those cases which respond to many forms of treatment which we decry. The question is, should we always close our eyes to such possibilities and condemn a treatment which awakens from their functional sleep those cells which only require to have either a stimulus from above by suggestion and training in attention, or from below by such methods of treatment? Can we see the case again in a year or so?

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[1] *Proceedings*, 1918, xi (Sect. Otol.), p. 79.

The PRESIDENT: I am interested to hear Dr. McKenzie's remarks on functional deafness, and its frequency in ordinary life. I wonder whether that is of the same nature which I have described as "domestic deafness": the husband does not hear his wife, or the wife her husband, but each of them hears anyone else without difficulty. I do not know whether it is that the constant hearing of one voice renders the nerve in any way exhausted so that it is incapable of appreciating a particular pitch as well as it does that of other people's voices, but I feel sure it is a very real type of deafness and not to be explained by mere inattention.

Mr. MARK HOVELL: This misuse of the term "hysterical" is greatly to be regretted. Taken literally, it may be supposed that Dr. Hurst and Mr. Mollison are under the belief that this young man's disability is due to some womb trouble.

Dr. DUNDAS GRANT: In regard to cases termed hysterical, I think it is the higher brain centres which have been out of gear, rather than the organs of hearing themselves. I take it that the hysterical state is one which has been suggested to the patient. There was probably some damage to the ear at the time of this child's fall, and though this has disappeared, the impression of inability to hear has persisted and may be said to have been "suggested" by the temporary deafness resulting from the injury, as we see in some military cases. There has been a sort of central torpor.

Dr. KELSON: A few weeks ago I had a case which bears on this. It was that of a lady who had been under my care with catarrhal deafness. After a time she got much better, but on hearing that her favourite child had to be operated on she became stone deaf suddenly, yet she regained her hearing after the child had made a good recovery. This was apparently functional.

Mr. W. STUART-LOW: Hysterical nerve deafness often takes the form of hyperæsthesia of the nerve, and I think that applies to the kind of case in husband and wife which the President has mentioned. Perhaps the lady, in such a case, cannot bear to hear her husband's voice, as such people may have a selective hyperæsthesia for certain tones, and that may be so in hysteria. I am sure Dr. Dan McKenzie is right in stating that there is a functional disturbance of the cortex, perhaps superadded to a condition of catarrhal deafness. I send these people to listen to music, and to the theatre to have a good laugh, for I am sure laughter has a bearing upon hearing, and wakens up the hysterically restrained cerebral cortical cells from the lethargy into which they have most probably sunk.

Mr. JENKINS: What is the patient's mental capacity? Does he show any mental inefficiency or insufficiency?

The PRESIDENT: Some of us noticed the dolico-cephalic shape of the youth's head, and his general unintelligent appearance, and we thought there might be some mental deficiency as well as the deaf-mutism.

Dr. ARTHUR F. HURST: I am glad the question of nomenclature has been raised, as I chose the expression "hysterical deafness" after the most careful consideration. The word "functional" would no doubt also be correct but it is not sufficiently definite, as functional merely means not organic, and there are many functional conditions besides hysteria. Although etymologically the word hysteria is an unfortunate one, it is impossible in this stage of the history of medicine to discard it, as no good substitute has been suggested, and it has become so universally accepted throughout the world.[1] During the war I had an exceptional experience of hysteria, and was led to modify the generally accepted definition in the following way: "Hysteria is a condition in which symptoms are present which have been produced by suggestion and are curable by psychotherapy."[2] It will be seen that nothing is said about any underlying mental condition, as I believe that there is nobody who may not develop hysterical symptoms if the suggestion is sufficiently powerful. It is true that the so-called hysterical type of person is more liable to develop such symptoms than other people, but this abnormal liability does not constitute hysteria any more than a tuberculous family history constitutes tuberculosis. In the case shown this afternoon the suggestion of deafness occurred during infancy, and was in the nature of an organic but temporary deafness, caused apparently by injury. There can be no more powerful suggestion of a permanent incapacity than a temporary incapacity of the same nature caused by organic injury or disease. The infant was learning to listen and to hear at this period, but being temporarily deafened he ceased to listen, and he was never taught to do so until eighteen years later, when he came under my care. Then the psychotherapy, in the form of explanation and re-education, was sufficient to teach him once more to hear, although of course a considerable amount of further education will be required before his hearing becomes normal. I do not think that he is in any way mentally deficient, but his apparent stupidity is due to the incompleteness of his education caused by his illness.

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[1] If Mr. Mark Hovell's criticism is meant seriously, we must presume that every time he speaks of gout and rheumatism he accepts the humoral pathology of the ancient Greeks.

[2] *See* "Scale Hayne Neurological Studies," i, No. 2, September, 1918, p. 106.

Vertigo in Conjunction with a Previous Radical Mastoid Operation.

By W. M. Mollison, M.Ch.

M. S., aged 36, was seen at Guy's Hospital in March, 1919, on account of attacks of severe vertigo: the attacks had begun four months before and occurred twice a week. Eight years ago a radical mastoid operation was performed on the left side and there was still some discharge: the man also suffered from persistent frontal headache and some tinnitus. Examination revealed an old perforation on the right side, on the left side there was otorrhœa from the radical cavity, on the highest part of the facial ridge and about the oval window there was a granulation.

The patient is extremely deaf. With the lateralizing test the fork is better heard in the left ear (radical operation side). There is no spontaneous nystagmus. Caloric stimulation produced a normal reaction on both sides, though that on the left was perhaps not so marked as that on the right side.

Is there enough evidence to suggest that an operation on the left labyrinth would cure the attacks of vertigo?

DISCUSSION.

Dr. Dan McKenzie: I do not feel qualified to express an opinion upon this, because I have had a precisely similar case, and I do not know what to do with the man. He is a soldier, and has had a radical mastoid operation, which has not done very well, as it has not healed. His labyrinth is functioning normally, and he can hear with it. His vestibular tests are normal too. If I open his labyrinth I think I may infect it, and set up labyrinthitis. I think it is a functional case, but the patient cannot work. He is not stone deaf; he can hear a loud voice.

Dr. Dundas Grant: Has Mr. Mollison tried the effect of small doses of quinine (say half or only a quarter of a grain) in cases in which there is vertigo from want of balance between the two labyrinths? The small quantity of quinine seems to quiet the more sensitive of the two and bring about fair balance.

Mr. W. M. Mollison (in reply): I have not tried quinine in small doses for this man. He has a granulation on the upper part of the facial ridge in the region of the oval window, between that and the round window, and my wish was to know whether it would be wise to open the labyrinth and destroy it, with the view of curing the vertigo. Mr. Cheatle, when speaking to me about the case, said he would open the mastoid wound again from behind, to see whether there was any fistula into the superior or external canal, and deal with that fistula in the first place, not opening the labyrinth until that had been done. I wondered if acute labyrinthitis might follow operation with that granulating area present. There is a large perforation in the other ear.

Mr. W. M. MOLLISON, M.Ch., showed :—

(1) *A Self-retaining Wound Opener for the Radical Mastoid Operation.*

(2) *An "Excavator" for opening Small Deep Cells in the Mastoid.*

Traumatic Facial Paralysis Produced in an Unusual Way.

By H. J. BANKS-DAVIS, M.B.

A BOY, aged 8, was climbing a wall, contrary to parental orders, when he fell and was impaled on a fine-pointed iron spike which perforated the tip of the mastoid process and produced instant facial paralysis. Reaction of degeneration on the left side was complete and the face is still paralysed after three months' electrical treatment.

The wound in the mastoid was minute and the skin lesion only required one stitch. Rapid healing resulted with no complications whatever. Hearing is unaffected, and an X-ray of the mastoid process was negative. The fine spike point must have penetrated the mastoid and "hit off" the facial nerve in the region of the stylomastoid foramen.

DISCUSSION.

Dr. DAN MCKENZIE : If these cases could be received early and operated upon, they would do well, and if seen soon after the accident an attempt to operate should always be made. Even if a tear takes place in the canal a chance of recovery is afforded by bringing the edges together.

Dr. H. J. BANKS-DAVIS : The boy is having regular electrical treatment and the palsy is diminishing. The nerve cannot therefore have been divided as originally reported. Mr. Douglas Guthrie, who is unable to be present, has sent me a few lines relating to the case of a boy, aged 11, who was drawing fence wire out of the ground when the loose end sprang up and entered the meatus, perforating the membrane and paralysing the facial nerve. The electrical treatment in some traumatic cases may have to be continued for as long as two years before recovery is complete.

Traumatic Destruction of One Labyrinth illustrating the "Marking-time" Test—a Simplification of the "Babinski-Weil" Test.

By J. Dundas Grant, M.D.

E. J., aged 29 (late gunner) had a fall of a distance of about 20 ft. in March, 1919, and states that he was unconscious for thirteen days. Deafness came on after the accident, almost complete on the right side and slight on the left. All signs point to elimination of the right labyrinth. There is no Romberg symptom, but on marking time with his eyes shut he turns round to the right. I offer this as a simplification of the Babinski-Weil test, though I do not consider its results quite so conclusive. It occurred to me that to cause the patient to mark time with his eyes shut would be a rough test as to his equilibrium on each separate foot, and I was struck with the rapidity with which he gradually pivoted round to the affected side.

In two cases out of a considerable number in which I have used the test, the patient turned towards the sound side, but in both of them there was an exceptional amount of general neurotic disturbance suggesting exaggerated functional activity of the vestibular portion of the damaged labyrinth.

Two Cases illustrating the Scope and Utility of Ossiculectomy.

By J. Dundas Grant, M.D.

Case I.—B. B., female, aged 21, was shown some months ago soon after the removal of her ossicles. Before the operation she was the subject of violent attacks of vertigo and headache with suppuration of several years' standing. The lower part of the membrane was absent and there was also a large perforation in the region of the attic; a mass of cholesteatoma ous material bulged from the latter opening and also below and in front of the neck of the malleus; it was very difficult to remove these masses and it was obvious that they could not be spontaneously extruded owing to the obstruction caused by the presence of the ossicles. Her hearing was reduced to a few inches for the whisper. After the removal of the ossicles the attacks of vertigo disappeared and although there is still some discharge and some ill-defined headache, the vertigo has entirely disappeared and her health is enormously better. The hearing has considerably improved.

Case II.—W. E., a middle-aged pensioner, had deafness of the right ear and discharge of about two and a half years' duration. In August of this year he developed attacks of giddiness. There was evidence of disease in the attic. As his hearing was extremely poor I decided to try the effect of ossiculectomy before resorting to the radical mastoid operation, the necessity for which seemed very probable. A week later he reported himself as feeling much easier.

DISCUSSION.

Dr. DUNDAS GRANT: I shall be happy to bring this subject up again on another occasion.

The PRESIDENT: Ossiculectomy seems to be a suitable subject for a general discussion. The tympanum of the man looked very clean and healthy, but there was a good deal of pus in the middle ear of the girl. Only this morning I saw at my clinic a case I had not seen for fifteen years. I asked an assistant at that time if he would remove the ossicles because there was caries of the incus. He used an incus hook to remove the incus, and severed the facial nerve, so that the right side of the face was completely paralysed. Her right face is still paralysed, but it is scarcely noticeable unless she laughs.

A Useful Attic Syringe.

By J. DUNDAS GRANT, M.D.

THE tip of the syringe is the same as that of Milligan's, but it is adapted to a U-shaped tube capable of holding about 10 minims of alcohol or other liquid. It is provided with an indiarubber tube and glass mouthpiece. The instrument is held by the U-tube and the liquid is drawn up into it by suction with the mouth. The tip plugs firmly into the U-tube and it has a ring pointing in the opposite direction from the curve of the extreme tip. It can, therefore, be rotated to any desired position. The necessity for such an instrument has been impressed upon me by the number of cases of attic disease presenting themselves at the Pensions Clinics, and it commends itself on account of the rapidity and ease with which it can be used.

The PRESIDENT: It seems to be forgotten by members of this Section that it is permissible to show cases of disease of the nasopharynx when they have any bearing on the organ of hearing, and it would add much to the clinical interest of these meetings if such cases were shown. I hope members will bear this in mind.

Section of Otology.

President—Mr. HERBERT TILLEY, F.R.C.S.

On some Anatomical Features of the Vestibule not previously recorded.[1]

By ALBERT A. GRAY, M.D.

I PROPOSE to describe some anatomical features of the human stapedio-vestibular articulation and adjacent regions which have not hitherto been recognized by anatomists.

The method of preparation is comparatively simple, and is as follows: The petrous portion of the temporal bone is removed from the skull, care being taken not to fracture the bone. The petrous portion is then sawn completely across with a fine fret-saw. This saw-cut must be made in a plane parallel with the footplate of the stapes. It should be made not more than 1 mm. internal to the footplate, and thus passes right through the vestibule, in an antero-posterior plane. The bone-dust and membranous structures of the vestibule are thoroughly removed by a jet of running water and a fine camel-hair brush. If the saw-cut has been made in the correct position the footplate of the stapes will be seen on its inner aspect, together with the adjacent regions of the bony vestibular wall. The semicircular canals and cochlea will have been sawn through.

The preparation is then dehydrated for a week or more in absolute alcohol. After that it is put for the same period of time into a mixture of equal parts of alcohol and xylol, and finally is put into pure xylol. It is ready for examination in a few days.

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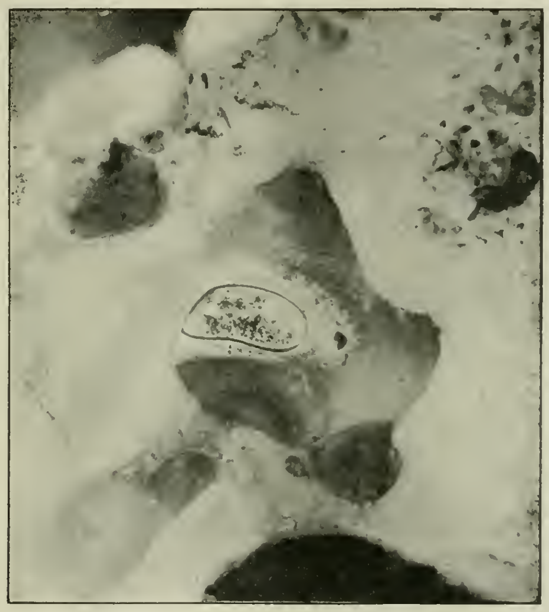
[1] At a meeting of the Section, held January 16, 1920.

If the preparation be now examined it will be seen that the clearing effect of the xylol has produced a change which reveals the fact that the bone surrounding the oval window has a structure different from that which is commonly assumed to exist. This effect is due to the considerable difference in the refractive indices of bony tissue and cartilaginous tissue, the latter being much more highly refractile than the former. Hence at any point at which the two tissues are in contact there is a considerable dispersion of light and the region becomes opaque, whereas in those parts in which there is a relatively large mass of either tissue it becomes more or less transparent. This effect is shown in slide 1, in which it will be seen that most of the bony surface of the preparation is homogeneous in appearance. The footplate of the stapes and a portion of the adjacent walls of the vestibule, however, present a speckled appearance, while the cartilage lining the articular surfaces where the stapes and the wall of the vestibule are in actual contact is rendered transparent, because it is pure cartilage without any bony elements in its composition.

Employing this method of preparation, I examined the region under discussion in ten temporal bones taken from ten different individuals, that is to say, one temporal bone from each subject. One bone was from a subject of the age of 14 at the time of his death ; the others were all from adults varying from the age of 30 to 60. Of the ten cases, the middle and inner ears were normal anatomically and physiologically in five ; uncomplicated chronic middle-ear suppuration was present in two ; chronic middle-ear suppuration, associated with septic meningitis, was present in two ; and chronic middle-ear suppuration associated with cerebral abscess was present in one.

As stated above, the footplate of the stapes and the adjacent tissue in the immediate vicinity of the oval window are not composed entirely of bony tissue in the ordinary sense, but of a mixture of bone and cartilage. As a consequence, this portion of the wall of the vestibule has the appearance of a band of speckled white surrounding the oval window. The cartilaginous element in the footplate of the stapes has long been recognized, hence there is no need to refer to it further. But, so far as the writer is aware, the cartilaginous element in the adjacent portions of the wall of the vestibule have not been described hitherto. In the portion above and below the footplate the band has a diameter equal to about one-third of that of the footplate itself in its small diameter ; but at the anterior and posterior regions the band is, in most specimens, considerably broader. Thus, in slides 1 and 4, at its anterior extremity, it projects downwards to a considerable distance and is deeply cleft. In the same two specimens it will be noticed that posteriorly also the band extends backwards to a considerable extent. The band is at its narrowest opposite the posterior third of the lower margin of the oval window (slide 2), and the next narrowest is at a corresponding point near the upper margin of the oval window. Thus the band is narrow at points opposite one another on the two horizontal walls of the oval window, near the junction of their middle and posterior thirds. It is important to notice this for reasons which will be given later.



Footplate of stapes and adjacent region as viewed from the vestibule.

In none of the ten cases examined was this semi-cartilaginous band absent. It varied, however, in different cases, both in breadth and also in the relative amounts of cartilage and bone of which it is composed. It was broadest in Case 7 and narrowest in Case 8, but the differences in breadth were not very great. As regards the relative amount of cartilage and bone present, the differences were much greater. In Cases 5 and 6 very little cartilage was present, and hence the band is hardly to be distinguished from the surrounding walls of the vestibule. In Cases 7 and 9, on the other hand, the cartilaginous element was predominant. Coincident middle-ear suppuration does not seem to have any effect upon this semi-cartilaginous band, since Cases 1 and 9 suffered from middle-ear disease, and in the latter both crura of the stapes had been eroded away by caries. In Cases 5 and 6, in which the cartilaginous element was only scantily present, the patients died from cerebral abscess and septic meningitis respectively; but there is no reason to suppose that these conditions were in any way associated with the relatively small amount of cartilage present in the structure under discussion. In none of the cases was there any septic infection in the labyrinth.

As regards the general anatomical significance of this cartilaginous element, I think there can be little doubt that it is a remnant of the original fœtal cartilaginous capsule of the labyrinth, in which the bony capsule develops later on. And the same probably holds true of the layer of cartilage which has long been known to line the vestibular surface of the footplate of the stapes. Minute islets of cartilage are also frequently found embedded in the bony capsule of the cochlea, as is well known, and indeed some of these may be seen in slides 7 and 9. Similar islets of cartilage are found in the walls of the semicircular canals, and no doubt all are remnants of the original cartilaginous capsule of the labyrinth. The islets of cartilage found in the cochlear portion of the wall of the labyrinth are supposed, as is well known, by some otologists to be a causative factor in otosclerosis. But, as the present writer has pointed out, they probably play a part that is passive rather than active.[1]

Another little anatomical detail hitherto unrecognized is revealed in these preparations. I refer to the deep cleft which passes forwards and downwards from the anterior margin of the oval window. It is seen in its most pronounced degree in slide 1, but it is also well developed in Case 4 (slide 4), and is quite plainly visible in slides 9 and 10. On the other hand, in slide 8 it is not present, and in slide 3 it is hardly distinguishable. In the remaining cases, 2, 5, 6 and 7, it is present, but is not seen in the illustrations because the photographs in these cases were taken from a direction which does not give a clear view of that particular region. In regard to this cleft, it is not possible, at present, to say what is its physiological significance, if indeed it has any. Similarly, its phylogenetic significance cannot be discussed until anatomical investigations have been made in animals other than man.

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[1] Gray, "Otosclerosis," p. 157. H. K. Lewis and Co., 1917.

These preparations reveal yet another anatomical feature in the labyrinth which, so far as I am aware, has not yet been described. This consists of a small foramen, which is to be found in the outer wall of the vestibule immediately behind the posterior margin of the oval window. It is seen most clearly in slides 4, 7, 8 and 10; and in slides 1 and 5 it is present but not so well seen. In slides 3, 6 and 9 it is not found. In slide 2 the saw-cut in making the preparation has removed the foramen if it was present.

It is not possible to say at present what purpose this foramen serves. Probably it serves to conduct a blood-vessel or a nerve and it should not be difficult by means of microscopic sections to decide the matter.

These preparations are further useful in determining the cadaveric position of the footplate of the stapes. But for this purpose a magnified stereoscopic image must be employed. When examined by that method it is to be found that, in the cadaver, the posterior third of the footplate is rotated inwards and the anterior two-thirds outwards. This was found in nine out of the ten cases examined. The remaining case was particularly interesting, because in it the head and both crura of the stapes had been eroded by caries, hence the stapedius muscle could not exercise any action upon the stapes. In this case there was no rotation of the footplate of the stapes. It appears probable, therefore, that either at the moment of death, or else during the rigor mortis, the stapedius muscle undergoes contraction and causes a rotation of the stapes round a vertical axis which passes through the footplate at the junction of the middle and posterior thirds. According to this evidence, therefore, the function of the stapedius muscle in man is to draw the anterior two-thirds of the bone outwards from the vestibule and at the same time to drive the posterior third inwards. This view is further supported by the fact that the annular ligament is longer at the anterior margin of the oval window than at the posterior margin, and would thus give greater freedom of movement to the anterior than to the posterior extremity of the stapes.

Note.—Referring once more to the band of cartilages in the wall of the vestibule in the region of the oval window, which is described at the beginning of this paper, it may not be out of place to make a short note in regard to its possible significance in pathological conditions. It is well known that the bony change in otosclerosis almost invariably first manifests itself in the wall of the labyrinth in front of the oval window, and it has been suggested that the reason for this lies in the fact that in this region there is a layer of cartilage which is liable to undergo the process of ossification. In view, however, of the anatomical structure described at the beginning of this paper, such an argument loses weight almost entirely. As described above, the layer of cartilage is no more abundant in front of the oval window than behind it. It is clear, therefore, that it is something more than the presence of cartilage in the wall of the labyrinth that determines the first bony change in otosclerosis to occur in the region in front of the oval window.

DISCUSSION.

The PRESIDENT: It is unnecessary for me to say how grateful we are to Dr. Gray for giving us a demonstration of this kind, nor to say how great an honour this kind of work confers on British otology. For many years original work on the specialty has been much neglected in this country and hence our indebtedness to Dr. Gray for his contribution of to-day.

Mr. SYDNEY SCOTT: In histological sections through the labyrinth with the stapes *in situ*, I have always been struck with the persistence and extent of cartilage cells, even late in life, certainly up to the age of 72. Dr. Gray's beautiful method of demonstrating these facts is new and he shows what I have not seen in sections, namely that cartilage extends across the central part of the footplate, as well as over the vestibular wall. The thick rings of cartilage supporting the attachment of the annular ligament are very constant.

Dr. A. A. GRAY (in reply): I must confess I had not noticed what Mr. Scott has just referred to until I made this discovery: then on looking at my microscopic sections I found it. These, however, do not give a picture of it, as seen here, *en masse*.

Meningitic Neuro-labyrinthitis.

By J. S. Fraser, F.R.C.S.Ed., and J. K. Milne Dickie, M.D.

(From the Laboratory of the Royal College of Physicians, Edinburgh.)

Though it is probable that the majority of cases of labyrinthitis result from middle-ear suppuration, a considerable minority are due to leptomeningitis. In the latter group labyrinthitis secondary to epidemic cerebro-spinal meningitis occupies the first place (Case III). The epidemic form of the disease is, however, not the only cause of meningitic labyrinthitis. Measles and pneumonia may be followed by meningitis and labyrinth suppuration (Case IV). It appears probable to the writers that Menière, jun., Brieger and Voss are right in regarding "mumps" deafness as secondary to meningitis. Osteomyelitic deafness may have the same pathology. The nerve deafness of secondary syphilis is closely associated with specific leptomeningitis.

The original site of infection may be the upper or lower respiratory tract, the parotid gland, the tonsil, the alimentary tract, or other part. In all cases, however, invasion of the blood-stream is probably the next step in the production of meningitis. We know that during an epidemic of "spotted fever" many people have meningococci in the nasopharynx for longer or shorter periods, and yet do not develop a blood infection and its consequent meningitis. Further, we know that of those who develop meningitis only a certain percentage become deaf. We do not know, however, why the labyrinth is affected in these latter cases. We may, if we like, put it down to some inherent weakness of the auditory and vestibular apparatus.

(A) DEAFNESS IN EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Netter states that deafness is the gravest complication of this disease. It is worst, on the whole, in the severest cases, though it may occur in the benign or abortive varieties. Deafness is most frequently noted in infancy, but then there are more cases in infants. As a rule the deafness comes on in the early stages—the first or second week, more rarely in the third week. In the great majority of cases both ears are affected. The onset is sudden and, according to Ker, there are seldom any warning symptoms such as tinnitus or giddiness, but, as Ker remarks, the patient is almost unconscious and does not complain. Foster and Gaskell state that deafness is less common in the posterior basic than in the epidemic form of the disease. In severe and protracted cases the deafness may only become evident after the return to consciousness, or it may develop more or less rapidly during convalescence. Rarely it is associated with blindness. Children as a rule become totally deaf and on recovery show a staggering gait (like the waddling gait of a duck) for some months or even a year after recovery. Adults, on the other hand, according to Politzer, rarely show total deafness. Statistics as to the incidence of deafness vary very much. Hildesheim noted only one case in 100. Schottmüller, in fifty cases, found only two (4 per cent.) suffering from deafness. Worster Drought and Mills Kennedy found permanent deafness in 4 per cent. of 160 cases (one case unilateral). Rolleston, in 502 naval cases, reported deafness in twenty-six (5·2 per cent.). Leszynsky, among fifty cases, had five with auditory nerve affection (10 per cent.). Gray states that in the 1906-7 epidemic in this country from 10 to 15 per cent. of the patients became deaf. Flexner gives a percentage of 12 to 33 in the pre-serum cases, and of only 3·5 in the cases treated by serum. Ker agrees that serum treatment has diminished the percentage incidence of deafness. He has only had nine cases of this complication among his last 216 patients. Three of the nine died. In three the deafness was total, and in the remaining three it was partial or limited to one side. In most of the nine cases deafness was present on admission. (Ker notes that deafness is by no means the only complication. He found arthritis in 10 to 15 per cent. of his patients as a result of blood infection, and states that orchitis may arise in the same way.) Altmann states that of sixty-three cases which recovered twelve were deaf (21 per cent.). Dufays reports deafness in 25 to 30 per cent. of patients, and Alt records percentages as high as 37·5. In nine of the fifty cases observed by Alt himself functional examination could not be carried out. Of the remaining forty-one twelve were deaf—i.e., 29 per cent. In five of these the deafness was present on the day of admission. The loss of hearing was sudden in all but one case. Alt notes that most of the deaf patients showed severe vestibular symptoms after recovery. The older the patient the sooner these symptoms pass off.

The deafness resulting from epidemic cerebro-spinal meningitis may be due to: (1) Hydrocephalus. This is probably the explanation of those cases which clear up after lumbar puncture, or after the recovery of the patient from the disease. (2) Purulent infiltration of the ependyma of the fourth ventricle. (3) Infiltration of the eighth nerve with purulent exudate. (4) Descending degeneration of the nerve structures of the cochlea following such purulent infiltration. (5) Purulent labyrinthitis. (6) Embolism of the internal auditory artery—i.e., septic embolism. (7) Otitis media?

It does not fall within the scope of this paper to deal with the path of infection in epidemic cerebro-spinal meningitis. We take the basal meningitis as an established fact and proceed to review the pathological changes which lead up to deafness. The stages of neurolabyrinthitis due to epidemic cerebro-spinal meningitis appear to be as follows: (1) Purulent infiltration within the arachnoid nerve sheath in the internal auditory meatus. If the infiltration stops at this point it would appear that deafness may nevertheless occur either from changes in the nerve or from secondary descending degeneration—i.e., atrophy of the spiral ganglion cells and nerve end organs. Alt's third case appears to be an almost pure example of this type. (2) Purulent invasion of the perilymphatic and, later, of the endolymphatic spaces of the cochlea, vestibule and canals—the invasion occurring along the nervous and vascular paths. When acute purulent labyrinthitis has reached a certain stage the pus may burst outwards through the windows into the tympanic cavity, and so give rise to meningitic otitis media. Even before such an actual bursting through occurs there may be congestion, swelling and infiltration of the mucosa of the window niches, with some exudate. Lucae, Heller, Nager, Alt (first case), Habermann (second case) have recorded microscopic examinations which showed this condition; our own Case III is a further example. (3) After the above stage has passed, if the patient lives long enough, there is formation of granulation tissue within the labyrinth. Habermann (first case), Larsen and Schwabach have recorded examinations of the labyrinth after death from epidemic cerebro-spinal meningitis which showed this stage. (4) Later on we have the formation within the labyrinth of new connective tissue and bone which partly or completely fills the hollow spaces of the inner ear. Alt's second case is an example, although the labyrinthitis had only existed for about eight weeks before death. Steinbrügge, Schultze, Moos, Scheibe, Baginsky, Theodore and Goerke have all recorded examples of this spontaneous cure of labyrinth suppuration with new bone formation.

(1) As an example of the results of the first or "purulent neuritis"

stage Alt's third case may be quoted : Male, aged 21, admitted February 7, 1907, with high fever, headache and vomiting. Herpes labialis present. Cervical tenderness. Knee-jerk absent. Marked nystagmus to the right. February 13, 1907 : Right abducens paralysis. Kernig positive. February 21, 1907 : Paralysis of right facial and aphasia. Patient cannot answer written questions. Complete deafness, with normal drumheads. Ptosis of left eye, convergent strabismus. Retraction of head. March 9, 1907 : Death. *Post-mortem :* Convolutions flattened and congested. Arachnoid thickened over cerebellum. Marked internal hydrocephalus. Ependyma thickened. Bronchi of lower lobes filled with pus. *Microscopical examination of left ear :* Internal meatus dilated, due to bony softening. A little pus present in the modiolus. The eighth nerve shows much infiltration between the bundles. Facial nerve less affected. Spiral ganglion cells infiltrated with connective tissue. Reissner's membrane depressed in basal coil. Membrana tectoria depressed and adherent to Corti's organ and Reissner's membrane. Corti's organ itself not recognizable as only a row of flat cells remains. Epithelium of stria vascularis not recognizable. Both scalæ are free from pus. Sacculus completely absent and replaced by connective tissue. Saccular nerve infiltrated with connective tissue. Utricle dilated and its sensory epithelium not recognizable. In the canals blood corpuscles and pigment are present in the connective tissue of the cristæ, which themselves appear shrunken. Bony and membranous canals almost unaltered. In the neighbourhood of the oval window there are strands of new formed connective tissue, with dilated vessels. There is some new connective tissue and bone formation in the region of the round window. The right ear shows much the same conditions.

(2) *Purulent Meningitic Labyrinthitis (with Secondary Otitis Media).*—Nager records the case of a male, aged 49. On the fifth day both middle ears were found to be normal. The patient was, however, completely deaf for voice, noises, tuning-forks, and whistle both by bone and air conduction. On the seventh day the patient died. *Microscopic examination of the ears* showed the whole labyrinth filled with homogeneous or cellular exudate. Both scalæ and the ductus cochlearis were affected. The cochlear canal was dilated, and only fragments of Corti's organ remained. The nerve endings of the vestibular apparatus were still recognizable but embedded in purulent exudate, while the perilymph space was filled with inflammatory products. The footplate of the stapes was pushed out, and at this

spot there were small masses of exudate in the tympanic cavity—i.e., the beginning of a meningitic otitis media. The nerve elements of the modiolus were surrounded by inflammatory œdema and the cochlear nerve embedded in pus.

Goerke has examined nineteen ears from cases of epidemic cerebrospinal meningitis. In seventeen there were more or less widespread changes in the labyrinth, even in cases in which the hearing was good till just before death. In three of the seventeen cases the cochlear aqueduct was the route of infection, while in eleven the meningitis extended along the eighth nerve to the labyrinth. In the three remaining cases there was a combination of the two routes. Goerke notes the unequal affection of the different parts of the labyrinth, e.g., in some cases the intravestibular and basal portion of the cochlea was mainly affected, while in others the canals were specially involved. The necrosis of the membranous labyrinth which occurs in these cases may be due to vascular thrombosis (Steinbrügge), or to toxins (Habermann). Politzer attributes the changes to the direct action of the meningococci themselves.

(3) *Formation of Granulation Tissue within the Labyrinth.*—Habermann has demonstrated complete destruction of the structures of the inner ear in a case of cerebro-spinal meningitis which had relapsed after complete deafness had occurred. The internal ear contained granulation tissue. In this case there was pus in the scalæ of the cochlea and in the vestibule. The joint between the stapes and the oval window was destroyed. The aqueduct of the cochlea was filled with granulation tissue. Habermann notes hæmorrhages in the sheaths of the acoustic nerve and necrosis of the nerve fibres.

(4) *Formation of New Connective Tissue and Bone in the Labyrinth.*—Alt's second case may be quoted as an example of the fourth stage: Male, aged 18, admitted with high fever, vomiting and headache. Herpes at left side of mouth. Paresis of right facial nerve. Kernig positive. Head bent backwards. Tenderness on pressure over cervical spine. Complete deafness with normal drumheads. Lumbar puncture gave cloudy fluid. Weichselbaum's micrococcus present. Death occurred *two months* after admission. *Post-mortem*: Hydrocephalus and meningitis; obliteration of foramen of Magendie; pleurisy; marasmus; adhesions between cerebellum and dura; ventricles much dilated; ependyma shows granulations. *Microscopic examination of left ear:* Internal meatus dilated and contains necrotic bone. Much pus around the acoustic nerve but only a little around the facial. The

cells of the geniculate ganglion stain badly and there are only slight remains of spiral ganglion cells. There is almost nothing left of the membranous labyrinth. The whole lumen is filled with new connective tissue containing dilated blood-vessels. Pus still present in parts. Semicircular canals filled with new bone. In the cochlea new bone formation is confined to the median part of both scalæ. Ductus endolymphaticus still recognizable. In the middle ear there is some pus in the niches and connective tissue around the stapes. Niche of the round window filled by a thick connective tissue layer. The membrane of the round window cannot be made out. In the *right ear* the changes are not so marked as in the left. Other observers have examined the ears of patients who died years after the attack of epidemic cerebro-spinal meningitis and have found the hollow spaces of the inner ear almost completely obliterated by connective tissue and bone formation.

OTITIS MEDIA IN EPIDEMIC CEREBRO-SPINAL MENINGITIS.

The middle ear may be involved by way of the Eustachian tube or by the pus in the inner ear bursting through the oval and round windows and so infecting the tympanic cavity. Opinions as to the frequency of otitis media vary greatly: thus Netter states that it is not common and is only found in advanced cases and at post-mortem examination. He admits, however, that the usual clinical signs of otitis media are not present, presumably on account of the condition of the patient. Altmann has examined twelve cases of deafness and found the middle ear unaffected. Dufays believes that the otitis media is due to the spread from the inner ear. On the other hand, Koerner states that otitis media is usually present in cases of deafness associated with epidemic cerebro-spinal meningitis. Goeppert found otitis media in 50 per cent. of cases suffering from epidemic cerebro-spinal meningitis and in 62 per cent. of post-mortems in this condition. Roosa found cicatrices in the drumhead in only eight out of fifty-four ears examined. The others were practically normal. On the basis of tuning-fork tests however he believes that a middle-ear affection is the main cause of the deafness. It is quite possible that the presence of otitis media may be merely a coincidence; thus Hessler records a case of acute middle-ear suppuration in epidemic cerebro-spinal meningitis in which meningococci were not found in the mastoid. Further, it seems likely that the incidence of otitis media may vary in different epidemics; certainly from the functional examination of cases of deafness following spotted fever it would appear that the labyrinth and eighth nerve are the structures involved.

Of 111 cases of deaf-mutism examined by one of us (J. S. F.) during the last two years at the Edinburgh Royal Institution for the Education of the Deaf and Dumb fourteen were due to meningitis. Of these fourteen, seven were cases of epidemic cerebro-spinal meningitis. Examination showed normal or only retracted drumheads in five out of the seven cases, while functional tests revealed absolute deafness and complete loss of vestibular reaction in six cases.

Alt saw only one case of acute suppurative otitis media without labyrinth involvement among his fifty cases, and in this there was no bacteriological examination of the pus from the middle ear. Alt had, however, seen a case of otitis media in 1897, in which the meningococcus was present along with other bacteria. Similar cases have been recorded by Frohmann and Netter. The former of these observers has reported a case in which there was a pure culture of the meningococcus in a bilateral otitis media. Rolleston reports otitis media in ten out of 502 cases. Fairley and Stewart saw two cases of suppurative otitis media in 450 patients. Worster-Drought and Mills Kennedy remark that chronic middle-ear suppuration may of course be present in patients suffering from epidemic cerebro-spinal meningitis; indeed they found this condition in seven out of 160 cases and, from the diagnostic point of view, call attention to the importance of the examination of films (meningococci) made from the aural discharge.

The conclusion seems to be that acute purulent (meningococcal) otitis media is not of frequent occurrence in "spotted fever." When it is met with it is probably secondary in most cases to meningitic labyrinthitis and is due to the pus in the labyrinth bursting out through the windows into the middle ear.

PROGNOSIS IN REGARD TO DEAFNESS.

Moos remarks that the percentage of cures and improvements is higher in the experience of the general practitioner, who observes cases during the epidemic, than in that of the specialist, who only sees the patients some weeks, months, or years after the outbreak of the disease. Sophian believes that cases of deafness which recover have been due to hydrocephalus. Such cases improve at once or clear up on lumbar puncture. It must be admitted that in the vast majority the deafness remains permanent. Of seven cases recorded by Alt, four remained totally deaf and in three the hearing returned to some extent. Politzer notes that occasionally there is some improvement after convalescence, but this is only temporary. Mygind records a case with recovery of hearing four years after the disease. Davis, on the other hand, has reported a case of gradually progressive bilateral deafness occurring three years after the attack. Of five cases recorded by Leszynsky, two were unilateral and three bilateral. Of the latter one remained completely deaf, while in the other two some slight improvement occurred on one side.

EPIDEMIC CEREBRO-SPINAL MENINGITIS AS A CAUSE OF DEAF-MUTISM.

Statistics on this point vary greatly. The discrepancy may be explained as follows : If there has been a recent outbreak of the disease in a country we have a high percentage of deaf-mutism due to epidemic meningitis, but if the country has been free from outbreaks for many years the proportion is low. Macleod Yearsley (1917) found that out of 1,114 cases of acquired deaf-mutism only eight were due to epidemic cerebro-spinal meningitis. Holger Mygind (1894) states that in only four out of 208 deaf-mutes was epidemic meningitis the cause of the affliction. Kerr Love (1893) has reported on 175 deaf and dumb children ; of these, eighty-one were cases of acquired deafness and among the latter thirteen were due to meningitis and brain fever, but apparently none of the cases had followed the epidemic form of meningitis. Hartmann (1880) has stated that 26·8 per cent. of deaf-mutes in his district were due to spotted fever. Roosa found that twenty-seven out of 147 deaf-mutes owed their condition to this disease. Our own statistics regarding a deaf-mute institution have already been given, but we may state here that of the thirty-four cases of deaf-mutism following "spotted fever" seen at Dr. Logan Turner's department during the years 1907-18 (inclusive), thirty were brought by the parents during the four years 1907-10.

MICROSCOPIC EXAMINATION OF THE EAR IN THREE CASES OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Case I.—Corporal G. was admitted to hospital on June 21, 1916. Patient had had violent headache for fourteen days, worse on the right side, with cerebral vomiting. Photophobia and opisthotonos present on admission. Temperature persistently subnormal. Pulse 40 to 60.

There was tenderness on tapping the right side of the head. Death on June 28, 1916.

Microscopical Examination of Right Ear.—The epithelium of the external meatus and outer surface of drumhead is desquamating. The tympanic membrane is distinctly thickened and shows well-marked new bone formation ("chalk patch" in drumhead (fig. 1)). In the tympanic cavity there is some new connective tissue between the ossicles and the outer wall of the attic. In the border cells the submucosa is thickened and there are "cystic spaces" in the mucous membrane. There is some old blood pigment in the submucous tissue.

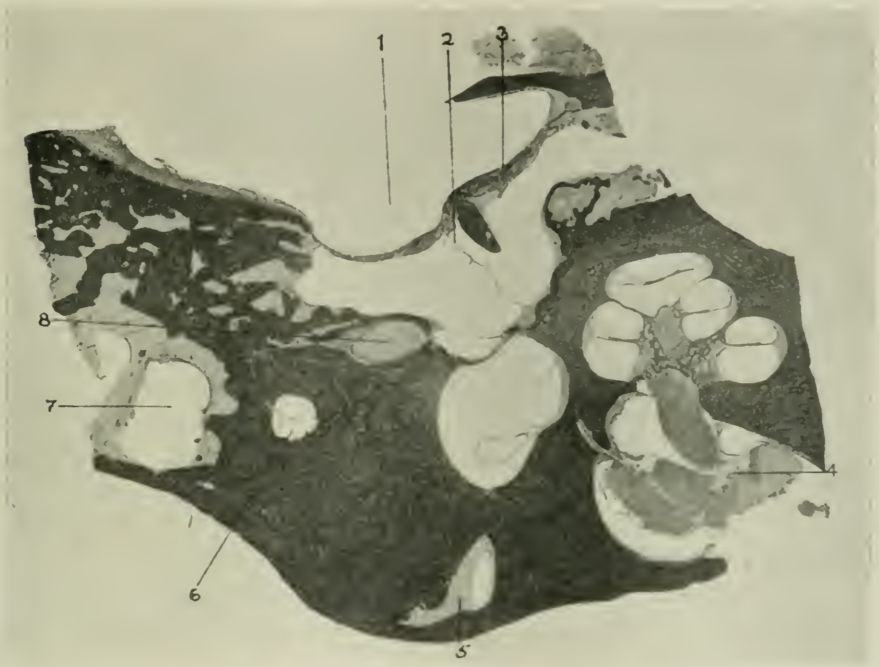

FIG. 1 (Case I).

Epidemic cerebro-spinal meningitis of about eight days' duration. Horizontal section through right ear, No. 135, × 6 diam. 1, external auditory meatus; 2, adhesion between drumhead and inner wall; 3, new bone formation in drumhead (chalk patch); 4, internal meatus, showing slight meningeal exudate; 5, crus commune (note faulty formation of walls of bony canal; 6, convexity of lateral canal; 7, antrum, with thickened lining membrane; 8, facial nerve. Note that long process of incus is absent; it should be situated between the handle of the malleus and the footplate of the stapes which is seen in the oval window. Middle-ear suppuration had evidently been present in childhood in this case.

in the floor of the aditus and in the antrum, and a band of mucous membrane (adhesion) stretching across from the anterior margin of the oval window to the tympanic membrane. The malleus is normal. The long process of the incus is eroded in its upper part and lower down it is absent. The crura of the stapes are entirely absent but the footplate is healthy. The mucous membrane in the niche of the oval window is thickened. There are adhesions between the pyramid and the anterior margin of the window. The mucosa in the niche of the round window is rather thickened. (There had evidently been a former chronic suppurative otitis media in this case which had left behind these bony deposits, submucosal thickenings, adhesions and erosions of bone.) The labyrinth capsule and surrounding bony tissue are healthy. *Cochlea* : Corti's organ is not well formed : the pillar cells can only be recognized in the middle coil. Reissner's membrane is in parts rather depressed. There is some new connective tissue formation in the canal for the spiral ganglion. The opening of the aqueduct of the cochlea is quite healthy. *Vestibule* : The utricle is healthy. The wall of the sacculus is ruptured (artefact ?) but the neuro-epithelium is healthy. The aqueduct of the vestibule is quite normal. There is no infiltration within the labyrinth of either the cochlear or vestibular nerves by the meningitis. *Canals* : The bony walls of the semicircular canals are badly formed in this case—a fairly common occurrence. The lateral, superior and posterior canals are healthy. *Internal meatus* : There is a little pus within the arachnoid sheath but there is no infiltration of the cochlear or vestibular nerves by meningitis.

(*Note.*—Except for the slight meningitis in the internal meatus the only noteworthy feature is the atrophy (?) of Corti's organ. This may have been a secondary result of the chronic middle-ear suppuration and subsequent sclerosed condition of the tympanum. On the other hand it may merely be an artefact or due to post-mortem changes.)

Case II.—Corporal F., died from epidemic cerebro-spinal meningitis after several weeks' illness. Post-mortem, May 12, 1916 (J. K. M. D.) : Body much emaciated. Brain—convolutions flattened. No exudate on cerebrum. Pituitary congested. On cutting across the crura cerebri a large quantity of cerebro-spinal fluid gushed out. Aqueduct dilated. Much thick exudate over pons. Frontal lobes adherent to each other. Lateral ventricles much dilated. Ependyma of whole ventricular system thickened and œdematous. Posterior wall of third ventricle very thin and pineal veins enlarged. Fourth ventricle enormously dilated, especially up into arbor vitæ. Lateral recesses also large. Foramen of Magendie closed by adhesions and plug of thick pus. No hæmorrhages on section of brain. Right middle ear opened up and found normal. Sphenoid and ethmoid sinuses also normal.

Microscopic Examination of Left Ear.—All parts of the middle ear cleft are entirely normal. *Cochlea*: Reissner's membrane is at the usual angle, showing apparently normal endo- and perilymphatic tension of the labyrinth. (In cases of meningitis it is often collapsed on to the top of Corti's organ.) Corti's organ is fairly well preserved. One can see the usual outline and the pillar cells, but most of the hair cells and supporting cells are desquamated. The cells of the spiral ganglion appear shrunken (artefact?). There is no pus in the scala tympani in the neighbourhood of the round window, but there is a little coagulated lymph present in the opening of the aqueduct of the

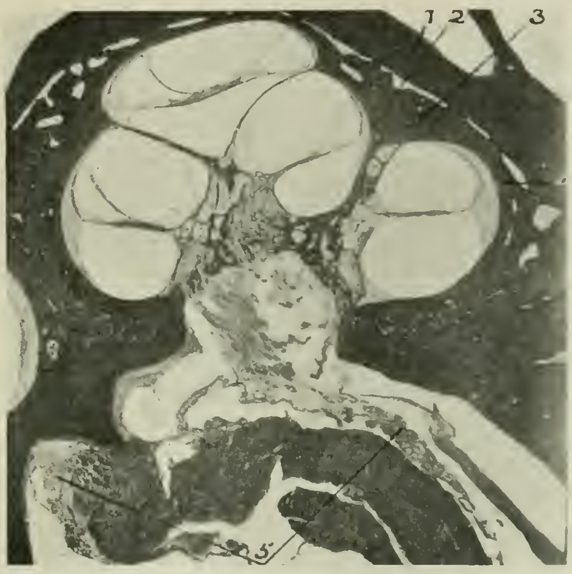

Fig. 2 (Case II).

Epidemic cerebro-spinal meningitis of several weeks' duration. Horizontal section through left ear, No. 130, × 9 diam. 1, periosteal bone; 2, lymph space between above and 3, cartilage bone; 4, endosteal bone; 5, meningitic infiltration within arachnoid sheath in internal meatus. Corti's organ is seen to be well formed in the apical coil, but in the basal coils it is desquamating. There is slight exudation in the basal coil on the right of the illustration (scala tympani) in the angle next the internal meatus.

cochlea. There is also some exudate in the scala tympani of the basal coil in the angle next the internal meatus (fig. 2). *Vestibule*: The utricle, saccule and ductus endolymphaticus are quite normal. The canals are also normal. *Internal meatus*: There are numerous small cells round the nerve filaments in the fundus of the internal meatus (meningitis) (figs. 2 and 3). The vestibular ganglion cells appear rather shrunken. There is a little hæmorrhage in the internal meatus just at the inner opening. (This specimen shows no disease of middle or inner ear, but there is some meningitis in the internal auditory meatus.)

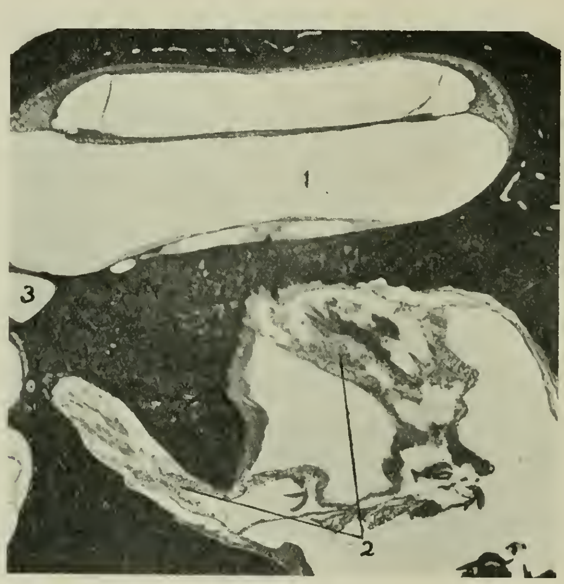

FIG. 3 (Case II).

Section 250, × 9 diam. 1, scala tympani normal; 2, lower part of internal meatus showing meningitic infiltration within arachnoid sheath; 3, niche of round window.

Case III.—Corporal D., admitted to hospital on August 7, 1916, suffering from epidemic cerebro-spinal meningitis. When examined in bed patient was found to be lying on the right side in a semi-comatose condition. Kernig and Babinski signs present. Meningococci found in cerebro-spinal fluid before death. Patient had *marked horizontal and rotatory nystagmus to the left* but was too ill for detailed functional examination of the ear. He was, however, able to answer questions when first one ear and then the other was closed with the finger.

Death occurred two days after admission. *Post-mortem* (J. K. M. D.): Pus in subarachnoid space all over cerebrum. No pus above cribriform plate. Pituitary body congested. Adhesions and thin fluid pus in interpeduncular space and cisterna pontis. No pus on upper surface of cerebellum. There was a mass of purulent exudate round the fifth nerve on the right side, and also round the trunks of the sixth, seventh, eighth, ninth, tenth, eleventh and twelfth nerves. There were localized collections of pus on the inferior surface of both cerebellar hemispheres and also in the cisterna magna. The foramen of Magendie was closed by recent adhesions. The fourth ventricle contained thick pus. On section of the brain both cerebral hemispheres showed numerous petechial hæmorrhages in the white matter, especially marked just beneath the grey matter of the sulci. Patches of hæmorrhage also present in the internal capsule but almost completely absent in the frontal and occipital poles of the brain. Pus was present in the

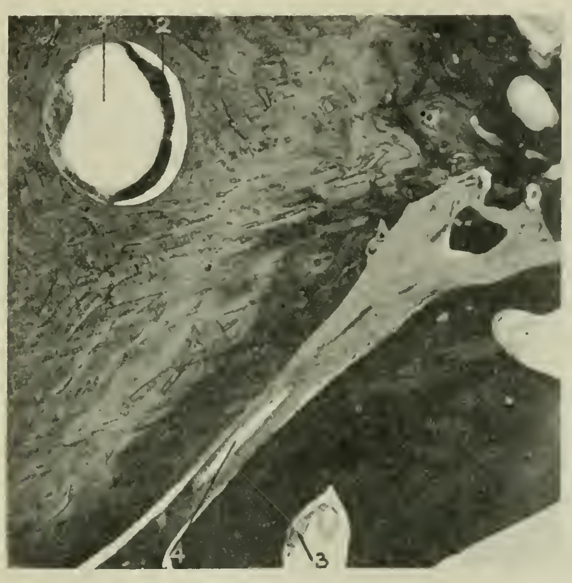

FIG. 4 (Case III).

Epidemic cerebro-spinal meningitis of a few days' duration. Spontaneous nystagmus to the left was present. Horizontal section through right ear, No. 10, × 9 diam. 1, endolymphatic space of ampullary end of superior canal healthy; 2, perilymphatic space containing hæmorrhage and purulent exudate; 3, smooth end of superior canal showing exudate in perilymph space; 4, vessel in fossa subarcuata.

posterior and inferior horns of the lateral ventricles and in the pineal recess. The sphenoid, ethmoid and frontal sinuses were opened up but were found normal. The left (middle and inner) ear was opened up and was also found normal.

Microscopic Examination of Right Middle and Inner Ear.—The tympanic membrane and Eustachian tube are healthy. *Tympanic cavity* : The middle-ear spaces are healthy, except in the neighbourhood of the oval and round windows. The mastoid cells are normal. The mucosa of the sinus tympani is thickened, the vessels are engorged, and there is pus, probably mixed with blood, in the niche (fig. 9). The lowest part of the tympanic cavity is perfectly healthy (fig. 10). The facial nerve and canal are normal. There are a few pus cells and some fibrin in the niche of the oval window but the footplate of the stapes is normal (fig. 7). The anterior portion of the annular ligament appears to be slightly infiltrated with pus cells as if there was an

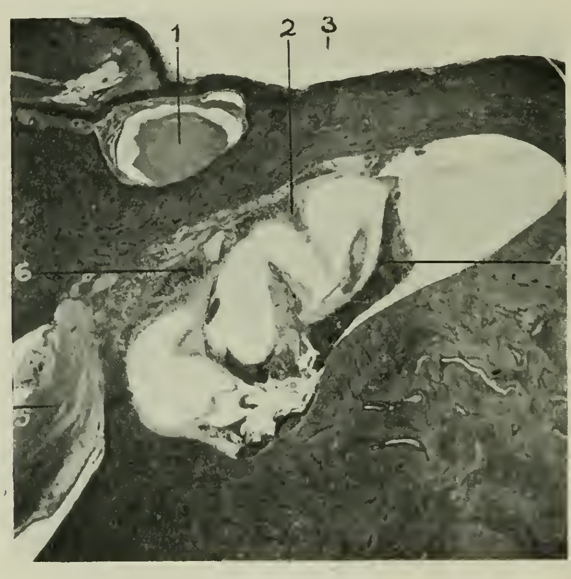

Fig. 5 (Case III).

Section 50, × 9 diam. 1, facial nerve ; 2, crista of lateral canal infiltrated with pus (the exudate is streaming through the apex of the crista, and, if the illustration is turned right round, gives the appearance of a volcano in eruption) ; 3, aditus ; 4, purulent and hæmorrhagic exudate in perilymph space ; 5, the vestibular nerve surrounded by purulent exudate ; 6, purulent and hæmorrhagic exudate at point of entry of nerve to utricle.

attempt to burst through from the labyrinth into the middle ear. The round window membrane is thickened and infiltrated and there is a layer of pus on the walls of the niche (fig. 8). The only parts of the middle ear which show any abnormality are the niches of the oval and round windows. There has apparently been no spread of the infection from these regions into the remainder of the tympanum. The labyrinth capsule and surrounding bony tissue are healthy. *Cochlea*: In the hollow spaces of the cochlea there is pus present in all coils, especially in the scala tympani and the scala vestibuli (fig. 6). In the cochlear canal itself the exudate is of a fibrinous nature. Corti's organ and the stria vascularis are desquamating. The spiral ligament is infiltrated with pus cells. In the apical coil Corti's organ is fairly well preserved, and the membrana tectoria is present. In the middle coil there is a great deal of pus in the scala vestibuli. In the basal coil Corti's organ and the membrana tectoria can be clearly recognized. Reissner's membrane is depressed in all coils. The helicotrema contains pus. The vein accompanying the aqueduct of the cochlea is very engorged.

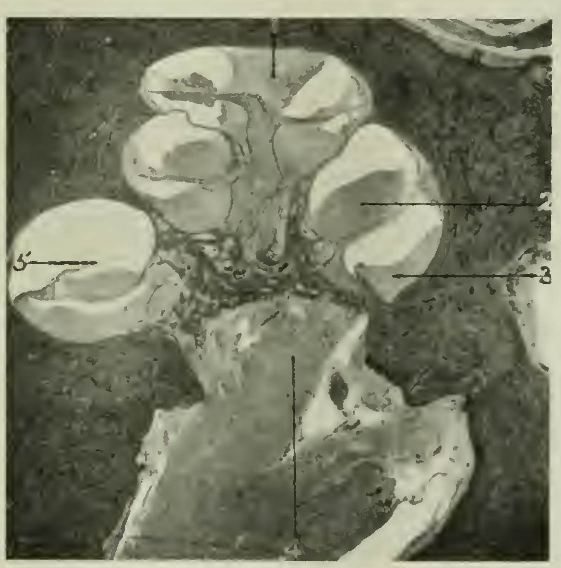

FIG. 6 (Case III).

Section 70, × 9 diam. 1, purulent exudate in helicotrema; 2, exudate in scala vestibuli; 3, same in scala tympani; 4, cochlear nerve with hæmorrhagic purulent exudate around; (5) endosteum detached from wall of scala tympani of basal coil (artefact).

There are some pus cells in the scala tympani just at the opening of the perilymphatic aqueduct (fig. 8). There are a good many pus cells in the hollow space of the bony spiral lamina. (The infection appears to have passed from the internal meatus along the vessels and nerves to the labyrinth, rather than by the aqueduct of the cochlea.) *Vestibule:* There is some hæmorrhage present in the perilymphatic space of the vestibule around the vestibular nerve (fig. 5). The nerve of the utricle and also the neuro-epithelium of the utricle are infiltrated with pus.

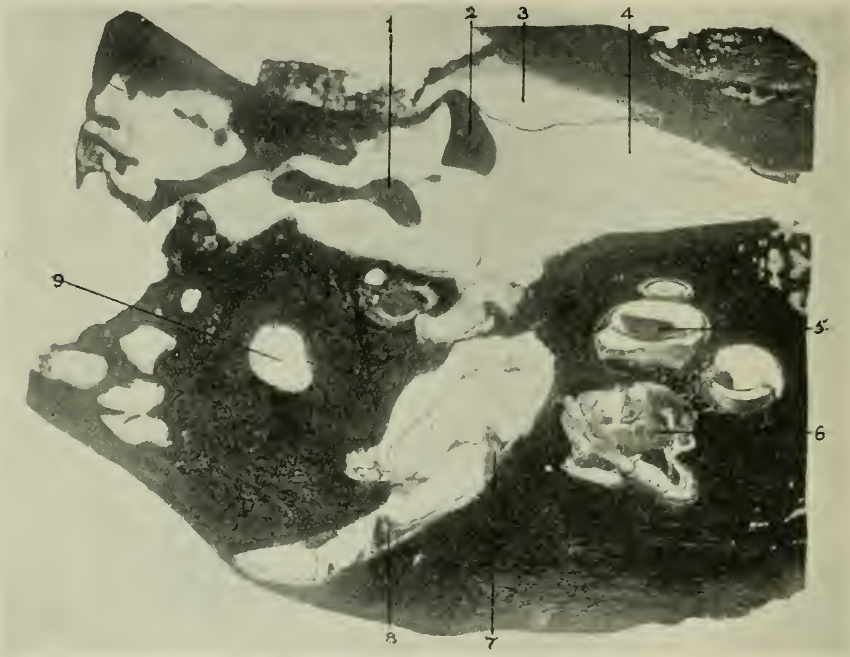

Fig. 7 (Case III).

Section 104, × 6 diam. 1, incus; 2, malleus; 3, external meatus; 4, tympanic cavity, healthy; 5, exudate in scala vestibuli of middle coil; 6, lower part of internal meatus with hæmorrhagic purulent exudate; 7, exudate in inner part of vestibule; 8, exudate in crus commune; 9, lateral canal almost free from exudate.

(On the whole the endolymphatic spaces, in the vestibule, canals and cochlea are almost free, so that the infiltration has been mainly in the perilymphatic space.) The nerve and the epithelium of the saccule are infiltrated with pus and there is pus in the perilymph space at the opening of the aqueduct of the vestibule (fig. 7). The saccule is ruptured (artefact ?). There is a little curdled lymph and possibly a few pus cells in the endolymphatic aqueduct at its vestibular end, but towards its cranial end the aqueduct is healthy. *Canals*: At the ampullary end of the superior canal the perilymph space is full of pus (fig. 4); the endolymph space is normal. The crista of the superior canal is infiltrated with pus. In the smooth end of the superior canal the perilymph space contains pus. There is also pus in the perilymph space of the external canal (fig. 5). The crista of the lateral canal is infiltrated with pus. The cupola of the lateral canal is disintegrated and *pus cells are seen passing through the apex of the crista into the endolymphatic space*. The appearance is somewhat like that of a volcano in eruption (fig. 5). The smooth end of the lateral and posterior canals contains pus in the perilymph space but none in the endolymph space (fig. 7). In the convexities of the superior and external canals the same conditions obtain. The crista of the posterior canal is infiltrated with pus. The perilymphatic space of the posterior canal is crammed with pus (fig. 10); the endolymphatic space contains

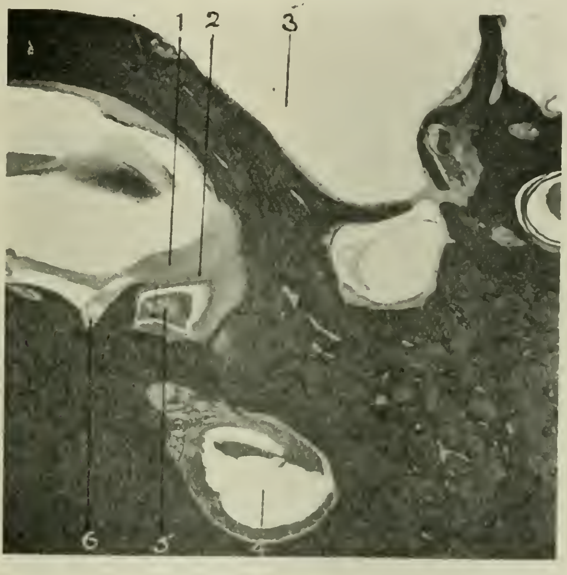

FIG. 8 (Case III).

Section 175, × 9 diam. 1, pus in scala tympani; 2, thickened membrane of round window; 3, tympanic cavity, healthy; 4, ampullary end of posterior canal with exudate in perilymph space; 5, purulent exudate in niche of round window; 6, exudate at cochlear opening of perilymphatic aqueduct.

curdled lymph and at the ampullary end there is a little pus. This purulent infiltration appears to be coming from the region where the nerve enters. *Internal meatus:* There is much pus within the arachnoid sheath (figs. 6 and 7). The vestibular ganglion is infiltrated with pus cells, but the exudate does not extend along the facial nerve as far as the geniculate ganglion. There is a considerable amount of purulent infiltration of the nerves passing into the modiolus. There is a great deal of pus in the lower part of the internal meatus. The purulent exudate extends into the narrow canal containing the nerve of the ampulla of the posterior canal.

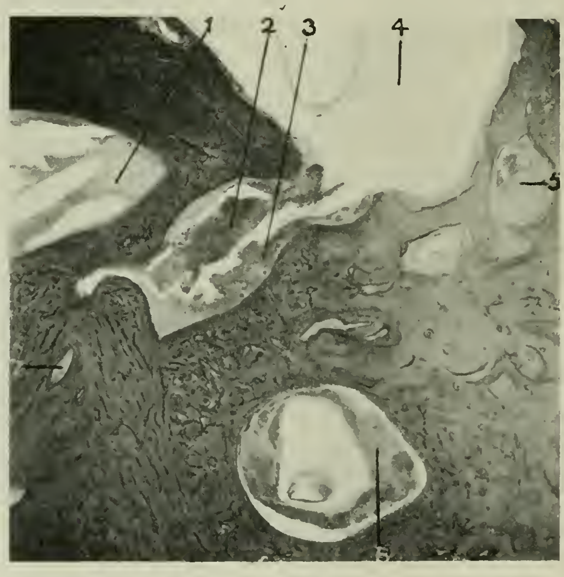

FIG. 9 (Case III).

Section 205, × 9 diam. 1, exudate in scala tympani of basal coil; 2, purulent exudate in sinus tympani; 3, thickened, infiltrated and engorged mucosa lining the sinus tympani; 4, tympanic cavity; 5, stapedius; 6, hæmorrhagic purulent exudate in perilymph space of posterior canal; 7, aqueduct of cochlea containing exudate.

Remarks.—In this case the basal meningitis had distinctly extended into the internal meatus on the right side, and had infiltrated into the labyrinth along the vessels and branches of the eighth nerve. The perilymphatic space throughout the labyrinth contained much pus, while in parts—especially in the region of the cristæ of the canals and the saccule—the neuro-epithelium was infiltrated with pus which was finding its way into the endolymphatic space. The congested and infiltrated condition of the mucosa in the window niches was certainly secondary to the purulent labyrinthitis, though at the time of death there had been no gross bursting through of the pus in the labyrinth into the middle ear. It is noteworthy that on *macroscopic* examination the left, middle and inner ear of this patient appeared to be normal.

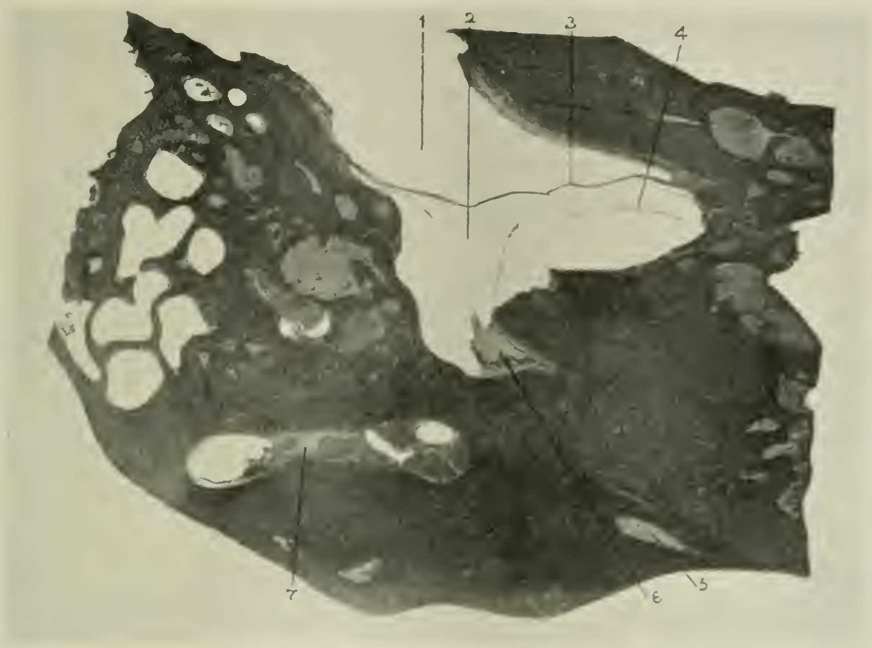

Fig. 10 (Case III).

Section 235, × 6 diam. 1, external meatus; 2, tympanic cavity; 3, membrana tympani; 4, normal mucosa detached owing to use of suction apparatus; 5, aqueduct of cochlea containing exudate; 6, sinus tympani also containing exudate; 7, posterior vertical canal with much exudate in perilymph space.

(B) Mumps Deafness.

Clinical Aspect.—Boot records two cases of mumps deafness. (1) Male, aged 21, on third day of disease had marked noises in the right ear, deafness and giddiness. There was normal hearing in the left ear, Weber lateralized to good ear. (2) Girl, aged 12, had complete deafness in the left ear on first day, with giddiness and vomiting. Boot has collected forty-nine cases of mumps deafness (twenty-two male and twenty-seven female). The age of the patients varied from 4 to 41 years, usually between 11 and 30. The middle ear was normal in twenty-seven cases and only abnormal in three; rest not noted. The onset of deafness varied from the fourth to the fifteenth day, usually occurring seven days after the beginning of the disease. The deafness came on suddenly in twenty-two cases, slowly in one; rest not noted. Giddiness was present in twenty-nine cases, absent in seven; no note in thirteen. Headache was present in six cases, absent in four; rest not noted. Subjective noises in twenty-five. Nausea and vomiting in nineteen. Fever in three. In ten cases the labyrinth disease was bilateral and in thirty-two unilateral; no note in seven cases. Boot distinguishes three types of the disease: (1) The cochlea alone is affected. (2) The vestibular apparatus is involved with normal or almost normal hearing. (3) Both parts of the labyrinth are affected.

Voss, on the basis of lumbar puncture, compares mumps deafness to that following syphilis, and attributes both to meningitis. Ruttin, in discussing Voss's paper, stated that there was probably more than one cause of mumps deafness. The inner ear affection might be due to serous or purulent labyrinthitis, or to retrolabyrinthine disease. The endolabyrinthine variety was exactly like a serous or purulent labyrinthitis — i.e., there was sudden deafness, marked vestibular symptoms, with nystagmus to the sound side, and loss of vestibular reaction. The nystagmus lasted only three or four days. On the other hand the retrolabyrinthine disease due to mumps was similar to the syphilitic affection. Here the disease was associated with prolonged vestibular symptoms, and there might be loss of the rotation reaction, with retained caloric reaction. Further, the cochlear and vestibular apparatus might not both be destroyed. Scheibe stated that he had seen a great deal of mumps deafness and had always found it to be unilateral. This fact spoke against a neuritis or meningitis. On the other hand, the absence of giddiness and noises were against labyrinthitis. Kümmel thought that mumps deafness was usually bilateral at first, but one ear recovered, so that later deafness was only found on the other side. Kirchner thought that the absence of labyrinth symptoms was in favour of a cerebral cause of the deafness.

Hubbard states that mumps is the cause of the nerve deafness in 3 to 5 per cent. of deaf-mutes. Deafness may occur before the parotid swelling. Gordon states that as a rule the symptoms of meningitis immediately precede the orchitis. Hubbard notes that in his own cases the destruction of both cochlear and vestibular functions might be complete or partial. He apparently regards the lesion as a labyrinthitis.

According to Feiling, May states that in cases of mumps deafness tinnitus and vertigo usually occur as premonitory symptoms. Deafness follows more or less suddenly, usually between the first and fourth days of the disease. In most cases it is complete and permanent. Ménière (junr.) considered that the lesion was probably a meningitis at the base. The fact that the seventh nerve is sometimes implicated supports Ménière's view.

Lake records two cases of deafness following mumps. The first was that of a woman, aged 35, who suddenly became deaf in the left ear at the end of the first week, with severe tinnitus, vertigo and sickness. The drumheads were normal. Functional examination showed marked nerve deafness. The second case was that of a girl, aged 17, in whom the right ear was affected. Lake thinks that the trouble was due to embolism of a large branch of the internal auditory artery. Mauthner has recorded the case of a female, aged 37. On the first day of the attack she felt slight noises in the left ear, which rapidly increased. In three days complete deafness. Two days after the onset, marked giddiness and nausea. Diplopia also present on eighth day from onset of giddiness. The stumbling gait improved in three weeks but the deafness and noises remained. The vestibular reaction, tested eight months after onset, was diminished. Mauthner thought that the deafness was very like that associated with acute infectious neuritis. Haike reported the case of a male, aged 19, who became suddenly ill with high fever, orchitis and unilateral deafness. There was an island of hearing between C_2 and C_3. There was no parotitis. The vestibular nerve was intact and the diagnosis was made of retrolabyrinthine neuritis. He regarded the case as one of mumps, without parotitis, and recalled a similar case of Gradenigo's.

Among the 111 cases of deaf-mutism examined by one of the writers (J. S. F.) only one case of mumps deafness was found. In this case the drumheads were indrawn but otherwise normal: the lower tuning-forks appeared to be heard (? vibrations felt); the vestibular reaction to the rotation test was present but somewhat reduced.

Pathology.—Monod was the first to demonstrate lymphocytosis of the cerebro-spinal fluid in mumps, even in cases where there was no clinical evidence of meningitis. This lymphocytosis is a constant feature and may be so marked as to constitute a true lymphocytic suppuration. In one fatal case there was definite meningitis at the base of the brain. Numerous other similar cases have been recorded. De Stella, in an article dealing with serous meningitis, states that one of his cases followed mumps and was associated with transient deafness in a boy, aged 12. The diagnosis of meningitis was formed by lumbar puncture. De Stella thinks that mumps deafness is caused by meningitis. Gordon has inoculated monkeys intracranially with filtered saliva from mumps cases. Of the ten monkeys so inoculated, four died, six recovered. Those that died first of all became quiet and appeared to be suffering from headache. Then irritability appeared, but there was no paralysis, squinting, or retraction of the head. There was, however, some rigidity of the arms and legs. Lumbar puncture was done on one and the fluid found to be under increased tension and turbid. *Post-mortem:* There was congestion of the meninges but no pus. Microscopically there was infiltration of the pia arachnoid with lymphocytes. Some nerve cells in both the cerebral cortex and spinal cord showed degenerative changes. Gordon holds that in a proportion of cases of mumps a virus occurs in the saliva that passes through a Berkefeld filter and is capable of producing in the monkey by intracerebral injection a lymphocytic meningitis, together with hyperæmia of the central nervous system and acute degenerative changes—i.e., that mumps is due to an ultramicroscopic virus comparable to that in poliomyelitis, small-pox, typhus, and a number of other acute fevers.

Russell L. Haden reports on the bacteriology of a mumps epidemic at Camp Lee. From the blood, the parotid secretion and the testis the organism recovered was quite uniformly a Gram-positive diplococcus which grew slowly. In Haden's nine cases the spinal fluid showed uniformly a pleocytosis of the mononuclear type. Clinically all nine patients had classical mumps. Cultures and smears were negative in eight cases. In one instance the fluid was opalescent and direct smears showed numerous Gram-positive diplococci. Haden holds that it is reasonable to conclude that the organism demonstrated in the spinal fluid was the one causing the primary parotitis. Blood cultures were taken from all cases of mumps on admission. Twenty-five cultures were taken from eighteen cases. Nineteen of the twenty-five were sterile; two were contaminated; four cultures on different patients showed in each instance a small Gram-positive diplococcus. After several transplants this grew readily on all media.

According to Tasker Howard the metastatic lesions in mumps are well known, and testify to the general nature of the infection. Orchitis, mastitis, pancreatitis, arthritis, encephalitis and meningitis are at times met with. Orchitis is occasionally recognized in the absence of any involvement of the salivary glands. Howard regarded his three cases as instances of mumps meningo-encephalitis, in spite of the absence of inflammation of the salivary glands. These cases occurred in the presence of a mumps epidemic, and in two of the three a Gram-positive diplococcus was recovered from the spinal fluid. This organism was found in direct smears and grown in pure culture in both cases. The spinal fluid in each case presented a moderate pleocytosis, characterized by a predominance of mononuclear cells. The conditions with which we are familiar which show this picture are: (*a*) syphilis, (*b*) tuberculous meningitis, (*c*) encephalitis lethargica, and (*d*) mumps. Two of the three patients were certainly not syphilitic. Tuberculous meningitis and encephalitis lethargica could be ruled out in all cases by the clinical course. So far as we have been able to find out there has hitherto been no recorded case of mumps deafness in which a microscopic examination of the ear has been carried out.

(C) INFLUENZAL DEAFNESS.

Haug considers the nerve deafness of influenza to be due to localized meningitis.

(D) TUBERCULOUS MENINGITIS.

Gradenigo has examined three cases of tuberculous and two of epidemic meningitis. He comes to the following conclusion: In the various forms of meningitis there are frequently inflammatory changes in the seventh and eighth nerves, owing to the passage of the infection carriers into the internal meatus. At first the changes are not so much in the nerve itself as in its covering. Later the pus passes between the nerve bundles and then enters the modiolus. Gradenigo always found the aqueduct of the cochlea intact. In most cases there were inflammatory changes in the middle ear, at least in one side.

(E) OSTEOMYELITIC DEAFNESS.

Castex has recorded the case of a male deaf-mute, aged 17, who spoke well till the age of 7. At this time he got osteomyelitis of the right tibia, with sequestrum formation, and was in bed for three years. During recovery deafness occurred without ear suppuration. A month later he was very silent, and his voice took on the tone characteristic of deaf-mutism. Siebenmann has found only four cases of osteomyelitic deafness in the records, and has observed three further cases himself. He states that osteomyelitis is a separate disease due to staphylococcal infection. It usually attacks young people, and begins with high fever and delirium. The ear is seldom diseased in the stage of high fever, more frequently during convalescence or a year later. The deafness comes on suddenly within a few hours or days. Both ears are affected as a rule. Subjective noises and giddiness are present in half the cases. In the others the deafness occurs without any irritative phenomena on the part of the cochlea or vestibule. As a rule the deafness is bilateral, but on one side there may be islands of hearing. Where the hearing is lost the static labyrinth no longer reacts. Siebenmann compares osteomyelitic deafness to bilateral metastatic panophthalmitis. Steinbrügge has examined a case microscopically and found changes in the labyrinth similar to those present in meningitis complicated by labyrinthitis.

(F) SYPHILITIC DEAFNESS.

Harrison states that syphilitic leptomeningitis is generally diffuse, and affects most often the base. Some degree of meningitis is extremely common in secondary syphilis, if we may judge by the very high percentage of cases in which pathological changes—increase of lymphocytes, presence of globulin, and a positive Wassermann reaction—are found in the cerebro-spinal fluid. Dreyfus reported pathological changes in nearly 80 per cent. of secondary cases. Mills has obtained a number of specimens of cerebro-spinal fluid at Rochester Row, and examination of these has shown pathological changes, indicating syphilitic meningitis in six out of thirty-six primary cases, eighty-seven out of 185 secondary cases, twenty-one out of sixty-five tertiary cases, and six out of fourteen congenital cases. None of these cases showed any other sign of brain or cord disease. In the great majority of cases, therefore, syphilitic meningitis is silent as regards clinical symptoms, if headache is excepted. Even headache is not very common.

Pathologically, the meninges at the base are extensively infiltrated and matted together with an exudate of lymphocytes and plasma cells. The process is most intense at the inter-peduncular spaces. The arteries at the base of the brain become involved. One or more of the cranial nerves passing through the diseased area are also frequently involved. Symptoms of a damaged cranial nerve very strongly suggest syphilitic meningitis. The eighth nerve may be affected in its bony canal, and one or both branches may be attacked.

Beck finds that *in 80 per cent. of syphilitic patients the bone-conduction for tuning-forks is more or less shortened although the hearing is normal.* Beck attributes this to raised intracranial pressure, as he finds the same condition in cases of brain tumour and hydrocephalus. Beck performed lumbar puncture on some of his syphilitic cases and found that after this the shortening of the bone-conduction disappeared for a time, though it returned in two or three days. Cases in which the primary sore is still present and the Wassermann reaction is negative do not show this shortened bone conduction. The latter only appears with the constitutional symptoms. The sign may be of value in the diagnosis of syphilis.

Frey states that cases of neuro-labyrinthitis in secondary syphilis are much more common than is usually supposed, and claims that the work of Moriac in 1879 proves this. Habermann, in 1896, reported sixty-six cases of disease of the acoustic nerve, thirty-four of which occurred in the secondary stage. Mayer has collected sixty-five cases occurring since 1896, and of these thirteen belonged to the secondary stage. Labyrinth symptoms—i.e, tinnitus, deafness, giddiness, nausea, vomiting, disturbance of balancing, and vestibular nystagmus—have occurred as early as the seventh day after the primary sore (Politzer, quoted by Pogany, *Monatsschrift für Ohrenheilkunde*, 1913, p. 1333). Mayer states that the trouble may come on three weeks after the primary sore. At the time of onset of the first skin rash neuro-labyrinthitis is by no means uncommon. Stuempke holds that the membranous labyrinth is involved by a syphilitic infiltration.

Beck has examined 600 cases of secondary syphilis and finds that complaints of giddiness and disturbance of balancing are by no means rare. In some of these nothing abnormal is to be found in the ears or nervous system. In others there are changes in the posterior cranial fossæ, as evidenced by nystagmus of central origin and cerebellar disturbance of balancing. The third group consists of vestibular cases which show alteration in the galvanic irritability of the vestibular nerve. At the onset tinnitus is present in most cases, while giddiness is noted in 50 per cent. These symptoms may be observed before the skin rash appears. In the later stages of secondary syphilis the ear trouble almost always comes on with a recurrence of the rash (Mayer). Disturbance of hearing is gradual and may vary in intensity. It is usually bilateral. Functional examination shows an affection of the eighth nerve

Vestibular symptoms vary from slight giddiness up to vomiting and disturbance of balancing.

During the war observations on the connexion between syphilis and nerve deafness were made in Dr. Logan Turner's Department by W. T. Garretson and Miss Mabel Fitzgerald. These observations have not yet been published, but they revealed a remarkably high percentage of positive Wassermann reactions in patients who showed nerve deafness at an unusually early age—i.e., below 55 or 60. It is to be hoped that Dr. Garretson and Miss Fitzgerald will before long publish their results in detail.

(G) DEAFNESS FOLLOWING MEASLES AND PNEUMONIA.

Nager records the case of a boy, aged 6, who had slight Eustachian catarrh at three years. Adenoids were removed. The child took measles while recovering from this operation and there was a sudden onset of meningitis, with sopor, restlessness, meningitic cry, high temperature and stiffness of the neck. After fourteen days, consciousness returned. During convalescence there was swelling of the elbow-joint, and the parents noticed that the boy did not respond to calls. Siebenmann diagnosed complete deafness. On account of the still present tubal closure the child was taken into the clinic, paracentesis carried out and transudate removed. After air douches the drumhead on both sides was normal. The deafness, however, remained complete. The vestibular apparatus was not tested. Three years later the child was killed by accident. At the post-mortem the brain and temporal bones appeared normal, but the seventh and eighth nerves were rather thin. The middle ear on both sides showed the same condition—recent acute catarrhal otitis media with exudate. The mucous membrane of the tympanic cavity was much thickened and infiltrated. The ossicles and joints, the stapes and margins of the window were all normal. In the left inner ear the canals were unchanged. The vestibule, however, showed increase of fibrous tissue in the perilymph space with strands joining the utricle and stapes. There was a layer of connective tissue with some new bone formation in the lower part of the vestibule filling the perilymph space. The utricle and saccule were dilated and the vestibular nerve rather atrophied. In the cochlea the opening of the perilymphatic duct was almost closed by connective tissue and new bone. In the basal coil the scala vestibuli showed new connective tissue and bone formation. The cochlear duct was dilated and Corti's organ was only an irregular group of cells. The cochlear ganglion cells were reduced in number and the connective tissue around them increased. The right labyrinth showed greater changes in the cochlea and less in the vestibule. Nager states that there was no point of breaking in from the middle ear to the labyrinth, so that he attributes the condition of the labyrinth on both sides to meningitis. Nager calls attention to the similarity of the appearances to those seen in certain cases of congenital deafness, especially to the "Siebenmann" type attributed to fœtal meningitis.

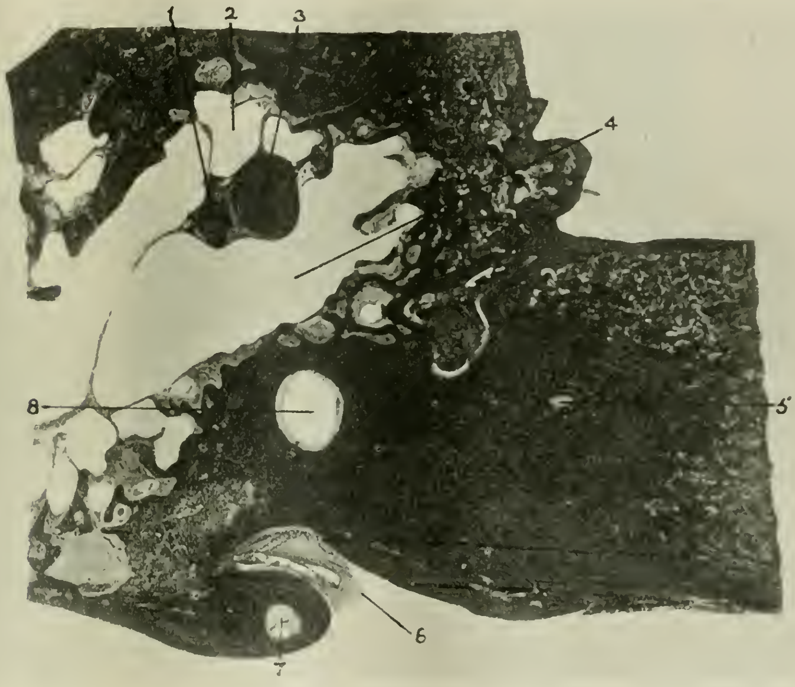

FIG. 11.

E. B., aged 17 months. Case of pneumonia following measles, complicated by meningitis and labyrinthitis. Horizontal section No. 80, × 6 diam. 1, incus; 2, outer attic, healthy; 3, malleus; 4, inner attic, healthy; 5, basal coil of cochlea just appearing; 6, wide fossa subarcuata; 7, smooth end of superior canal with exudate in perilymph space; 8, ampullary end of canal.

Siebenmann has recorded six cases of deaf-mutism following measles. In three the trouble was undoubtedly of middle ear origin; in the remaining three the route of labyrinth infection was an open question. In 50 to 60 per cent. of cases of deaf-mutism following measles the tympanic membrane is normal. This would point to a meningitic origin. Meningitic symptoms in measles are not very rare and are usually attributed to the high fever. The meningitis of measles is relatively favourable as regards life and yet may lead to disease of the labyrinth. The meningitis and labyrinthitis are probably due to the pneumococcus.

Meningitic Neuro-labyrinthitis following Measles and Pneumonia.—(For the clinical notes of this case the writers are indebted to the late Major Melville Dunlop, and for the temporal bone to Dr. Carnegie Dickson.) E. B., male, aged 17 months, admitted to Royal Hospital for Sick Children April 23, 1914, with shortness of breath of fourteen days' duration. The child had had measles four months before admission, followed by broncho-pneumonia. Examination on admission showed rapid breathing, enlarged glands in neck, axillæ and groin. Pulse 162. Temperature 101·8° F. Examination of lungs showed areas of dullness with tubular breathing. Child rather drowsy. Von Pirquet negative.

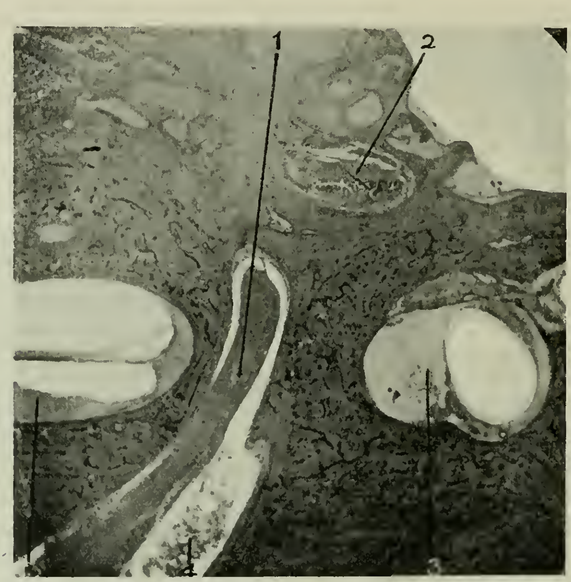

Fig. 12.

E. B., pneumonia and meningitic labyrinthitis. Section 115, × 9 diam. 1, facial nerve ; 2, facial nerve on inner wall of attic ; 3, exudate in perilymphatic space of vestibule just below ampullary end of superior canal ; 4, purulent exudate around facial nerve ; 5, exudate in scala tympani of basal coil of cochlea.

Later there was evidence of consolidation at left base, but exploration was negative. The evening temperature, however, remained elevated, 101° F. to 103° F. A fortnight after admission twitching of the whole of the right side developed. The left pleura was again explored but no fluid obtained. Lumbar puncture evacuated turbid fluid under greatly increased tension. The child became comatose and died on May 10, 1914. *Post-mortem:* Rachitic chest with marked beading. The right lung showed marked congestion and œdema, with emphysema. The left lung was much collapsed and also showed marked bronchiectasis.

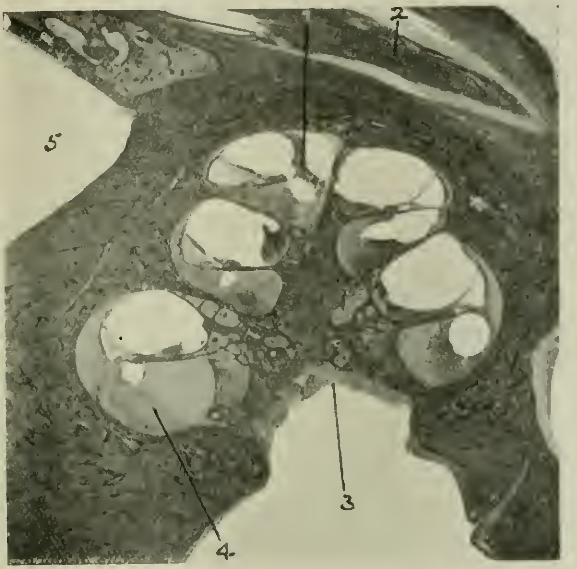

FIG. 13.

E. B., pneumonia and meningitic labyrinthitis. Section 185, × 9 diam. 1, purulent exudate in helicotrema; 2, tensor tympani; 3, pus in fundus of internal meatus; 4, purulent exudate in basal coil of cochlea; 5, carotid canal.

Examination of the head revealed copious pale greenish purulent exudate in subarachnoid space. There were toxic changes in the internal organs. Bacteriological examination of the *fluid from the bronchi and meninges gave a pure growth of pneumococcus in both cases.*

Microscopic Examination of the Right Ear.—The tympanic membrane and Eustachian tube are normal (figs. 15 and 18). *Tympanic cavity:* There is slight swelling of the mucosa of the attic and a little exudate in the mastoid cells (figs. 11 and 15). The lateral canal prominence is normal. There is slight swelling of the mucosa and a little muco-purulent exudate in the sinus tympani (fig. 18). The lower part of the tympanic cavity is normal. There is slight sero-purulent exudate in the niche of the oval window (fig. 15). The footplate of the stapes is normal. The mucosa of the round window niche is slightly swollen (fig. 17). The ossicles, muscles and joints are normal. The labyrinth capsule and surrounding bony tissue are healthy. *Cochlea :* There is much pus in the scala tympani in all coils (fig. 13). This extends up to the helicotrema, but in the scala vestibuli the purulent exudate is not so marked. There is some curdled lymph and a few pus cells in the scala media (fig. 14). Corti's organ is well preserved; one can see pillar cells in all coils and even the hair cells. Reissner's membrane is slightly depressed. The cochlear aqueduct contains pus throughout (fig. 17). *Vestibule :* There is much purulent infiltration in the perilymph space of the vestibule (fig. 15). The saccule is ruptured and pus is seen infiltrating the nerve to the saccule.

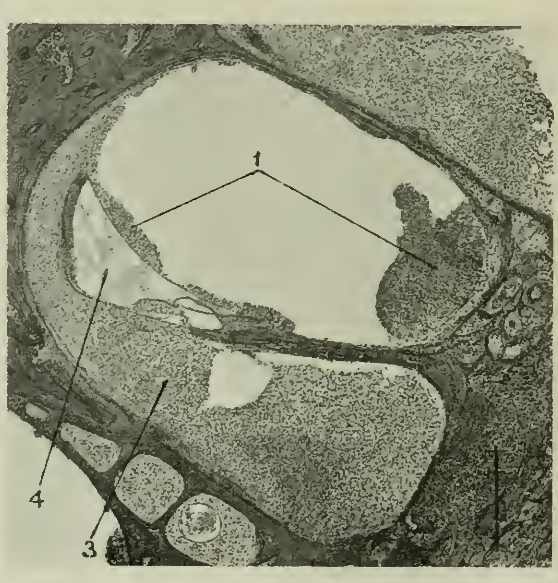

FIG. 14.

E. B., pneumonia and meningitic labyrinthitis. Section 185, × 38 diam. 1, purulent exudate in scala vestibuli—to the left it is lying on upper surface of Reissner's membrane; 2, spiral ganglion infiltrated with pus; 3, Scala tympani full of pus; 4, cochlear canal with fibrinous and slightly purulent exudate.

The ductus endolymphaticus is normal. (As one would expect from the route of infection, there is more pus in the cochlea than in the vestibule.) *Canals :* The superior canal shows cellular exudate in the perilymph space (fig. 11). The crista of the superior canal itself is healthy. There is very little pus in the convexity of the posterior canal, but a considerable amount in the perilymphatic space at the ampullary end (fig. 18). The epithelium of the crista and the cupula are normal.

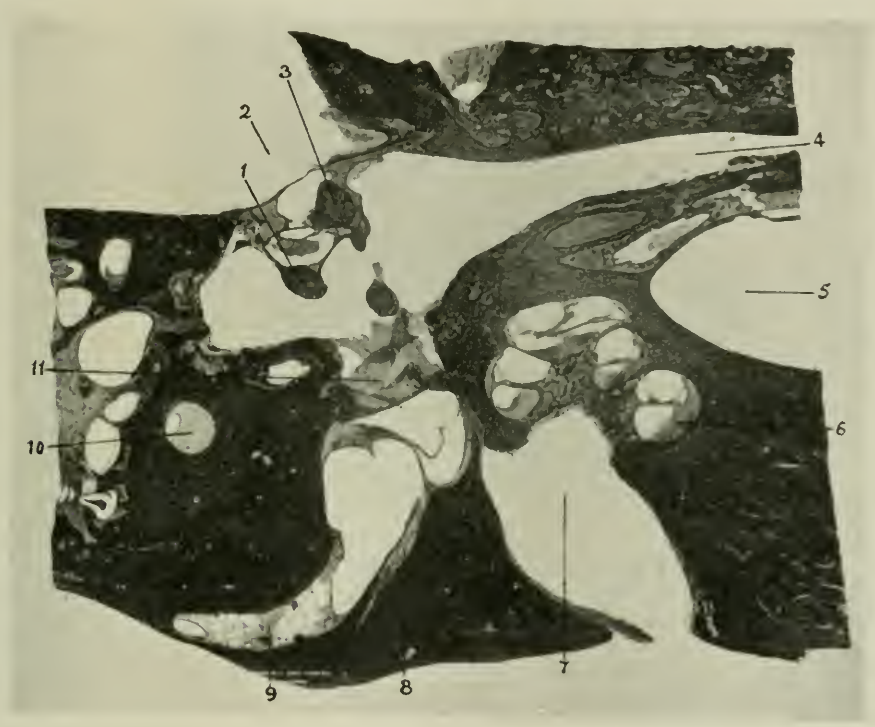

FIG. 15.

E. B., pneumonia and meningitic labyrinthitis. Section 205, × 6 diam. 1, incus ; 2, external meatus : 3, malleus ; 4, Eustachian tube, normal ; 5, carotid canal ; 6, exudate in scala tympani of basal coil ; 7, internal meatus (the nerves have unfortunately been torn out) ; 8, endolymphatic aqueduct ; 9, exudate in perilymph space of crus commune : 10, lateral canal ; 11, niche of oval window showing thickened mucosa with some exudate.

In the smooth end there is practically no pus. The lateral canal shows purulent infiltration in the perilymphatic space (figs. 15 and 16). There is very little pus in the convexity of the lateral canal. The smooth end shows great vascular congestion. *Internal meatus :* Both branches of the eighth nerve have unfortunately been torn out at the post-mortem. There is meningitis in the upper part of the internal meatus extending along the seventh nerve almost as far as the geniculate ganglion (fig. 12). Although the nerves have been torn out the meatal fundus at the base of the modiolus is crowded with pus (fig. 13).

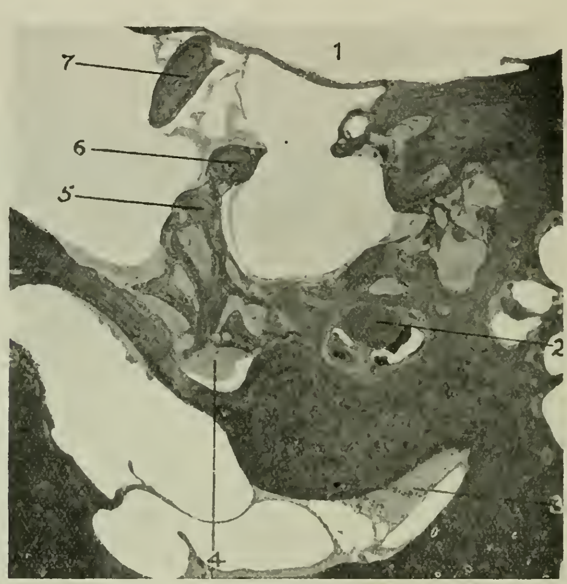

Fig. 16.

E. B., pneumonia and meningitic labyrinthitis. Section 245, × 9 diam. 1, external meatus; 2, facial nerve; 3, exudate in perilymph space of smooth end of lateral canal where it joins the vestibule; 4, exudate in niche of oval window; 5, head of stapes; 6, long process of incus; 7, malleus.

CONCLUSIONS.

(1) Meningitic neuro-labyrinthitis is a frequent cause of deafness and deaf-mutism.

(2) Deafness due to epidemic cerebro-spinal meningitis is certainly due to meningitic neuro-labyrinthitis. Measles and pneumonia may also be followed by meningitis, and secondary neuro-labyrinthitis. In acquired syphilis and mumps leptomeningitis is of common occurrence and is associated with inner ear deafness, which is probably to be explained by neuritis or neuro-labyrinthitis (no microscopic examination as yet). Certain cases of deafness after influenza and osteomyelitis may also be of meningitic origin.

(3) The original source of infection may be in the respiratory tract, parotid gland, genital organs, long bones, or elsewhere.

(4) A blood infection (septicæmia) probably in all cases forms the connecting link between the primary disease and the onset of meningitis.

(5) Meningitic neuro-labyrinthitis is usually but by no means always bilateral. The onset is usually sudden. Irritative symptoms, such as tinnitus and giddiness, are often present but may not be observed owing to the mental condition (coma) of the patient. In epidemic cerebro-spinal meningitis and parotitis, deafness usually occurs early in the course of the disease.

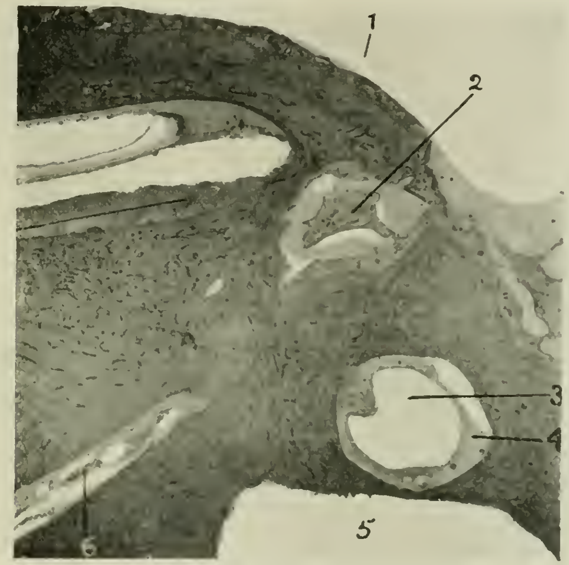

FIG. 17.

E. B., pneumonia and meningitic labyrinthitis. Section 275, × 9 diam. 1, tympanic cavity with slightly swollen mucosa on promontory; 2, seropurulent exudate in niche of round window; 3, endolymphatic space of ampulla of posterior canal, normal; 4, exudate in perilymph space; 5, jugular bulb; 6, exudate in perilymphatic aqueduct.

(6) Deafness due to meningitic neuro-labyrinthitis may be associated with other metastatic lesions, e.g., orchitis, arthritis, mastitis, blindness or paralysis of the oculo-motor nerves.

(7) The infection usually passes along the subarachnoid space from the base into the internal auditory meatus and then along the nerves

and vessels to the labyrinth. In some cases the perilymphatic aqueduct is the route of invasion, while in others both paths may be involved.

(8) As a rule both the cochlear and vestibular apparatus are affected. Frequently the cochlear apparatus is the one mainly or alone involved; rarely do we have a more or less isolated affection of the vestibular apparatus.

(9) The pathological changes producing the deafness may be: (*a*) hydrocephalus; (*b*) changes in the walls of the fourth ventricle; (*c*) purulent infiltration of the eighth nerve with subsequent descending neuritis accompanied by atrophy of the spiral ganglion cells and Corti's organ; (*d*) purulent labyrinthitis which, if the patient lives long enough, is followed by the formation of granulation tissue and, later,

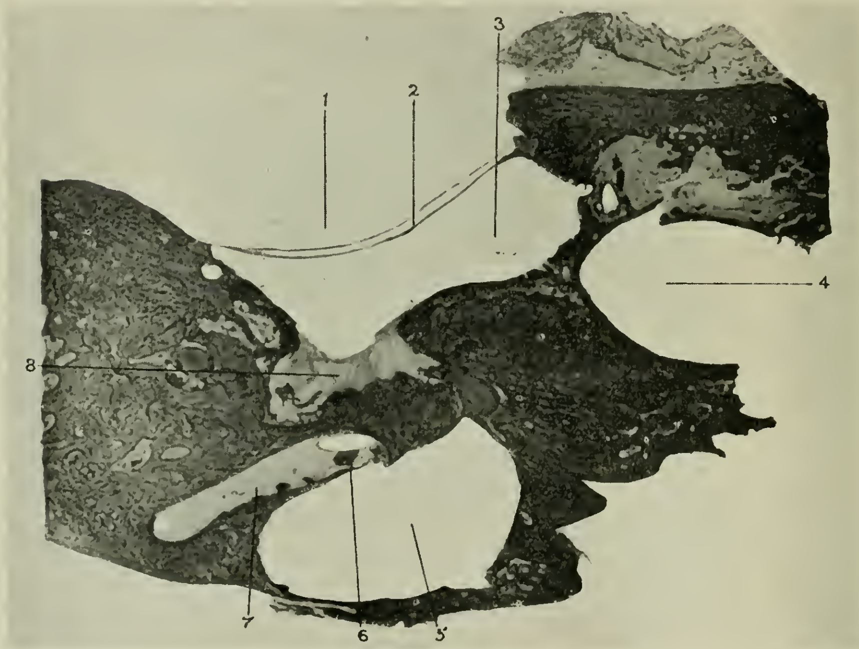

Fig. 18.

E. B., pneumonia and meningitic labyrinthitis. Section 295, × 6 diam. 1, external meatus; 2, tympanic membrane (the epidermic layer is peeling off); 3, lower part of tympanic cavity, healthy; 4, carotid canal; 5, jugular bulb; 6, thin bony wall separating ampullary end of posterior canal from jugular bulb; 7, perilymyh space of posterior canal with exudate; 8, exudate in sinus tympani.

of new connective tissue and bone in the hollow spaces of the labyrinth.

(10) The resulting deafness is as a rule complete and permanent in the ear (or ears) affected.

(11) Vestibular symptoms (loss of balancing and waddling gait) pass off rapidly in adults, but in young children they may last as long as one year.

(12) In cases of sudden nerve deafness with or without vestibular symptoms lumbar puncture should be performed and the cerebro-spinal fluid examined chemically and microscopically. The Wassermann reaction of the fluid should also be tested and cultures made.

(13) Repeated lumbar punctures are of use in treatment, especially in cases of deafness due to hydrocephalus. Small doses of potassium iodide and hypodermic injections of pilocarpin have been used in the treatment of meningitic neuro-labyrinthitis, but apparently without success.

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REFERENCES.

Alt. *Monatsschr. f. Ohrenheilk.*, 1908, xlii, p. 2; "Die Taubheit infolge von Meningitis cerebrospinalis epidemica," Leipz. u. Wien, Fr. Deuticke, 1908.

Altmann. *Klin. Jahrb.*, 1906, xv, p. 627.

Baginsky, B. *Arch. f. Kinderheilk.*, 1900, xxviii, p. 24.

Beck. *Münch. med. Wochenschr.*, 1913, lx, p. 2778.

Boot. *Journ. Amer. Med. Assoc.*, 1908, li, p. 1961.

Castex. *Centralbl. f. Ohrenheilk.*, 1904, ii, p. 154.

Davis, H. J. *Proc. Roy. Soc. Med.*, 1913, vi (Sect. Otol.), p. 68.

Dufays. *Rev. de Laryngol.*, 1910, xxx (2), p. 273.

Feiling. *Quart. Journ. Med.*, 1915, viii, p. 255.

Foster and Gaskell. "Cerebro-spinal Fever," Cambridge, 1916.

Frey. *Centralbl. f. Ohrenheilk.*, 1911, ix, p. 259.

Goeppert. *Klin. Jahrb.*, 1906, xv, p. 523.

Goerke. *Verh. Deutsch. Otol. Ges.*, 1906, xv, p. 227.

Gordon. Report of the Local Government Board, 1914.

Gradenigo. *Ann. de maladie de l'Oreille*, 1889, xv, p. 528.

Gray, A. A. "Diseases of the Ear," 1910.

Habermann. *Zeitschr. f. Heilk.*, 1886, vii, p. 27; *ibid.*, 1892, xiii, p. 324; "Die luetische Erkrank. des Gehörorgans," 1896.

Haden. *Amer. Journ. Med. Sci.*, 1919, clviii, p. 698.

Haike. Report of the Berlin Otologische Gesellschaft, November 4, 1910.

Harrison. "The Diagnosis and Treatment of Venereal Diseases in General Practice," Oxford Medical Publications, 1918.

Hartmann. *Archives of Otol.*, 1880, ix, p. 11.

HELLER. *Deutsch. Arch. f. klin. Med.*, 1867, iii, p. 482.
HESSLER. *Arch. f. Ohrenheilk.*, 1907, lxxiii, Teil 1, p. 194.
HILDESHEIM. *Lancet*, 1905, i, p. 1332.
HOWARD. *Amer. Journ. Med. Sci.*, 1919, clviii, p. 685.
HUBBARD. *Trans. Amer. Otol. Soc.*, 1915, xiii, pt. 3.
KER. "Infectious Diseases," 2nd. ed. (advance proof).
KOERNER. "Lehrbuch d. Ohren-Nasen- u. f. Kehlkopf-Krankheiten," 5te Aufl., J. F. Bergmann, Wiesb., 1914, p. 550.
LAKE. *Journ. of Laryng.*, 1905, xx, p. 153.
LARSEN. *Nord. Mediciniskt Arkiv*, xxii.
LESZYNSKY. *Med. Record*, 1906, lxix, p. 325.
LOVE, KERR. *Arch. of Otol.*, 1893, xxii, p. 170; *Glasg. Med. Journ.*, 1893, xxxix, p. 442.
LUCAE. *Arch. f. Ohrenheilk.*, 1870, v, p. 188.
MAUTHNER. *Wien. med. Wochenschr.*, 1911, lxi, p. 2089.
MAYER. *Arch. f. Ohrenheilk.*, 1908, lxxvii, p. 188.
MOOS. *Zeitschr. f. Ohrenheilk.*, xii.
MYGIND. *Centralbl. f. Ohrenheilk.*, 1913, p. 75.
Idem. "Deaf-mutism," Rebman, Lond., 1894.
NAGER. *Verh. Deutsch. Otol. Ges.*, 1906, xv, p. 163; *Zeitschr. f. Ohrenheilk.*, 1907, liv, p. 217.
NETTER and DEBRÉ. "La Méningite Cérébro-Spinale," Paris, 1911.
POLITZER. "Diseases of the Ear," 1902.
ROLLESTON. *Lancet*, 1919, i, pp. 541-653.
ROOSA. *Arch. of Otol.*, 1884, xiii, p. 65.
SCHEIBE. *Zeitschr. f. Ohrenheilk.*, 1895, xxvii, p. 100.
SCHULTZE. *Virchow's Archiv*, 1890, cxix, 1.
SCHWABACH. *Zeitschr. f. Ohrenheilk.*, xxiv; *Zeitschr. f. klin. Med.*, 1891, xviii, p. 273.
SIEBENMANN. *Zeitschr. f. Ohrenheilk.*, 1907, liv, p. 1; *Rev. hebd. de Laryngol.*, 1907, xxvii, (2), p. 39.
STEINBRÜGGE. *Zeitschr. f. Ohrenheilk.*, 1892, xxii, p. 192.
DE STELLA. *Arch. Internat. de Laryngol.*, 1909, xxviii, p. 14.
STUEMPKE. *Centralbl. f. Ohrenheilk.*, 1909, vii, p. 450.
THEODORE. *Verh. Deutsch. Otol. Ges.*, 1913, xxii, p. 375.
VOSS. *Centralbl. f. Ohrenheilk.*, 1913, xi, p. 541 (Ruttin, Scheibe, Kümmel and Kirchner discussed Voss's paper).
WORSTER-DROUGHT and MILLS KENNEDY. "Cerebrospinal Fever," Cambridge, 1919.
YEARSLEY, MACLEOD. *Journ. of Laryng.*, 1917, xxxii, p. 117.

DISCUSSION.

Mr. J. S. FRASER: I wish to suggest that probably these cases of meningitic labyrinthitis are very much more common than they are at present thought to be. I fancy they must be a very frequent cause of deaf-mutism. A child, before the age at which it learns to talk, suffers from meningitis—it may be connected with pneumonia or some zymotic disease—and infiltration occurs along the eighth nerve: it passes to the cochlea, destroys the inner ear, and, consequently, the child never talks. Such a child may be considered as an example of congenital deafness, whereas it is really a case of acquired deafness due to labyrinth suppuration occurring before the age of speech development.

Dr. A. A. GRAY: Mr. Fraser has shown us some extremely beautiful slides. I think his explanation as to the occurrence of deaf-mutism in meningitis is very probably correct; because, after epidemic cerebro-spinal meningitis, deafness is much more common than is blindness. If the deafness were due to the effect of the changes in the meninges, why should there be that difference? Is not the optic nerve as liable to be damaged by the activity of the micro-organism, or by the inflammatory change, as the seventh and eighth nerves? I think, therefore, Mr. Fraser's suggestion is more probably correct than the ordinary view that it was due to the effect on the auditory nerve in its course through the cranium.

Mr. H. J. BANKS-DAVIS: Does Mr. Fraser think any child could recover from a suppurative labyrinthitis and live?

Dr. URBAN PRITCHARD: I was going to ask the same question as Dr. Banks-Davis has put. If you stop short of suppuration, I think his explanation is correct. If there is suppuration there, I do not see how the patient can live.

Mr. C. E. WEST: We constantly see cases become sterile and cease to suppurate in all localities. In the mastoid we see old collections of fluid which have been purulent and are now sterile, all that is left being the residue of the former purulent process. That is suppuration in a closed space, similar to that of the labyrinth. My idea is that these cases are, in the histological sense, suppurative labyrinthitis, and there is a complete resolution of the process, but, of course, with the destruction of all the elements in the structure of the internal ear. I would like to raise one small point as a side issue. I was much interested in Mr. Fraser's specimens—and I would like to thank him personally very much for them—and in connexion with them he spoke of a chronic adhesive process in the middle ear. I am an agnostic about this process: I doubt that it exists. I think that what Mr. Fraser showed us are the residua of the fœtal pad which occupies the middle ear. I think some may be post-suppurative processes, but many are mere residua.

Dr. DAN MCKENZIE: We feel very much indebted to Mr. Fraser for having added, by this contribution, to the laurels he has already gained in this section of our work: he is proceeding to put our knowledge on labyrinth diseases upon a sound pathological basis. It is remarkable that we have had to wait so long for that to be done. However, it is being done now. Some of the pictures shown to-day are really most beautiful, especially the enlarged one exhibiting suppuration of the cochlear canals: it is a wonderful piece of expert histological work. One or two points which Mr. Fraser raises in his paper are of practical interest. In one of the pictures he showed that infiltration of pus has taken place along the facial canal to some extent, I presume from within. In going through work dealing with facial paralysis, as I have been doing recently, the question has occurred to me—which I shall be glad to have settled—whether meningitis is ever induced by middle-ear suppuration extending along the facial canal and meninges only, or is it not the case that those formerly reported as due to filtration along the facial canal are also cases in which there was labyrinth suppuration from without? This, I think, is important, because it may be one of the routes by which meningitis may be induced from middle-ear disease. In most of the old records of meningitis attributed to this condition I have been struck by one or two facts: either that the condition of the labyrinth was not mentioned at all, or, if it was mentioned, the statement was made that there was pus in the labyrinth. From this one would suppose that in many of the earlier cases, at all events, it is impossible to say whether the meningitis was actually induced by infiltration along the facial canal alone. If Mr. Fraser can enlighten me as to this I shall be glad. In his description of the first case Mr. Fraser agrees that mumps deafness is probably secondary to meningitis also. I think, however, it is the case that in mumps deafness there is often unilateral deafness, whereas, presumably, the meningitic would be bilateral. Another point concerns the vestibular reaction in mumps deafness. In most cases it has been present. If there were meningitis affecting the cochlear branch of the eighth nerve, it would also probably affect the vestibular branch, and there would be absence of the reaction from the vestibular side.

The question of vestibular reaction and deaf-mutism has been considerably gone into abroad, and I understand Mr. Fraser is going through that work himself.

Dr. P. WATSON-WILLIAMS: We cannot see Mr. Fraser's specimens and hear his reports without learning something: certainly I have learned a great deal. We are very grateful to him. One of the points which impressed me was the suggestion that deaf-mutism might be attributed to a suppuration involving the labyrinth in the way he has described. And the question arises as to how far these children might recover, even if the condition was suppurative. Now, "suppuration" is a relative term: even when there are proportionately few polymorphs it is still, pathologically, suppurative, though macroscopically there might not be much turbidity, and if it is right to assume that a slightly suppurative meningitis may recover, one ought to remember that, and not regard suppurative meningitis as necessarily past recovery, in which case treatment is not likely to be energetically pursued. I think such cases do recover, even without the aid of the more recent therapeutic methods, but a greater number would probably recover if we were not so disheartened by the prospect before us. I remember a case in which I was agreeably surprised. Before I had time to investigate the case fully, he developed symptoms of suppurative meningitis. I did a labyrinthectomy, opening up the external semicircular canal and the cochlea. He had a well-marked suppurative meningitis. I had given up hope, but repeated lumbar puncture and the use of colloidal silver, intravenously and intrathecally, produced a very happy recovery. If we do not look upon these cases as hopeless, but carry on active therapeutic measures, and all the resources at our disposal, more children will recover, even if it be as deaf-mutes.

Mr. STUART-LOW: I have been much struck by the number of such cases as Mr. Fraser mentions in the large clinics at the Central London Throat, Nose and Ear Hospital, where I worked with Dr. Grant for years, and we have always paid special attention to them. I always made a point of taking the cases into the hospital, and tried to do my best for them, never despairing of doing some good. I have often performed a cortical mastoid operation, sometimes having been pushed to do so by the urgent request to "do something." This in some instances has had the effect of giving relief by draining the ear and setting up a better circulation, even if it did not directly affect the labyrinth. I also often gave repeated small doses of mercury, and have had the cases brought to me afterwards with a large degree of deafness, but this has sometimes improved on the small doses of mercury given for a long period. I would go a step further than Mr. Fraser and ask the route by which the organisms obtain access to the labyrinth. We know the pathological products are often pushed in, so to speak, from the meninges, but does not the infection originally get in through the Eustachian tube and the middle ear? I am a strong believer in the occurrence of fugitive, evanescent otitis media which occurs sometimes without symptoms, and I think it is then that the invasion of the labyrinth takes place.

Mr. SYDNEY SCOTT: I feel much interested in Mr. Fraser's communication, especially in reference to the question whether the infection in the labyrinth originated in the meninges or in the middle ear. In the sections from the children's ears, Mr. Fraser demonstrates the infection in progress in the fossula rotunda. I think it would be difficult to accept the view that the infection spread to the middle ear from the meninges without clinical evidence, which was perhaps wanting in these young infants. It is possible that in these cases the infection in the middle ear was the circumscribed and unresolved part of a widespread otitis media, which preceded the meningitis.

Mr. FRASER (in reply): In answer to Mr. Banks-Davis, I do not see why a child with suppurative labyrinthitis should not live. I agree with what Mr. West said about the disease or infection dying out. If an adult can recover from a purulent infection of the pia arachnoid, why should not a patient be able to recover from a purulent inflammation of the labyrinth? Surely infection in the subarachnoid space is in a much more vital place than when it is in the labyrinth. Many deaf-mutes have survived both purulent meningitis and purulent labyrinthitis of meningitic origin. Remarks have been made about what I called a "chronic adhesive process." What I meant was, that those were post-suppurative conditions. Perhaps "cicatrized tympanum" would be a better term. I believe the first of the patients had had a middle-ear suppuration, and that this had passed off and left these "chalk patches" in the tympanic membrane and adhesions in the tympanic cavity. With Dr.

McKenzie, I do not think that infection from the middle ear passes along the facial canal and causes meningitis. I should say that when the labyrinth becomes infected from the subarachnoid space the internal meatus is the most common route, secondly the aqueduct of the cochlea. I think infection of the meninges from simple mastoiditis can occur via the vessels of the fossa subarcuata from the inner wall of the mastoid antrum. The veins and probably also lymphatics pass beneath the dome of the superior canal and through the fossa subarcuata. I have seen cases of mastoiditis and meningitis in which there was no labyrinthitis and no extradural abscess, and apparently no other route of infection. There was tremendous engorgement of these veins in the fossa subarcuata. Dr. McKenzie said he could hardly understand how a unilateral suppuration of the cochlea could be of meningitic origin, but I think we know that even after epidemic cerebro-spinal meningitis, where the infection certainly comes from the meninges, the deafness is not always bilateral, though usually it is. Hitherto there has been, so far as I know, no microscopical examination of the inner ear in a case of mumps deafness. Dr. Watson-Williams spoke of operation in cases of meningitis. In Dr. Turner's clinic in Edinburgh we operate on practically all our cases of purulent meningitis, except on those in which the patients are moribund—e.g., where a brain abscess has already burst into the lateral ventricle. Dr. Turner has recorded about seven cases of cure of purulent meningitis after operation. I have had one such case myself. With regard to the route of infection, Mr. Sydney Scott thought my last case might have had fugitive otitis media, and that the infection had lingered in the region of the round window, and passed into the cochlea. I do not agree with him. I have already recorded in the *Journal of Laryngology* (June, 1914) a case of acute suppurative otitis media with labyrinthitis and meningitis with rupture of the tympanic membrane. In that case the infection was obviously coming from the tympanic cavity: one could see the pus cells making their way into the vestibule round the margin of the stapes and into the cochlea, through the round window. The appearances were very different from those in the last case I showed. I think a parallel to the present cases is to be found in swelling of the cheek in association with an inflamed tooth. With labyrinth suppuration of meningitic origin there is a little congestion and swell in the tympanum at the place where the labyrinth comes closest to the middle ear—namely, the round and oval windows. I think that is all there was present in these cases. Some German and Austrian observers have even recorded cases in which there has been a bursting outwards of the pus from the labyrinth through the oval window into the tympanic cavity. In this way middle-ear suppuration may arise as a result of labyrinthitis o meningitic origin.

Sarcoma of Middle Ear arising in Eustachian Tube.

By RICHARD LAKE, F.R.C.S.

W. T. W., aged 33, came to consult me on November 4, 1919. He complained of deafness in the left ear in 1918. In November of that year he had pain in the head and left ear, at which time the patient was living in British Columbia. In May, 1919, he saw a specialist in Vancouver who removed a post-nasal growth and the tonsils, as well as an aural polypus; the nasal as well as the aural growth recurred in six weeks. Further operation on November 15, 1919. Growth visible, projecting from external meatus. Diagnosis: New growth of middle ear or of Eustachian tube.

Operation, November 6, 1919, assisted by Dr. Zamora: The auricle was reflected by my flap and a dark mass was seen obviously coming from the middle ear. The mass was removed, and a radical mastoid was completed as far as the removal of bone was concerned. The middle ear, antrum and attic, were found full of a dark mass of new growth which was removed by curettage. The dura was exposed by bony absorption over a large area and the growth itself appeared to come from the Eustachian tube. In the nasopharynx, which one cannot examine without an anæsthetic, a diffused ill-defined mass was felt involving and extending from the Eustachian tube. Healing of the cutaneous wound was immediate.

In view of the pathologist's report (attached) radium emanations seemed indicated, and Captain Hayward Pinch prescribed, and the Radium Institute most kindly loaned, a tube which was buried in the middle ear for twelve hours, on November 20, 1919.

Some facial paralysis came on in September, 1919. One severe attack of pain on Christmas day, 1919. Health is improving.

Report on Tissue removed from Ear (Mr. P. P. Laidlaw).—Four pieces of tissue were submitted; all were cut. A portion of meatus: This showed nothing of particular interest. Of the three pieces of tissue from the middle ear, the smallest, an almost black-coloured mass, showed merely necrotic tissue with a few leucocytes and many bacteria. The other two pieces were red vascular masses. On section they showed newly developed tissue partially covered with stratified epithelium, and divided irregularly by fibrous tissue septa. In the deeper parts spicules of dead bone were observed, and in one section a tiny nodule of cartilage. In the meshes of the fibrous tissue, besides blood-vessels, there were large collections of irregular shaped cells, which however tended to be spindle shaped. The nuclei of these cells were seen to be irregular in size, chromatin content, and number. A few cells were worthy of the term giant cells. Mitoses were seen but they were not numerous. These abnormal cells abut directly on the endothelium of the blood-vessels, and there is very little fibrous tissue apart from the larger septa referred to above. The whole shows that there can be little doubt that there is a new growth of the nature of a spindle-celled sarcoma involving the middle ear. A few of the cells show pigment, but as this gives the test for iron in most cases it is probably blood pigment.

DISCUSSION.

The PRESIDENT: I should like to ask whether that patient had a serous discharge for a long time previously to the complaint of deafness in 1918. I have seen eleven cases of endothelioma of the nasopharynx. In three the patients had a serous discharge from one ear, and slight deafness, and the discharge was the first symptom for which they had been treated for many weeks, of course without good effect. The ultimate diagnosis rested on the discovery of the primary lesion, invading the pharyngeal orifice of the Eustachian tube. Any patient who has a long-continued discharge of serum from the ear which does not respond quickly to the ordinary methods, should make us suspect that the cause may be a new growth in the nasopharynx. Sometimes there may be very little swelling in that neighbourhood in the early stages of the disease. I think Sir William Milligan has laid stress on the same point. Mr. Lake's case seems to be a success, so far.

Mr. R. LAKE (in reply): This man had no previous discharge. The dose of radium bromide was 58 mgr. screened through 1 mm. of silver.

Transplantation of Anterior Half of Masseter Muscle for Facial Paralysis.

By SOMERVILLE HASTINGS, F.R.C.S.

J. M., A WOMAN, aged 35, was shown before this Section on November 17, 1916. She had had a mastoid operation performed eight years previously, and has suffered from very severe giddiness for three years. She was almost completely deaf on the left, and gave a well-marked fistula symptom with nystagmus to the right, and falling to the left. The exhibitor asked the opinion of the meeting as to the treatment of the case. Labyrinthectomy was advised, and an anterior labyrinthectomy was carried out on March 7, 1917. The labyrinth was opened on each side of the facial nerve canal, and the result, as far as the giddiness is concerned, has been admirable in every way. Unfortunately, three weeks after the operation, left facial paralysis began to develop, with some necrosis of bone and discharge from the ear, which continued for several months. The reaction of the degeneration slowly developed in the facial nerve, and was reported complete in October, 1919. On November 26, 1919, her condition was that shown in the photograph, and an operation was performed. A crescentic portion of skin was removed, the left masseter split, the anterior half detached from the jaw, and the lower end of the divided portion stitched with catgut to the tissues of the angle of the mouth. It will be seen that the deformity is reduced and a certain amount of movement of the left angle of the mouth is now possible.

DISCUSSION.

The PRESIDENT: Has anyone present seen a case of facial paralysis treated by using the temporal muscle, instead of the masseter, thus avoiding a scar in the cheek? I believe it has been done.

Dr. KELSON: I congratulate Mr. Somerville Hastings on the ingenious method exhibited in this case, but I would ask him whether he thinks, with that extent of scar, the game is worth the candle, especially in a woman.

Mr. SOMERVILLE HASTINGS (in reply): I think this woman has been really improved by the operation. She tells me she has now much less difficulty with her food. Part of the scarring is due to the removal of a crescentic piece of skin. At the time I did the operation I did not know that temporal muscle had been used for this purpose. But even if that muscle is used, it is necessary to bring it over the zygoma, and there must apparently result an unsightly swelling on the face. I would like to know whether members have had experience in the use of the temporal muscle in cases of facial paralysis.

Case of Stenosis of the Meatus following Otitis Externa.

By E. D. D. DAVIS, F.R.C.S.

THE Rev. G. P., who has resided in India for many years, has suffered from otitis externa and dermatitis for ten years. On examination there was considerable stenosis at the junction of the bony and cartilaginous meatus. It was impossible to obtain a view of the drum, but at a later date it was seen that there was some discharge from the middle ear. After preliminary treatment a conservative mastoid operation was performed on October 23, 1919: the antrum was normal, and at the same time the cartilaginous meatus was excised with the exception of a small strip of the anterior wall. A large tube was kept in the meatus and antral cavity for ten days. No skin-grafting was done.

The result is satisfactory, but one attack of dermatitis has occurred since. The tests show an improvement in hearing.

DISCUSSION.

Dr. P. Watson-Williams: I was interested in seeing this gentleman, because some years ago, before he went to India, I saw him once or twice, and he then had such well marked dermatitis, apparently of septic origin, that the meatus was occluded. I think I treated him with hydrarg. ammoniata, but very shortly after I saw him he passed from my care, and I understand he was in the hands of someone in India, and, after being seen a good many times, a foul plug of wool was found. I do not think that plug was present when I saw him. After the removal of the plug he still continued to have trouble, and our colleague Mr. Davis found it necessary to perform an incomplete mastoid operation. As the dermatitis persists, it is conceivable that the organisms are gaining deep access. It is interesting to see this sinus, even after the use of a large flap. If hydrarg. ammoniatum is used, it must be persevered with until the dermatitis thickening has completely disappeared for some time.

Mr. J. S. Fraser: For a time I could make nothing of these cases of dermatitis of the external meatus associated with stenosis, though I tried the usual remedies, such as nitrate of silver, and so on. Then I called in the aid of a dermatologist, Dr. Cranston Low, who found several of the cases had seborrhœic dermatitis, and obtained from them a practically pure culture of the seborrhœa organism. On his advice I started treating these cases with an ointment consisting of 10 gr. precipitated sulphur, 10 gr. salicylic acid, to 1 oz. vaseline. The meatus was packed with a strip of gauze on which this ointment was smeared, and after a day or two two strips were used. I have not found anything to do so well as this.

Mr. J. F. O'Malley: I have had a large number of these cases during the last five years, but I have never seen one so extremely bad that the drainage of the middle ear was quite dammed up. I understand how in this case Mr. Davis was obliged to remove tissue so as to ensure draining of the ear. During the last two years my practice in these cases is the same as that mentioned by

Mr. Fraser. I think the active principle in that ointment is the salicylic acid. What I really use is Lassar's paste, on a strip of gauze 2 in. long: this I gently insert until I think I have reached the inner end of the meatus. If this is done from day to day, absorption goes on, and after a few weeks I have been able to look into the middle ear cavity, I have tried tubes when the meatus gets larger, but they usually aggravate it.

Mr. H. J. BANKS-DAVIS: I remember a case in a lady some years ago, with a profuse discharge from both ears, and extensive dermatitis of two years' duration. The condition had been diagnosed as chronic otorrhœa, but there was no perforation, and it was obvious that the discharge came from the ear canal. There was intense irritation, and nothing I did seemed to benefit it for months. I then ordered a paste of bismuth subnitrate, 60 gr. to the oz. of zinc ointment. This quickly relieved the irritation, and then the eczematous condition slowly subsided.

Mr. SOMERVILLE HASTINGS: I saw this case before Mr. Davis did, and I spent the better part of an hour in trying to get the smallest speculum into the meatus to get a view of the drum, but I failed. I think Mr. Davis has done right in enlarging the meatus, because the patient seemed to be in danger with a blocked meatus and suppuration of unknown origin going on behind the obstruction. I feel sure there will now be no essential difficulty in getting the dermatitis cured.

Mr. W. STUART-LOW: I have proved that cocci causing this condition fall from the scalp into the ear, and if the condition of the scalp is attended to, the ear trouble will soon be arrested. I almost always call in the skin specialist to these cases, and he seems to rely on liquor hydrarg. perchlor. with spirit lotion rubbed in at night, the latter clearing the way for the mercury and carrying it into the deeper layers of the scalp, and so eradicating the trouble.

The PRESIDENT: If you soak a strip of gauze in an alcoholic solution of boracic acid and leave it in the meatus for eight or ten hours on two or three occasions many such cases get well. It acts in the same way as the other antiseptic remedies which have been suggested.

Mr. E. D. D. DAVIS: I am sorry I did not have a bacteriological examination made. At his first visit I could not see the drum and I watched the case for six weeks and then thought I could see a perforation with middle-ear discharge. I did a conservative mastoid operation, and found the antrum to be normal, and removed all the cartilaginous meatus except a small strip of the anterior wall. The patient has had the condition ten years, and many remedies have been tried without success. He has to return to India, and in view of the stenosis I considered it safer to do a conservative mastoid operation. Suppuration still continues and I am beginning to regret that I did not do the radical operation.

Post-mortem Specimens from a Case of Cerebellar Abscess complicated by Thrombosis of the Lateral and Cavernous Sinuses.

By E. D. D. Davis, F.R.C.S.

A woman, aged 60, came to the hospital on December 8, 1917, complaining of earache of three weeks' duration followed by occipital headache. Right otorrhœa had existed from the age of 7, following measles. The patient looked ill. Temperature 99·6° F., pulse 84. There was no history of a rigor, vomiting, or of tinnitus. No local tenderness of mastoid or cerebellar region. Pupils equal, reacting to light. Coarse lateral nystagmus to right. Weakness of right orbicularis palpebrarum. Disks normal. Marked vertigo on rotation but not on irrigation. Gait normal, no incoördination, knee-jerks exaggerated and symmetrical. Right ear showed granulation tissue in the posterior superior quadrant, with the flaky discharge characteristic of cholesteatoma. Almost complete loss of hearing on the right, with marked loss of bone conduction.

Right radical mastoid operation performed a few hours after the first consultation. The mastoid antrum was deeply placed and surrounded by dense sclerosed bone. It contained a chronic cholesteatomatous abscess with pus under tension. No fistula found and no indication of an intracranial abscess. Lumbar puncture showed clear fluid and normal tension, and later examination showed lymphocytes and globulin. Patient improved, but nystagmus remained. Six days after operation she became unconscious and a second operation was performed with practically no anæsthetic. An opening was made through the inner wall of the mastoid antrum. Though the lateral sinus and middle fossa were exposed the access was very limited and bleeding occurred from the region of the superior petrosal sinus. Exploration of the posterior fossa by this route was very unsatisfactory, and a larger second opening was rapidly made behind the lateral sinus in the posterior fossa. The dura opened and puncture of the cerebellar hemisphere with sinus forceps revealed about ½ oz. of thick pus. A metal drainage tube was inserted and the patient made a rapid recovery for eight days, regaining consciousness soon after operation. Lumbar puncture showed normal fluid. On the eighth day after the second operation, patient had a rigor followed by signs of left cavernous sinus thrombosis. The right cavernous sinus thrombosis developed some days later. She died twenty-four days after the second operation.

The temporal bone, showing the operation areas, is exhibited.

There was no meningitis, but both lateral and cavernous sinuses contained suppurating blood clots.

Mr. ARTHUR CHEATLE: Here we have the same old story of acute infection of the middle ear tract during a specific fever, in an acellular type of bone with a dense outer antral wall causing a chronic discharge. Knowing what we now do, I think such a history should be impossible. I do not criticize Mr. Davis's treatment in the final stages of the case, for he did all that was possible to save life. I would like to ask how the Committee appointed by the Section to deal with the aural conditions under the Ministry of Health Bill is getting on and when it is going to report?

Section of Aspergillus from the Ear.

Shown by ARTHUR CHEATLE, F.R.C.S., and W. D'ESTE EMERY, M.D.

SECTION of aspergillus material. It shows squamous epithelium, the mycelium and the spores, and, at least, one sporangium cut exactly centrally and showing its structure very perfectly.

The material from the ear was washed in sterile water, and a black nodule was picked out. This was washed repeatedly in sterile distilled water (to remove contaminations as far as possible) and planted on maltose agar (1 per cent.).

The specimen shown was grown for ten days at the room temperature. A similar specimen kept at 37° F. gave about the same development in three days. The material was removed from the cavity of an old radical operation, and was causing pain and discharge.

DISCUSSION.

Mr. ARTHUR CHEATLE: Last year I had seven of these cases in private practice in ten months, whereas formerly about one case in two years was the average. In one case I was able to prove that the source was the bath water, for from the water in the cistern Dr. Emery was able to grow the aspergillus. Probably the increased frequency in the number of cases was due to the fact that during the war there has been neglect in cleaning out cisterns. I would like investigations of cisterns to be continued.

Dr. URBAN PRITCHARD: It is extraordinary how rare this condition is in England and how common in other countries. It used to be very common in Vienna, and I believe it is the commonest form of ear disease in Bombay.

Case for Diagnosis.

By H. J. BANKS-DAVIS, M.B.

MAN, aged 35, motor driver. Two years ago he had a "tumour removed from behind the ear" at a special hospital: Post-aural incision. Intense pain day and night ever since. The external meatal orifice and the swelling in front is so tender that examination is almost impossible. The tragus is red and the meatus occluded; there is a fissure at its lowest corner; no aural discharge. The meatus seems encroached upon, secondarily, by the hard swelling over the parotid which appears to be in connexion with the malar bone, producing perichondritis.

DISCUSSION.

Mr. ARTHUR CHEATLE: I think it is a case of malignant disease. Dr. Bond evidently removed something from the meatus by means of a post-aural incision and further growth has occurred. I had a similar case in a young subject many years ago, and in spite of several operations it ended fatally; it is recorded in the *Transactions of the Otological Society of the United Kingdom.*

Mr. SYDNEY SCOTT: Is it not worth while to remove part of the growth to ascertain the nature of the disease now? I would remove a wedge-shaped piece from the external auditory meatus, and examine it histologically. If it proves to be malignant, I should recommend a widespread incision, with the parotid salivary and adjacent lymphatic glands.

Mr. H. J. BANKS-DAVIS: I think the swelling in front of the ear is a secondary perichondritis. It has gone on for two years, and pain is incessant.

Later Note.—On January 21, I cut out the meatus, curetted the cavity and cauterized the parts with Paquelin's cautery. The middle ear and membrane were not involved. The pathological report is that the growth is a "columnar-celled carcinoma originating in one of the sebaceous glands or hair follicle ducts in the external cartilaginous meatus." Pain ceased: X-ray treatment is being carried out.

Section of Otology.

President—Mr. Herbert Tilley, F.R.C.S.

(Dr. Albert Gray, Vice-President, in the Chair.)

Congenital Cupping of the External Auditory Meatus with Keratosis.[1]

By Richard Lake, F.R.C.S.

The patient has a deep depression occupying most of the external two-thirds of the meatus, with the exception of the orifice. The orifice was very narrow but is now dilated up to its greatest extent and so a good view of the condition is obtained. Probably no keratosis is evident.

Patient's mother has been for some time under my care for the results of a similar condition.

Exhibitor would like to hear especially any remarks on suggested treatment.

DISCUSSION.

Dr. Peters (for Mr. Lake): The case is now a little different since the notes were sent. Originally there was almost complete stenosis of the meatus while the cup-like expansion was filled with a frothy mass. Mr. Lake would be glad of suggestions to deal with the expansion of the meatus: he thinks that the specimens exhibited in the Toynbee Museum are examples of this condition, keratosis obturans. I believe they are exhibited there as sebaceous cysts.

Mr. Somerville Hastings: Is it usual to find the meatus contracted in this condition? I have never seen it before.

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[1] At a meeting of the Section, held February 20, 1920.

Dr. URBAN PRITCHARD: Mr. Lake now seems to have got the patient almost well, so we cannot really judge what was the former condition.

The CHAIRMAN (Dr. A. A. GRAY): I am not well acquainted with the condition. If this is as difficult to treat as keratosis in the throat, it must be tedious to deal with.

Dr. PETERS (in reply): In this case the meatus was first dilated by means of a tube, then ether was instilled. The ether caused some burning, but it also has an anæsthetic effect. Mr. Lake thought it might be advisable to try to diminish the cupping by dissecting away the lining of the meatus and introducing a piece of cartilage. The expansion in this case is not extreme. I have not had a great experience of such cases, but I believe atresia is not uncommon in them. In the mother there is a similar condition of keratosis, with partial stenosis.

Case of Purulent Otitic Meningitis; Operation; Recovery.

By J. F. O'MALLEY, F.R.C.S.

PENSIONER A. G. History of left aural discharge from September, 1915. Right ear also moist at times.

September, 1919: End of first week he collapsed in the street and was treated by a doctor, who gave him "ear drops" and said "it was not serious." End of second week: Pain set in, in the left ear, and he had attacks of vomiting and was unable to stand.

September 21: He was removed by ambulance to the Brook Hospital.

September 22: I was called in to see him. Right ear: Large perforation of tympanic membrane and some pus in middle-ear cavity. Left ear: Profuse suppuration from middle-ear cavity, with total loss of membrane and much granulation tissue posteriorly in tympanum. Left mastoid tender to pressure, but no superficial swelling. He complained of intense headache and it was painful to flex the neck, which was rigid. Nystagmus not present. Equilibration not tested as he was too ill. Temperature, 103·4° F.; 103° F. the previous day. Kernig's sign absent.

I had him removed to the Royal Herbert Hospital; lumbar puncture was done and cerebro-spinal fluid examined.

Pathologist's report (Captain Young): "September 23, 1919: 25 c.c. of slightly opalescent fluid drawn off. Very little increase in pressure. Deposit fine, greyish, clinging to test-tube wall. Cell count (approximate):

Polymorphs, 45 per cent.; mononuclears, 55 per cent.; Gram-positive diplococci and Gram-negative bacilli, with marked polar staining, found in considerable numbers, some intra-cellular, some extra-cellular. Proportion of degenerated pus cells, not very large."

September 23, 1919: A mastoid operation was performed and marked sepsis found.

September 24, 1919: He felt more comfortable, not sick, and temperature had fallen to 100° F.

September 25, 1919: Still better and temperature normal and no pyrexia since. Another lumbar puncture was done on this date. The report is as follows:—

(Captain Young): "September 25, 1919: Total leucocyte count, 10,500 per cubic millimetre. Differential count: Neutro-polymorphs, 87 per cent.; small lymphocytes, 6 per cent.; large lymphocytes, 3 per cent.; hyalines, 2 per cent.; transitional, 2 per cent. High percentage of polymorphs suggests strongly pus under pressure. Culture: (1) A tetracoccus; (2) proteus vulgaris; (3) a short Gram-negative cocco-bacillus at present unidentified."

He has made a slow, but uninterrupted recovery. There has been considerable formation of granulation tissue in the posterior part of tympanum at the inner end of the posterior meatal wall and I have recently removed this.

Labyrinthitis; Diffuse Purulent Meningitis; Labyrinthectomy; Cure.

By P. Watson-Williams, M.D.

E. T., aged 37, admitted September 23, 1919. History of left ear running since he was a boy. Much pain occurs in ear at times.

On admission: Little discharge from left ear. Vertigo, falls to left. Objects viewed swing to left. Slight mastoid tenderness left side. Spontaneous nystagmus to left. Caloric test increases nystagmus to right with hot water. Lies in bed on right side; complete left bone deafness. The caloric test response suggested an acute infection of the labyrinth, and when first seen fistula reaction was obtained, from which it may be inferred the labyrinth was not "dead" at that time.

September 26, 1919: Pain at back of neck; head retraction. Lumbar puncture: increased pressure, turbid fluid, 40 c.c. removed.

Pathological report of cerebro-spinal fluid: Diplococci (stained film); many polymorphs; culture sterile. Temperature, 100° F. The rapid development of definite symptoms of meningitis rendered immediate operation necessary and prevented fuller observation beforehand. Radical mastoid operation. Labyrinthectomy. The external semicircular canal opened and followed into the ampulla, and the cochlea opened up.

September 27, 1919: General condition fair. Daily dressing and lumbar puncture. The cerebro-spinal fluid clearing gradually, pressure slightly increased.

October 2, 1919: Headache better. Cerebro-spinal fluid clear (a few polymorphs). Wound clean.

October 6, 1919: Headache. Head retraction. Cerebro-spinal fluid pressure, 15 c.c. removed by lumbar puncture, very turbid; 1 c.c. collosol argentum injected intrathecally. Temperature irregular. Pathological report: 16,800 leucocytes per cubic millimetre; 90 per cent. polymorphs; diplococci (and *Streptococcus brevis*).

October 7, 1919: Better; 3 c.c. collosol argentum intravenously before puncture.

October 8-17, 1919: Daily lumbar puncture preceded by intravenous collosol argentum 3 to 5 c.c. Cerebro-spinal fluid gradually cleared up. Some incontinence of urine. Temperature steadier. Gradual convalescence to present condition.

December 6, 1919: Present condition generally very good. Walks with no vertigo. Facial paresis but no R.D. Small clean sinus not yet healed.

Summary.—In this case we have a definite case of labyrinth infection with consequent meningitis. On draining the labyrinth, the meningitis began to clear up. Ten days after the operation there was an exacerbation of the headache and the cerebro-spinal fluid was like pus. The cell count reached 16,800 cells per cubic millimetre, with 90 per cent. polymorphonuclears showing phagocytosis, and *Streptococcus brevis* was identified. The recovery from this condition was sufficiently complete and rapid to be called startling. The technique was the alternate intrathecal and intravenous injection of silver, until it was observed that the silver appeared more efficacious when intravenous injection only was practised. There were no untoward effects from the injections. The temperature steadied almost at once after they were started; it became slightly irregular again when they were stopped, but there was never any cause for anxiety in the general or local condition.

DISCUSSION.

Mr. O'MALLEY: The bone conduction on the affected side in my case is shortened by half. He has some hearing, but there is no response to the caloric test. Recently, when removing excessive granulation tissue that grew in the mastoid cavity, I could not get it to epithelialize, and I did lumbar puncture again and found the fluid normal. I did this because the patient was already under the anæsthetic, and I took the opportunity of examining the present state of the cerebro-spinal fluid.

Mr. E. D. D. DAVIS: With regard to Dr. Watson-Williams's case, was the patient drowsy or comatose? I take it he did not vomit, and optic neuritis was not present. The cerebro-spinal fluid was turbid, but it is not stated whether micro-organisms or pus cells were present. There is no mention of pus having been found when the labyrinth was opened, and at a later date an exacerbation of symptoms occurred, with head retraction. It is known that a certain number of subjects of labyrinthotomy die from meningitis, but whether there was any subsequent infection of the meninges in this case I do not know. The case does not appear to have been a very clear case of meningitis, and I think that the diagnosis of diffuse purulent meningitis is not quite accurate.

Mr. E. WATSON-WILLIAMS: The patient had symptoms of meningitis *before* the operation; pain at the back of the neck, head retraction, and turbid cerebro-spinal fluid, which was under pressure. Although organisms were not cultivated from that, they were cultivated later. Later the *Streptococcus brevis* was found: the diplococci were seen at the start. The exacerbation came on on the tenth day, and it could hardly have been due to infection through the labyrinth wound, which by that time was apparently covered by healthy granulations, it having been dressed continuously with silver-soaked gauze, and there was no further rise of temperature then. The increased headache was probably due to temporary discontinuance of lumbar punctures, and was soon relieved on their being resumed. There is no note of pus being seen in the labyrinth when it was opened, but there was the fistula in the external canal.

Mr. G. W. DAWSON: I have not had a case of purulent meningitis, but I have had one of serous meningitis. That patient was unconscious, with paralysis of the left side of body, and I diagnosed it as meningitis. I incised and drained the meninges, and the patient recovered. With regard to Dr. Watson-Williams's case, it is not clear to me that it was necessary to open the labyrinth. I have operated on about 120 mastoids in the last few years, and I have never yet had occasion to open the labyrinth, though in several of my cases there were symptoms similar to those in Dr. Watson-Williams's case.

Dr. Dan McKenzie: I have been impressed at different times by the good results reported here as following repeated lumbar puncture in meningitis. So much so, that I feel rather inclined in future to limit my efforts to this mode of drainage. But one essential in all cases is to remove the primary focus of infection, and that being so the labyrinth, if it is infected, should be opened and drained. And, if it is opened, the further step of draining the meninges through the internal auditory meatus is certainly worth trying. We are generally led to believe that lumbar tapping or drainage is free from danger, and, as we know, the cannula is sometimes left in the spinal canal for hours on end. But I have recently had a fatality due to spinal tapping. The patient was a boy, aged 10, and was suffering from acute otogenic meningitis. I tapped the lumbar spine under an anæsthetic, withdrawing from 30 to 40 c.c. of turbid cerebro-spinal fluid. Respiration at once became very slow, and then gradually stopped altogether. Artificial respiration kept him alive while I explored the cerebellum, as this sudden paralysis of respiration, of course, pointed to a cerebellar abscess. Nothing was found in the posterior fossa, not even cerebrospinal fluid. The boy lived for only half an hour afterwards, artificial respiration being continued throughout. The diagnosis of general purulent meningitis was confirmed by post-mortem. There was no abscess in the brain. This accident clearly proves that spinal tapping must be conducted with moderation, at all events in children. What happened, I take it, was that the cisternæ at the base were emptied, and the brain stem sank into the foramen magnum, "corking" it, so that the medullary centre for respiration became paralysed by direct pressure.

Mr. Somerville Hastings: These two cases show us that cases of apparent meningitis may get well if a mastoid operation only is performed or if collosol argentum is injected. I should be glad to hear whether members still carry out labyrinthine drainage through the internal auditory meatus in meningitis when the labyrinth is destroyed. I have done this, but all the patients on whom I have performed this operation have died, whereas in those in whom I have done less—lumbar puncture, or mastoid only—there have been recoveries.

Mr. Watson-Williams: I have had two cases of collapse, but not very alarming, following the intravenous injection of Crookes' collosol silver, but it was with a pre-war specimen, and we were inclined to think it was no longer "colloidal." With the new silver preparation I have never seen an unfortunate result.

Mr. Stuart-Low: In suspected cases of meningitis with headache, a temperature, vomiting, and a history of ear trouble, my practice is to perform a cortical mastoid operation in the first place, and then, if the serious symptoms still continue, it is easy to proceed to an opening up of the external semicircular canal through the route of this operation. It is safer than lumbar puncture. I think Dr. McKenzie's fatal case points to the wisdom of what I say.

Mr. G. J. JENKINS: If there is evidence of septic labyrinthitis and meningitis in neighbourhood of the internal auditory meatus, it is indicated that the region should be drained through the labyrinth. I prefer West and Scott's operation and have had several cases of recovery from the condition by this operation. Lumbar puncture cannot be of great value in such conditions.

Mr. SYDNEY SCOTT: I cannot help thinking that those who do not yet admit the rationale of the translabyrinthine operation, which is applicable to cases of leptomeningitis following labyrinthine infection, would not fail to find reasons for adopting the procedure if they paid regular visits to the post-mortem rooms in any large general hospital. It was there I found a not inconsiderable number of cases of meningitis, which came from general wards, in which the path of infection from the ear to the posterior cranial fossa was shown by histological sections to pass through the labyrinth. If we get such cases of meningitis early enough, I believe recovery will be more frequent, and I am glad to hear Mr. Jenkins's observations on this matter. With regard to Dr. P. Watson-Williams's case, I fail to understand why the labyrinth was opened. Are we justified in regarding this as a case of infective labyrinthitis? If so, how is it that the labyrinth reacted normally to the caloric tests? I believe a labyrinth to be infected only when it is defunct, and I should not feel justified in opening the semi-circular canals and vestibule in such a case unless they are defunct.

The CHAIRMAN: I agree with Mr. Scott as to the findings in the post-mortem room. Recently I had a case in the Western Infirmary, Glasgow, examined after death. The infection came through the saccus endolymphaticus, and spread to the posterior fossa. It was not a diffuse purulent meningitis, but the meninges over all the surface of the brain were intensely injected. There was no pus except in the posterior fossa, around the foramen magnum; one would almost call it an abscess formation. It is interesting to note that in Dr. Watson-Williams's case the streptococcus was found, and yet, with that virulent organism present, the patient recovered.

Mr. O'MALLEY (in reply): Many interesting opinions have been expressed during the discussion. At the time I was called upon to treat the case my mind was much exercised as to what degree of operation I should perform. The lumbar puncture and examination of the cerebro-spinal fluid showed the case to be one of purulent meningitis, but the organisms present were not of the most virulent type—namely streptococci, and some of the organisms were intracellular, indicating that the patient's resistance to infection was responding well and that the case was therefore not very acute. At the time of operation I also found gross sepsis in the mastoid, and for these reasons I refrained from doing more than providing complete drainage from this region, holding in reserve the more extensive operation of intra-cranial drainage for next day, if necessary. I agree with those who advocate the larger operation in acute fulminating cases, and the favourable result obtained here by a less active line of procedure would not make me hesitate to follow the plan they recommend.

Mr. E. WATSON-WILLIAMS (in reply): The notes were not very full when the patient came in, but we were satisfied at the time that the labyrinth was infected. We had very little opportunity of doing more complete tests on this man, he was too ill.[1]

Case of Menière's Syndrome.

By LIONEL COLLEDGE, F.R.C.S.

PATIENT is a male, aged 42. Five years ago he suffered from repeated attacks of giddiness for twelve months. He recovered from this after an operation on the nose. For the last two months he has suffered from repeated attacks of vertigo, falling forwards. The pavement appears to come up and hit him. On one occasion he fell so suddenly as to cut his face, but usually has sufficient warning to catch hold of some support. He also suffers from deafness and tinnitus in the left ear. The tympanic membranes are normal, except for slight retraction of the left. Eustachian tubes are patent. Catheterization relieves the tinnitus temporarily but does not improve the hearing. Weber's test: The sound is referred to the right ear. The left ear shows marked loss of bone conduction and positive Rinne. The loss for high forks is pronounced. Hearing in the right ear is normal. Caloric test with cold water elicits no response in either ear. Rotation produces no nystagmus, except a few movements lasting two or three seconds on rotation from right to left. Rotation produces vertigo and nausea which the caloric test with cold water does not. Wassermann's reaction is positive.

There has been a great amelioration in the symptoms since the patient has been treated with inunctions of mercury.

DISCUSSION.

Mr. COLLEDGE: What struck me about the patient was, that he had a very diminished irritability in both vestibules; and, although he is better, he still gets attacks once a week. In spite of his Wassermann reaction being positive, I do not think it necessarily follows that the symptoms are due to syphilis. He has got rather low blood-pressure, 110. I ask for recommendations as to any treatment likely to do him good; he has been having these attacks, on and off, for seven years.

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[1] The case will be reported in greater detail in the *Journal of Laryngology*, July, 1920.

Mr. STUART-LOW: Following on the recommendation of Dr. Dundas Grant, I have often found great relief to the patient by administering $\frac{1}{2}$ gr. doses of quinine three times a day, which I learnt when his assistant surgeon; it acts as a tonic to the vestibulular nerves and braces them up. Dr. Wyatt Wingrave proved years ago that tea and tobacco should be forbidden these patients, as there is, as it were, a scotoma in the ear, just as tobacco causes one in the eye. It is also most advisable and advantageous to keep the patient in bed for some days, or even a week, while the vertigo lasts.

Dr. KELSON: Five years ago, I showed here a case of similar character, in which the man was very giddy, and was prevented thereby from following his occupation, that of house-painter. Following what was done in a case of Mr. Jenkins, I removed part of the wall of the external semicircular canal. The giddiness ceased, and the man returned to his work. Two years later I had a letter from the doctor stating that the man was then still all right as regards giddiness. I cannot quite understand how this result is obtained.

Dr. WILLIAM HILL: Mr. Stuart-Low said Dr. Dundas Grant had proved the value of quinine in such cases as this. I do not think that "proved" is the right word to use. I may say small doses of quinine have usually failed to give any relief in my cases, and I have never observed a case in which it did much good, though I know there is evidence of occasional good from it in the hands of others.

The CHAIRMAN: I find rest in bed is most useful in these cases, but it needs to be prolonged rest, and this makes life almost unendurable to these patients. I have not found much benefit from drugs, except bromides, and even in that case the dose must be such as to make the life of the patient miserable, and the remedy may be worse than the disease.

A Note on Vestibular Function.

By DAN McKENZIE, M.D.

THE following speculations on the relationship of the vestibular with the cochlear organ suggest that as they are associated in space so their functions are not so different as they are generally, if tacitly, assumed to be.

That the vestibular end-organ, in addition to its stimulation by varying pressure or waves of endolymph, also reacts to sound-waves is probable, for the following reasons:—

(1) The same system of water-tubes is common to both organs. Endolymph bathes both the scala cochleæ and the membranous semicircular canals. Anatomically, therefore, both sense-organs in sharing the same fluid, are both alike exposed to sound-vibrations. That being so, it would be strange if one of the organs was insensitive to them. On the other hand, it might be urged that the same reasoning might apply to the cochlea, and that as the cristæ of the canals are stimulated by massive endolymphatic movements so also the hair-cells of the cochlea ought to be similarly stimulated. But this objection is removed when we consider that the shape and disposition of the canals favour those movements, whereas the cochlea is shut off from them by the interposition of the saccule, the utricle, and their fine connecting tubules.

(2) The footplate of the stapes, through which sound vibrations pass to the endolymph, lies as near, or nearer, to the sense-epithelium of canals as it does to that of the cochlea. Thus it is to be noted that while the arrangement of the labyrinth is such as to favour the confinement of massive fluid movements to the canalicular side, on the other hand it is such as to render possible the general distribution of molecular vibrations from the stapes throughout the whole of the labyrinth.

(3) Vertigo is known sometimes to be caused by loud sounds.

(4) Noise-deafness, produced by the habitual exposure to harsh noises, is associated with a reduction in the responses to the vestibular tests. (D. M., confirmed by J. Ritchie Rodger.)

If it is true that over-loud sounds destroy or damage the canalicular sense-epithelium, then that epithelium must obviously be sensitive to sound waves. But the question now arises: what physiological purpose can be served by the reaction to sound-waves of the vestibular end-organ? The question may be answered as follows: It is known that general muscle tonus is under the control, more or less, of sensory impressions derived from the semicircular canals and transmitted through the cerebellum to the skeletal muscles. These sensory impressions and the resulting muscle tonus are usually referred to the agency of endolymph movement following the varying movements of the head. But muscle tonus persists when the head is perfectly motionless, and for this reason some have assumed the hair-cells of the cristæ to be ciliated cells with a perpetual lashing movement, hastened or slowed by endolymph movements, but not initiated by these movements.

But it is also agreed that muscular tonus is powerfully affected by sound and the only point in doubt is whether the sound operates through the cochlear epithelium only, or, as I now suggest, also through the canalicular cells.

As bearing upon this point it is noteworthy that in the "start" of the body that frequently follows a sudden loud noise, I have at times observed that the "start" precedes in time the reception of the sound by the consciousness; that is to say, the start is direct as well as involuntary, being effected through a shorter nerve-path than that through the higher cerebral centres, and I suggest that it is the result of a sudden and violent stimulation of the canalicular hair-cells. The protective nature of the "starting" at unexpected sounds is obvious.

For these reasons, therefore, I make the suggestion that the vestibular organ, in addition to being stimulated by relatively massive changes in endolymphatic pressure, may also be responsive to the molecular vibrations of sound.

DISCUSSION.

Dr. D. McKenzie: , Since this note was distributed I have received from Dr. P. McBride the copy of an article written by him many years ago,[1] in which he makes the same claim for the canalicular response to sound waves that I am making now. Indeed, his arguments are almost precisely the same as mine, with the exception of No. 4 above, which is necessarily of recent origin. McBride further suggests that the intimate connexion between sound-reception and muscular movement in animals results in a turning of the animal's head towards the quarter from which the sound emanates.

Dr. A. A. Gray: This is a very interesting subject physiologically. The question is whether the vestibule responds to sound waves. In respect to Ewald's theory, that the function of the vestibule and canals is to give tone to the skeletal muscles, I do not think there is much evidence in support of such a theory. Patients who have had both labyrinths destroyed are in no way deficient in muscle-tone. Furthermore, in such animals as the whale and the porpoise, in which these structures have undergone a remarkable degree of atrophy, the muscle-tone can hardly be said to be diminished! Ewald put far too much weight on physiological experiment, and too little upon evidence derived from clinical observation and from comparative anatomy.

[1] *Journ. Anat. and Physiol.*, xvii.

Facial Paralysis with Nerve Deafness and Impaired Vestibular Reactions. A Case for Diagnosis.

By DAN MCKENZIE, M.D.

THE patient is a woman, aged 27, whom I first saw a week ago. The history is obscure, but, as far as can be made out, four years ago, after "influenza" left facial paralysis appeared. It seems to have been complete at the start. She attended one of the hospitals for nervous diseases, and was treated with "electricity" for some time. Since then nothing further has been done, and no notice was taken of the severe deafness in the corresponding ear. According to her story, she has always been a little deaf in this ear, but it has lately got worse. Cross-questioning has elicited the fact that she has had at least two attacks of severe vertigo with vomiting ("giddy, bilious attacks"), the first of these being about a year previous to the onset of the paralysis. There is no history of herpes either before or since the attack of "influenza." Thus we may probably exclude herpes.

Her present condition shows absolute facial paralysis of the left side of the face, with severe (nearly absolute) nerve deafness in the ear of the same side. There is well-marked spontaneous nystagmus to the right, and a feeble response to the cold caloric test in the left ear. But there is no other indication of a gross cerebellar lesion, or of raised intracranial pressure. There is no error in pointing; no dysdiadokokinesis; no changes in the optic disks; and no involvement of any other cranial nerve. Thus, in view of the long duration of the symptoms, we are probably dealing with a definite local lesion affecting the seventh and eighth nerves, and, as herpes seems to be excluded by the history, I am inclined to think that the lesion is probably a small tumour in the internal auditory meatus, which has interrupted first and most completely the facial, and later the cochlear and vestibular nerves.

Later Note.—Further examination shows that there is nerve deafness also in the right ear, and that the Wassermann (blood) reaction is "partial positive," so that the lesion is probably syphilitic and meningeal.

Rodent Ulcer of External Auditory Canal.

By SYDNEY SCOTT, F.R.C.S.

S. R., AGED 59, first seen August, 1912, complaining of four years' painless ulcer. Membrana tympani natural. An ulcer about the meatus extends along its posterior and interior walls.

Operation August 14, 1912: An incision was made as for a mastoid operation and the whole of the external osseous and cartilaginous canal was excised. The area was skin-grafted. Pathologist's report: "Rodent ulcer."

Operation, November 21, 1913: Removal of a small recurrence. Section still showed rodent ulcer.

Operation, September 12, 1916: Excision of ulcer of doubtful character at site of previous operation. Pathological report: "None of the tissues sent for section shows any trace of malignancy."

[Histological sections shown.]

Epithelioma of External Auditory Canal which Invaded the Tympanum.

By SYDNEY SCOTT, F.R.C.S.

E. A., AGED 25, first seen January, 1917, complaining of deafness and otorrhœa.

Exploratory operation, February 15, 1917: Pathologist's report: "Squamous-celled carcinoma" (Dr. Stansfeld, confirmed by Professor Andrewes).

Removal of growth, February 23, 1917. Operation notes: "The radical incision was prolonged forwards and downwards to de-limit a broad flap including the pinna. The flap was turned forward and the osseous and the rest of the fibro-cartilaginous meatus was removed, with the remains of the tympanic ring. The outer attic wall, membrana tympani and ossicles were also removed with the exception of the stapes which was felt and left *in situ*. The geniculate ganglion of the facial was exposed and the stylomastoid foramen was seen, the Fallopian canal being intact. The Eustachian tube, the promontory, the pelvis ovalis were all identified. The pre-auricular gland was removed but found histologically free from growth.

Patient was presented at the meeting of the Otological Section, May 18, 1917, on account of the comparatively early age of incidence.[1]

[Histological sections shown.]

Notes on Epithelioma of the External Auditory Canal which invaded the Mastoid Antrum without affecting the Drum.

By SYDNEY SCOTT, F.R.C.S.

THE greater part of the temporal bone had been removed four months before death.

ABSTRACT OF NOTES BY MR. K. BELLWOOD, HOUSE SURGEON, ST. BARTHOLOMEW'S HOSPITAL.

A. H., aged 60. Admitted to St. Bartholomew's Hospital on May 25, 1919. History of right otorrhœa for at least six months with swelling behind the right ear. Twitchings of right side of face, six weeks. Facial palsy, right, four days.

On admission: Right pinna pushed outward and forward and fluctuating swelling over mastoid region. Brawny swelling above and in front of auricle. Meatus blocked by swelling from above—purulent discharge. Glands in neck not enlarged—no tenderness. Facial palsy. Infranuclear type, right. Eye movements normal—no nystagmus. No vertigo. Temperature, 99·8° F.; pulse, 128; respiration, 24.

Treatment: May 25, 1919.—Incision over mastoid as for Schwartze operation by house surgeon. Abscess drained under periosteum. A mass of material (? granulation) presented through the junction of cartilaginous and bony canals. This also occupied the middle ear with erosion of bone round aqueductus Fallopii and tegmen. Dura mater roughened. Wound packed. Thought to be ? cholesteatoma. Patient's condition satisfactory. May 29, 1919.—Further exploration by Mr. Scott raised suspicion of neoplastic nature. Piece removed for section.

Pathological Report on Section: Squamous-celled carcinoma. June 5, 1919: Radical removal (Scott); horse-shoe flap; central excision of auricle and adjacent skin. Parts removed: Parotid gland; temporo-mandibular joint, *in toto;* posterior portion temporal muscle; lower two-thirds of auricle; entire external auditory canal; membrana tympani and ossicles, including stapes; labyrinth; lateral sinus packed and occluded; facial nerve found near incus.

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[1] See *Proc. Roy. Soc. Med.*, 1917, x (Sect. Otol.), p. 129.

N.B.—Membrana tympani and ossicles found intact. Mucosa swollen. Growth around external osseous canal. Normal cells apex of mastoid.

Patient made a good recovery. The wound granulated well and proceeded to heal steadily. The general health was good, and temperature and pulse normal. Granulating area treated by fulguration twice weekly. Sparks $\frac{1}{12}$ in. long. Discharged August 6, 1919.—Small area only remaining with granulations. Patient failed to present herself again at hospital. Information received that she died about the end of September, 1919. Details not obtained.

DISCUSSION.

Mr. SYDNEY SCOTT: These three cases were brought forward as examples of primary malignant disease of the external auditory canal. Though the middle ear was found to be involved in the two cases of epithelioma, the evidence shows that the middle ear was invaded from the external auditory canal, in one case as a polypoid extension through a perforation of the drum, in the other by direct destruction of the bony antero-inferior wall of the antrum. For, in both cases, all the ossicles and the apical mastoid cells were absolutely intact, and in one case the tympanic membrane was perfectly normal and complete, and in the other, though incomplete at the site of invasion of the tympanum, was quite free from all signs of inflammation. The first patient, who had a rodent ulcer, has recently developed a small external fissure in front of the lobule of the ear, and it is owing to the doubt as to its nature she is here to-day. I propose to excise this and shall report whether it shows signs of a recurrence of the original disease.[1] The second case, the young woman who was shown here about three years ago, but has not turned up to-day owing to an attack of malaria, wrote from India three months ago complaining of pain and swelling in the site of the former operation. When I saw her last week I could find no swelling, but I should have been glad to have obtained the opinion of members as to whether there was any evidence of deep-seated recurrence. There is certainly no swelling in the parotid region, the scar appears to be quite sound, and I can find no enlarged glands in the neck. A specimen of the primary growth, showing the characteristics of epithelioma, is beneath the microscope. As to the third case, since I arranged to show this patient, I find she died in an infirmary, where, unfortunately, there was no autopsy. Though she was said to have died of apoplexy, I learn from the medical officer that it is quite likely that death was due to a brain abscess and meningitis. Apparently she was admitted more or less moribund. The interest of the case centres about the condition of the ear when first admitted to St. Bartholomew's Hospital last May. She was sent in one night as an emergency mastoid case, having an œdematous swelling over the mastoid, protrusion of the pinna, discharge from the ear, and facial paralysis. The house surgeon opened a superficial mastoid abscess and finding what he took to be cholesteatomatous material, naturally concluded the case would require to be dealt with more fully than he was permitted to do, and left the case for me to see next day. I noticed a suspicious growth protruding from the external auditory meatus and had this examined histologically. The section is shown beneath the microscope and presents the typical appearance of epithelioma. Exactly how extensive the disease was I did not know, but a few days later, having first ligatured the external carotid artery, I turned down a large Ω flap from the side of the head and removed the whole of the parotid salivary gland, the posterior half of the temporal muscle, and excised the temporo-mandibular joint (because the disease was seen invading the root of the zygoma), the whole of the mastoid process, tympanic plate, and as much of the petrous portion of the temporal bone as it was possible to remove without opening the carotid canal or internal auditory canal. During this procedure it was found evident that the growth began in the osseous portion of the external auditory canal and had not attacked the tympanic membrane, but had eaten itself into the mastoid antrum by direct extension upwards and backwards. Hence it had destroyed the tegmen and the base of the mastoid without attacking the mastoid cells in the apex of the bone. The facial canal was opened and destroyed in the third part of its course. Though the growth resembled cholesteatoma in appearance, it was distinctly friable instead of being soft and greasy. The outer part beneath the skin had apparently suppurated, forming the superficial abscess with which the patient was admitted, but there was no pus elsewhere. It will be recalled that the second patient had an inframeatal abscess when she was first seen three years ago, at the time the primary growth was removed. In spite of the complete excision of the whole temporo-mandibular joint, temporal as well as mandibular portions being removed, there was no obvious distortion of the jaw, and, being edentulous, the patient found no discomfort from the loss of this articulation. She showed the usual signs following removal of the labyrinth, but as regards the wound she made a good recovery. When she left the hospital only a small area was granulating. As the dura mater was involved, diathermy was inapplicable, and all one could do was to curette the membrane down to what appeared its normal thickness. Fulguration was applied by Dr. E. P. Cumberbatch, but with little expectation of success, and the patient was lost sight of when she left the hospital until we heard of her death four months after the operation. The principal points which lead me to describe this case here were (1) the preservation of the drum and auditory ossicles, though imbedded as it were in a mass of malignant growth ; (2) the necessity for excising the whole of the temporo-mandibular joint and the manner in which its loss was borne.

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[1] Ulcer excised March 3, 1920, and found histologically to be "rodent ulcer" again.

Mr. J. E. O'MALLEY: The fact that Mr. Scott's third case came to him as one of urgency is of interest to me because two years ago I had a similar case brought as an emergency case to the Royal Herbert Hospital, Woolwich. A soldier, aged 36, came in with a history of chronic discharge from his ear. There was an extensive swelling of the external meatus and also over the mastoid. It was only when I came to do the plastic part of the operation on the ear that I began to be suspicious of its nature. I had a portion examined, and it was shown to be epithelioma, so that a more extensive operation had to be carried out. Such a case presents a serious pitfall from the point of view of prognosis.

Dr. DAN McKENZIE: The last case of epithelioma of the ear I showed here was operated on by means of the diathermy knife and although an extensive area was exposed, including the bone of the mastoid, healing quickly followed a graft on the raw surfaces. I wish to take this opportunity of once more impressing upon members the value of diathermy excision of cancerous growths. I think that the time may come when it will be considered bad surgery to remove malignant growths in any part of the body with the cold knife. The diathermy knife kills all living cells in its neighbourhood, and thus it sterilizes the wound it inflicts so far as cancer cells are concerned. The cold knife, on the other hand, as we know, is very apt to sow living cancer cells upon the edges of its wounds. There is a further point which comes into play in cancer operations in the region of the ear, and that is that probably no harm is produced when the diathermy kills bone. It is a peculiar and advantageous quality of this agent that its wounds are aseptic and remain aseptic, so that long-drawn-out bone suppuration does not, as we might expect, follow a diathermy burn of osseous tissue.

Mr. H. J. BANKS-DAVIS: What is the opinion of members of the Section in regard to after-treatment of cases of malignant disease of the meatus. After the preliminary operation I have sent patients to the Radium Institute. I now hear they do not do "so well under radium as under X-ray treatment." Last meeting I showed a man aged 34, who had intense pain in his ear. He had been operated upon at Golden Square two years before and the report was columnar-celled carcinoma. He had perichondritis, and told me he had "not slept for two years." Mr. Cheatle diagnosed malignant disease, and this it proved to be. I excised the meatus, but on going deeper I found he had a larger growth. There was no involvement of the drum or mastoid cells. A large opening was made, and pain ceased from that moment. What ought to be done with him now? I agree as to the suddenness with which malignant disease makes itself known. A patient with malignant disease of the gullet may say: "I have not been able to swallow since this morning," or "since yesterday," and have had no similar symptoms before, although the disease is of quite old standing.

Mr. CYRIL HORSFORD: I may mention a case which is of considerable interest. When I first saw the man, he had a large abscess involving the parotid gland. There was swelling of the face, but nothing in the region of the mastoid. I felt suspicious about it. I had to make free incisions in order to get the swelling down. On microscoping a portion of the tissue, it proved to be malignant disease. I had the benefit of Mr. West's opinion, and he helped me at the operation. We decided upon an extensive operation, excising the whole growth as far as possible. The tests showed some impairment of hearing as a result of some obstruction in the meatus, but the middle ear was not destroyed. We excised, in one mass, the greater part of the parotid gland, and then we found the middle ear also was involved, and an area the size of a florin in the middle fossa destroyed. Therefore the dura mater in that region was exposed. No more was done, because it was felt that the dura mater was infected. The wound healed, the cavity filled up, and we were able to send him back to Africa with an almost healed wound. Dr. Finzi told me it would not be wise to use radium, because any dose powerful enough to influence the disease would have a bad effect on the dura mater.

Dr. W. HILL: I have only treated two cases of malignant disease of the ear. One of these can be described as an urgency case: it was sent to the hospital with a mastoid swelling which required opening at once. It was evident on inspection that there was a fungating carcinoma of the meatus, extending into the concha, and in addition there were acute mastoid symptoms. In each of my two cases a partial operation only was done, but in each the pain was relieved for quite a long time. In one of these I tried radium soon after the operation; I do not know that it made much difference: the patient died miserably all the same. I also used radium water as a dressing in the other case, because there was an American giving away a large stock over here who was anxious for me to try it. I used it on four hospital patients after ordinary methods and they seemed gratified, but in view of its feeble potency I question its therapeutic value.

Mr. G. J. JENKINS: Have members paid sufficient attention to the lymphatic tracts in connexion with the external auditory meatus and middle ear. The anterior part of the meatus, the floor particularly, drain between the osseous and membranous portions of the external auditory meatus. The posterior part is drained deep to the anterior surface of the mastoid, and the other parts of the middle-ear tract are drained to the pterygoid region. If we are to take away the whole of a carcinoma in the region of the external auditory meatus, close to the tympanic membrane, a very extensive operation is required. I do not think we shall get first-class results until that is recognized. We must include the whole external auditory meatus, the whole of the anterior wall, and forward into the parotid region at least, as well as the glands below the pinna.

Dr. PEGLER: I think it is worth inquiring to what extent if any, these cases of malignant disease of the external ear are liable to be followed by metastatic deposits. I have not myself heard of any such developments.

Dr. A. A. GRAY: I never saw deposits of cancer in the liver and other internal organs in these cases. Even in cases of cancer of the tongue one seldom finds such internal deposits, nor yet in cancer of the uterus. It is mostly in breast cancer cases that the deposits are seen.

Mr. SYDNEY SCOTT (in reply): I have never seen a case of rodent ulcer or epithelioma of the external auditory canal cured by radium. Radiologists whom I have consulted, have invariably advised excision when excision was possible. Anatomical considerations of the lymphatic channels, to which Mr. Jenkins refers, would lead us to sacrifice the whole of the parotid salivary gland; but the patient in the second case refused to have this done, nor would she agree to any operation on the cervical glands, and so far there is no sign of metastasis, three years since the primary growth was removed. As Mr. Horsford remarks, concerning his and Mr. West's case, it is when the operation is well advanced that we may find the condition hopeless as regards complete removal, because the dura mater may be found involved in the growth without any previous indication of its being attacked. This is the one great barrier against complete extirpation. Dr. A. A. Gray's remarks are important, and support the view that the disease proves fatal by local infiltration, not by remote metaplasia. I agree with Dr. Dan McKenzie that necrosis of bone is no deterrent to the employment of diathermy. I have induced those who hesitate to apply diathermy to waive their objection, for the necrosed bone presents a remarkably clean appearance which gradually crumbles away, if it has been invaded by malignant disease. Diathermal destruction extends farther than it is safe to go with the knife and herein is its value.

Case of Marked and Easily Elicited Labyrinth-Sinus Symptom: Pressure Nystagmus.

By SYDNEY SCOTT, F.R.C.S.

A. M., AGED 52. Right otorrhœa in childhood after scarlet fever, none for thirty years; right deafness ever since. November 1919: "Walked crooked" . . . turned round to the right. Two weeks ago, when drying her ear, she pressed it, and immediately turned violently round to the right and nearly fell into the fire; at the same time a mist came over her eyes so that she could not see. This lasted a few minutes. Next day she was walking along and again turned suddenly to the right and fell down. Local condition: Bilateral attic perforation. Labyrinthine nystagmus obtained by patient pressing her right ear; primary horizontal deviation of both eyeballs to patient's left. Slightly quicker secondary phase of ocular movement to patient's right.

Primary slight forced movement of head to left. There is also present lateral nystagmus on deviation of the eyes to the left, chiefly in the right eye. Caloric test normal, for superior and for external semicircular canals.

DISCUSSION.

Mr. H. J. BANKS-DAVIS: I was interested in this case from teaching aspects. On pressure over one ear she has a marked nystagmus. The first movement is to the left; then there is a quick return to the right. Would you claim this as nystagmus "to the left" or "to the right"? I do not think our nomenclature is really correct in this respect for teaching purposes. Mr. Scott calls it a "first movement to the left," but really the "observed movement" of the eyes is in the opposite direction, and this is what students at once notice and it is difficult to impress upon them that this very apparent movement is of no consideration compared with the initial primary movement in the opposite direction which is often hardly noticeable to untrained eyes. The nomenclature which we, as otologists, employ does not convey to others less expert what nystagmus indicates. We judge by one movement and then by another—i.e., by the grosser and "more apparent" movement, and this is very natural.

Mr. G. J. JENKINS: Wilson, of Chicago, has made a careful study of this subject and dealt with the two movements of nystagmus. The quick movement of nystagmus he localized to the higher centres and the slow to the sub-thalamic centres. The slow movement is the more important, but I think we should adhere to the present description as it is established and convenient.

Mr. SYDNEY SCOTT (in reply): The terms "nystagmus to the right" or "to the left" are, as Dr. Gray says, pure conventions. I was careful to point this out in early dissertations on this subject as Mr. Banks-Davis will probably remember.[1] We recognized a primary (i.e., the slower) movement followed by a secondary and nearly always much faster movement in the opposite direction. It is, curiously enough, the direction of this secondary phase which designates the nystagmus. For the secondary phase, the rapid jerk becomes more intense the more the eyeballs are turned towards the direction of the secondary movements. When the visual axes are turned toward the primary direction no nystagmus may be discernible. But when deviated in the direction of the secondary phase, then the nystagmus is most plainly observable. Generally speaking, we may say the primary movement is in the same plane as, or in a parallel plane with, and in the same direction, as the flow of fluid which we believe takes place in the ampulla of the corresponding semicircular canal when we employ the caloric or rotation tests, or when, as in the present case, we transmit change of pressure to a sinus in the wall of the external semicircular canal.

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[1] "The Problem of Vertigo," *Proc. Roy. Soc. Med.*, 1909, ii (Sect. Otol.), pp. 41-80.

Section of Otology.

President—Mr. HERBERT TILLEY, F.R.C.S.

Case of Chronic Suppuration in the Left "Attic" with few Objective or Subjective Symptoms while Extensive Suppuration was found to be present in the Antrum and Mastoid Cells.[1]

By HERBERT TILLEY, F.R.C.S.

THE patient was a girl, aged about 18, who complained of some deafness and occasional pain in the left ear. The ear had frequently been "moist" since childhood. Examination showed a small perforation in Shrapnell's membrane from which pus could be seen to exude when suction was applied by a Peters-Siegle's speculum. The rest of the membrane was normal in all respects. There was slight pain on deep pressure over the outer walls of the antrum and the mastoid process.

Owing to the mild degree of the symptoms the patient had been advised to have no operation and I had much difficulty in persuading her or her parents that surgical intervention was imperative. Ultimately they consented, and at the operation I found the antrum full of pus and a track of suppuration extending through the mastoid cells and denuding the lateral sinus over an area the size of a pea. The diseased areas were removed and the conservative mastoid operation was completed.

The case is shown because ossiculectomy is still advised by some otologists in the treatment of chronic attic suppuration and it is my opinion that it should be discarded, because, in the vast majority of such cases, the antrum or the mastoid cells form the true focus of infection.

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[1] At a meeting of the Section, held March 19, 1920.

I do not show this case because there is anything particularly uncommon about it, but because some of its features are of practical interest. The patient was an apparently healthy girl, only slightly deaf in the left ear. There was no obvious discharge. It was only on examining the drum that one found there was a moist perforation in Shrapnell's membrane. She was advised by a doctor to have nothing done, and it was said that a mastoid operation was out of the question. I urged the operation, however, and the mastoid antrum was found to be full of pus, which had tracked down the lateral sinus. I think the time has come when ossiculectomy in chronic abscess of the attic should be an exceptional procedure, because the infection is nearly always in the antrum, and unless that cavity is effectively and permanently drained I do not think improvement in these cases will be the rule, and unnecessary risks will be run. Ossiculectomy in such cases seems to me to be bad surgery, for it does not take cognizance of the true source of the infection and the reason for the continuance of the disease. When the drum appears to be in a good condition, and the hearing is good, it is difficult to persuade people that they may have a serious condition present. I think this case exemplified all the features which are met with in a latent case, which may later end in sudden disaster.

DISCUSSION.

Mr. ARTHUR CHEATLE: The result in the President's case is excellent. I wish to say one word about ossiculectomy: that I did not know that any aural surgeon at the present day throughout Europe or America dreams of doing ossiculectomy.

Mr. STUART-LOW: I agree that this is a very good illustration of the necessity for operation. It is the kind of case in which I insist on operation in the face of but very slight apparent evidence of trouble. There are a number of specialists who still use attic syringes: they have been exhibited at our meetings. To syringe such a case as this is bad practice. I have had the same experience as the President, that in some instances the patient has gone to somebody else and been seriously ill later—some, indeed, have lost their lives. With regard to ossiculectomy, I have seen one or two of our members do it very skilfully, but I have records showing that this procedure may light up a latent infection more deeply situated. I take exception to the site of Mr. Tilley's incision: it is too far back—an inch from the ear. I make an incision in the retro-auricular groove, and then it leaves no noticeable scar, an important matter, especially in the case of a female. The incision should also tail off under the lobule so that no mark whatever is visible some time later.

Mr. E. D. D. DAVIS: What is the experience of members in cases of chronic attic suppuration? Mr. Tilley's case was, apparently, one of acute suppuration of the attic, with pain. There is another type of case, which is seen frequently in older subjects, namely, a chronic attic suppuration with occasional discharge, and perhaps a similar condition has been present on the other side, but has healed. These people hear well and they have no symptoms except the discharge. I have opened the mastoid in some cases and found the disease to be confined to the attic, and usually cholesteatomatous. But as the mastoid antra were normal, conservative operation has been done. What is the experience of members of the conservative operation in such cases? In order to clean out the attic thoroughly it is necessary to remove its outer wall in the auditory meatus, and sacrifice the upper part of the drum. I have done that in some cases, but the result has not been very satisfactory, and I have had to perform a radical operation later. Often such patients decline operation as they have no pain, the discharge is only occasional, and the hearing good. How do these cases end? Probably they become more deaf, or undergo an acute attack of suppuration followed by an intracranial complication.

Dr. DAN MCKENZIE: An interesting question arises in regard to those perforations in the attic which I have seen mentioned in foreign journals. Are those perforations ever caused by anything but cholesteatoma? Cases such as Mr. Davis has described are very common. In some the disease is discovered accidentally. The patient is only slightly deaf, but there is perforation and obvious cholesteatoma. The question arises, What is the best thing to do for them? A number get well with ordinary simple treatment. If you operate, what operation will you choose? My experience of the modified operation in these cases has not been very good. I have had only one or two with a genuine cholesteatomatous perforation of the attic in which there has been a dry ear and a good membrane left. In order to get them dry, I had to attend to them very carefully for a long time afterwards. The great difficulty in the case of operating in the antro-tympanic region is the avoidance of sepsis. If you take a dry ear and operate upon it, as has been tried in various types of deafness, your difficulty does not lie really in the operation—it may be very successful—but in the fact that you cannot maintain asepsis of the cavities. That is probably the reason why all the various methods of operating on dry ears fail in the long run: suppuration has been induced, and scar tissue has been formed.

Mr. HUNTER TOD: I think the President has introduced what may be a very useful discussion on a matter which might very well be made a subject for a special debate. He has shown us to-day a case such as we all meet with. These cases require much judgment with regard to their treatment. I do not agree with the emphatic manner in which Mr. Cheatle has condemned the operation of ossiculectomy. I do not consider the statement should be made that no aural surgeon does ossiculectomy, but I agree that this operation should not be done except after grave consideration. Nevertheless, it is by no means an unjustifiable operation. Some years ago I published fifty cases in which I had performed ossiculectomy. These were followed up as well as possible, and to my knowledge only three required the mastoid operation at a later date. One patient died some years later from lateral sinus thrombosis, but he had completely neglected further treatment after the primary operation. Before performing the operation of ossiculectomy I always warn the patients that they may have to submit to the mastoid operation later on if a cure be not obtained. The treatment to be adopted depends on the individual case; one cannot treat all cases in the same way. In cases of simple suppuration, merely a mucous membrane lesion with no cholesteatoma, removal of the malleus, incus, and outer wall of the attic usually effects a cure. In cases in which the attic wall is involved, with the presence of granulations or cholesteatomatous formation, ossiculectomy is not advisable because the antrum is usually also affected. In cases suitable for ossiculectomy one also has to consider the hearing power of the patient, remembering that after the operation of ossiculectomy the hearing power is the same as after the complete mastoid operation. If the hearing be very poor, ossiculectomy via the external auditory canal is indicated. In cases of chronic attic suppuration, in which the hearing power is still good, the conservative mastoid operation should be performed. I agree with the President that one is sometimes surprised in cases of chronic attic suppuration at the extent of the disease found within the mastoid cavity, of which there is very little evidence before operation. But these cases are usually associated with cholesteatoma, which is a contra-indication for operating through the auditory canal. I agree with Mr. Davis that in the operation of ossiculectomy the outer attic wall should always be removed. I have recently operated on a medical man suffering from chronic attic suppuration of both ears, giving rise to headaches and a feeling of depression. On the side on which there was marked deafness I performed the radical mastoid operation. On the other side, owing to the hearing power being good, I did the complete mastoid operation, with complete removal of the outer attic wall, but carefully preserved the tympanic membrane and ossicles. The attic region was full of cholesteatomatous débris surrounding the head of the malleus, and extending backwards and filling up the mastoid cavity. To ensure complete removal of the diseased bone it was necessary to remove a portion of the tegmen tympani forming the roof of the attic. A good recovery was made, and the patient has now got almost normal hearing power, with complete eradication of the bone disease. I do not think it has been sufficiently recognized how complete an operation can be performed in cases of chronic attic suppuration without further impairing the hearing power. The chief point in the conservative mastoid operation is to remove the outer attic wall without inflicting injury on the tympanic membrane or ossicles.

Mr. J. F. O'MALLEY: I am inclined to support Mr. Cheatle's attitude on this question. I have frequently had cases of the kind, and I have put to myself the question, If I remove the ossicles am I thereby going to get rid of the whole disease? What is the condition of the mastoid, and of the antrum? That is a question I have not been able to answer. It is conceivable there may be cases of attic suppuration in which the disease is purely local—caries of the neck of the malleus, and by removing that bone one takes away the focus of the disease. And if there is not disease in the aditus, or in the antrum, or in the mastoid at the same time, the case does well and the discharge dries up. But I do not see how anybody can state before operation that the disease is confined to the attic. That is the difficulty in deciding to do ossiculectomy: when the patient submits himself to operation he expects it to be the final one.

Dr. W. HILL: I admit I do not regard ossiculectomy as an operation which has been particularly successful in my hands. I have done it in cases in which it has not been sufficient. Nevertheless on several occasions I have been particularly pleased with its results. I used to see Dr. Grant perform it fifteen years ago when Mr. Hunter Tod was embarking on the operation on what I then thought was an unjustifiable scale, though I did not consider it was unjustifiable in all the cases in which he undertook it. I consider there is a place for the operation, though in only having three cases return for the more thorough mastoid operation I think Mr. Tod must have had some luck. Sometimes I have combined a slight atticotomy with the ossiculectomy. It should not be understood that the operation is tabooed by this Section; I agree it should not be done very often, and it is difficult to know when to do it. Certainly I do not think anyone should be ostracized for defending the operation.

Dr. DUNDAS GRANT: The aurist who never does ossiculectomy is not making the most of his resources. It is absurd to say that this operation has no place at all. There are certainly cases in which the ossicles are matted together and serve no good purpose but only a bad one—namely, that of impeding the escape of discharges from the attic. One cannot always tell whether there is disease beyond the power of the *vis medicatrix naturæ* in the deeper mastoid cells. In the President's case it was obvious, from the pain on deep pressure over the walls of the mastoid process, that there was deeper disease. When that is so, it is a good reason for not being content with ossiculectomy. Some time ago I showed here a girl who had masses of cholesteatomatous material protruding from the attic, above and also behind the handle of the malleus. She had severe vertigo and headache, and when I removed the ossicles these symptoms disappeared. I also showed, some years ago, a gentleman who had been subject to attacks of what was considered to be epilepsy. He was referred to me by a neurologist, who thought the attacks might be due to a peripheral disturbance in the ear. I removed the ossicles, and since then he has been free from these attacks. There was mobility of the stapes, and the labyrinth was active, so that by means of the Siegle's speculum one could, by compression and still more by suction, induce violent forced movements. In such cases, then, the ossicles should certainly be removed.

Dr. KELSON: It has been said that if patients with a discharge from the ear, and very little else, have operation proposed they go away. Personally, I do not blame them at all. I think it is a question whether the pendulum has not now swung too far the other way. I am certain a good many of these cases can be cured by appropriate treatment without any operation. There is a sort of feeling extant now: "Discharge from the ear, operation." Looking back through a considerable number of years, one recalls a number of chronic cases which have been permanently cured by cleansing methods, such as peroxide, spirit, and so on. I think the mistake has consisted in doing too much operation. I read in a book posing as an authority that it was absurd to say that such things as spirit drops and peroxide could possibly cure chronic suppuration. I am sure that statement is absolutely untrue, and it indicates that swing of the pendulum to which I have referred.

Mr. MARK HOVELL: I support Dr. Kelson in what he has just said, that non-operative treatment is not carried out as often as it might be. In the last year or two I have had several good results from local treatment, especially from the use of collosol argentum, 2 c.c., injected subcutaneously. This drug often has a remarkable effect.

The PRESIDENT (in reply): I am glad this case has excited some interest. From the varied remarks which have been made it is obvious there are two opinions on the subject. There are those who think, with Mr. Cheatle, that ossiculectomy should practically never be performed: he says no aural surgeon performs it at the present day. There are others who think there is a place for that operation. And that brings us back to Mr. Tod's suggestion that it might be a good subject for a special debate—i.e., that those who have had experience of ossiculectomy should tabulate their experiences and results so as to give some idea of the class of case in which removal of the ossicles might be successful, and those in which it would be a failure, and the mastoid operation indicated. At present I have the feeling when I see a case of chronic attic suppuration that I do not feel I can be sure of success unless I can explore the antrum, where the infection nearly always proves to be. I would say, in answer to Mr. E. D. D. Davis, the notes of this case do not form a complete history: the patient has had ear symptoms since childhood, and I think the recent attacks of pain must have been due to sub-acute exacerbations of a chronic infection. I do not think it is advisable now to attempt to take up the time of the Section by replying to all the questions asked because the question of a special debate on this subject may receive the attention of the Council of the Section.

A Diploëtic Type of Temporal Bone and its Surgical Importance.

By Arthur H. Cheatle, F.R.C.S.

The type is characterized by the presence of a layer of diploë running through the outer antral wall, continuous with the diploë of the rest of the bone. The layer runs between the outer compact layer

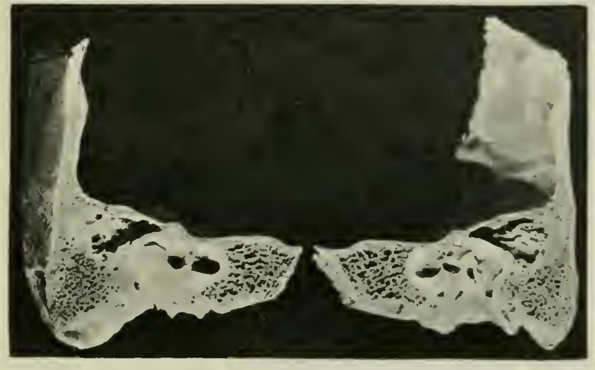

Fig. 1. (R.)

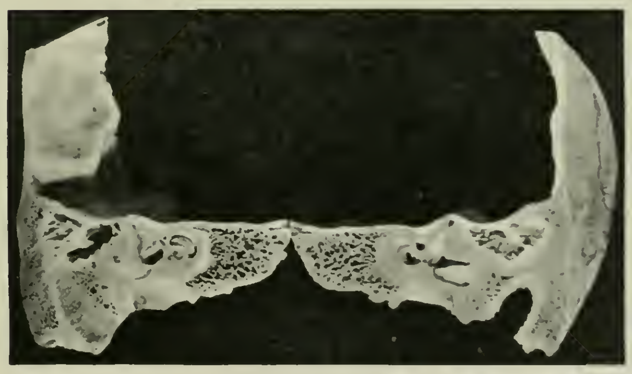

Fig. 2. (L.)

Figs. 1 and 2.—Male, aged 6½. Right and left bones. The interiors are symmetrical. A layer of diploë runs through outer antral walls and is continuous with diploë in the rest of the bone. The squamous diploë is not extensive.

and the cells which always line the inner aspect of the outer antral wall, but is separated from them by a layer of dense bone of varying thickness. The type is rare and of the bones shown from eight individuals, seven are from males. In one set in which the diploë is very conspicuous, the man was a congenital imbecile. The bones are most frequently devoid of cellular development, or a few cells may be present in the mastoid. The type is not always symmetrical. The surgical importance lies in the fact that an acute infection of the antrum is likely to lead to a rapidly spreading infection throughout the whole diploë of the bone (osteo-myelitis) with acute septicæmic symptoms and blood infection. On opening the bone in such a case, congested diploë is found, with beads of pus scattered through it, and, in order to reach healthy parts, an extensive operation is usually necessary.

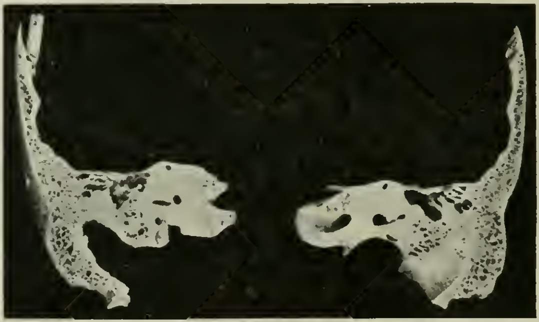

Fig. 3.

Male, aged 9. Right bone: twice sectioned. Diploë in outer antral wall continuous with that of squama and mastoid. Diploë in the squama is well developed and extends well upward.

Royal College of Surgeons Museum, 509-17.—Male, aged 6½. Both bones. The interiors are symmetrical. A layer of diploë runs through the outer antral walls and is continuous with the diploë in the rest of the bone. The squamous diploë is not extensive.

Royal College of Surgeons Museum, 126-1.—Male, aged 9. Right bone. Twice sectioned. Diploë in the outer antral wall continuous with that of the squama and mastoid. The diploë in the squama is well developed and extends well upwards.

Royal College of Surgeons Museum, 403-1.—Male, adult. Left bone. Acellular, with thin layer of diploë running through the outer antral wall.

Royal College of Surgeons Museum, 604-1.—Female, aged 28. Both bones. Acellular types. The right outer antral wall is dense. On the left side, a thin layer of diploë runs through the outer antral wall.

Royal College of Surgeons Museum, 525-1.—Male, aged 33. Both bones. Congenital imbecile. The diploë in the outer antral wall is very well marked on both sides. The elements forming the bone are marked off from one another, on the left side especially, by thin layers of dense bone.

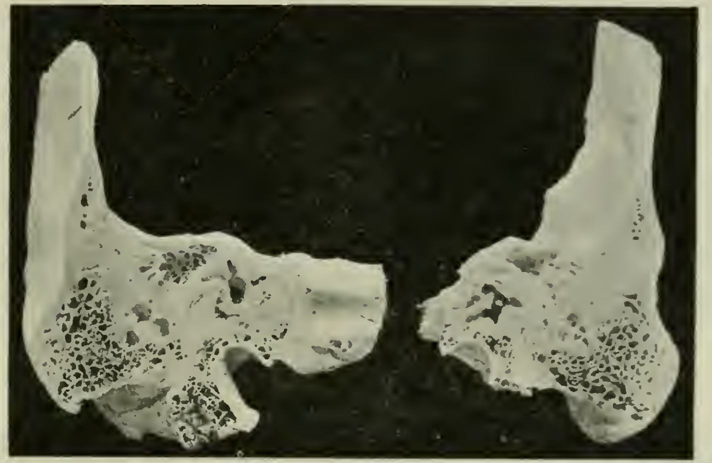

FIG. 4. (R.)

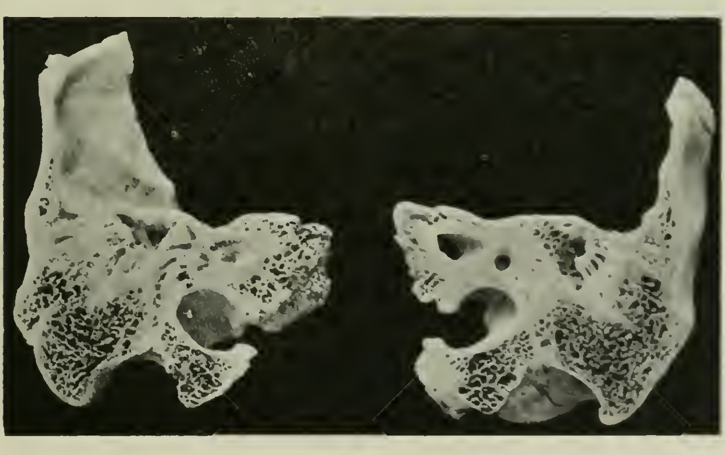

FIG. 5. (L.)

Figs. 4 and 5.—Male, aged 33. Congenital imbecile. Right and left bones. Diploë in outer antral wall very well marked on both sides. Elements forming bone marked off from one another, on left side especially, by thin layers of dense bone.

Royal College of Surgeons Museum, 296-1.—Male, aged 50. Left bone. Acellular type with diploë running through the outer antral wall.

Royal College of Surgeons Museum, 254-1.—Male, aged 41. Left bone. Acellular type with a thin layer of diploë in the outer antral wall.

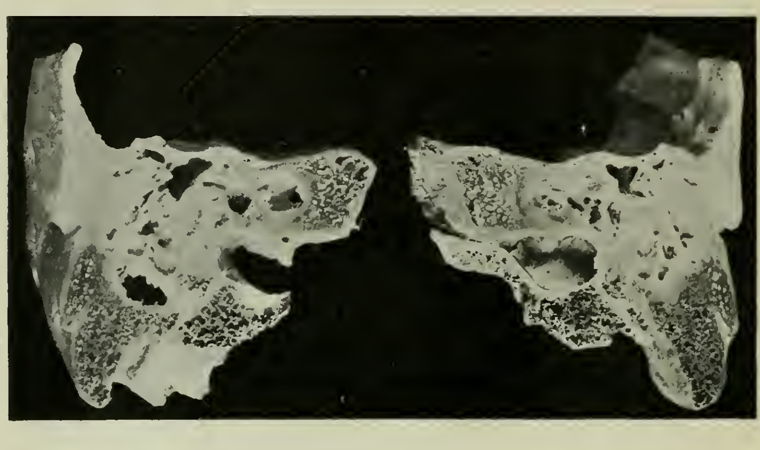

Fig. 6. (R.)

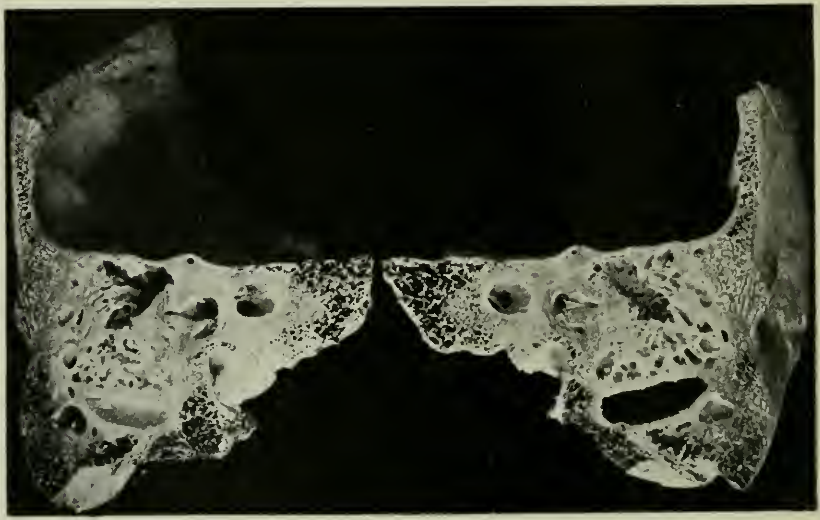

Fig. 7. (L.)

Figs. 6 and 7.—Male, aged 37. Right bone (fig. 6) outer antral wall dense. Some cells in upper mastoid. Diploëtic lower mastoid. Left bone (fig. 7), outer antral wall has track of diploë running through it. Cells in upper mastoid. Diploëtic lower mastoid. Zygomatic, squamous, and petrous elements well marked off from one another.

Case of Bezold's Mastoiditis.

By J. F. O'Malley, F.R.C.S.

Pensioner, J. H., aged 25. Admitted to Royal Herbert Hospital on February 16, 1920, with a history of discharge from right ear for two and a half years. Pain in and behind right ear and up side of head for three weeks. The pain began when he was at sea returning from Australia. Swelling in neck below right ear one week. On admission he appeared to be extremely ill. Temperature 101° F. Free discharge from right ear, very tender over right mastoid and a fluctuating swelling in the neck.

Operation, February 17, 1920: Radical mastoid; very extensive bone disease found, extending through tip of mastoid into abscess in neck. Perisinus abscess found with masses of granulation tissue on sinus wall.

His recovery has been steady and uninterrupted.

DISCUSSION.

Dr. Dundas Grant: There is a curious resemblance between the floor of the meatus in that case and in that of a little boy I have shown to-day, in whom an abscess had burrowed under the periosteum in the floor of the meatus in connexion with what may be a broken-down gland under the sternomastoid, a sort of Bezold's abscess beginning the other way round, burrowing into the ear, instead of burrowing outwards. In my case the disease is more probably a tubercular caries of the postero-inferior part of the tympanic bone.

Mr. W. Stuart-Low: I wonder whether Mr. O'Malley, in addition to the mastoid incision, incised the neck behind or in front of the sternomastoid. It seems to be a typical case of this procedure.

Mr. Colledge: I see Mr. O'Malley says the disease extended through the bone into the abscess. I have had three such cases in the last twelve months, and in none of them did there appear to be any definite communication between the tip of the mastoid process and the abscess, although there was a large abscess under the sternomastoid. I do not think there is necessarily any anatomical communication, although it is usually the case.

The President: I do not know how far the experience of members will bear out that of Mr. Colledge as to the difficulty of finding a communication, even if one exists, or whether the swelling is merely a lymphatic gland breaking down. In most of my cases I have been able to squeeze pus upwards and see it come through the mastoid fistula, although one may have been unable to find the actual communication with a small probe. French observers have stated that Bezold's mastoiditis is liable to occur in diabetics.

Mr. W. M. MOLLISON: I have always been able to find an anatomical connexion between the swelling in the neck and the mastoid; by pressing on the swelling pus can be pressed up, and this reveals the track through the bone.

Mr. O'MALLEY (in reply): Mr. Stuart-Low is right; I omitted to mention the fact that I opened the abscess behind the sternomastoid. In reply to Mr. Colledge, it was possible by using pressure on the abscess below the mastoid to bring some pus into the tip of the mastoid. All the cortex of the mastoid was destroyed, and its interior was filled with granulation tissue, the latter also covered the lateral sinus, and pus practically surrounded it. In the neck there was a large quantity of foul smelling pus. These are not now very common cases, because, as a rule, they are taken in hand early. This case was extreme from the fact that the illness started as he was returning from Australia, and as he was at sea for a long time he had no opportunity of consulting anybody about it.

Case in which a Blow on the Mastoid Process caused Partial Destruction of the Auditory Nerve with Destruction of the Vestibular Nerve.

By T. B. LAYTON, M.S.

LIEUTENANT C. C. M., R.F.A., aged 33, appeared before the Aural Board for assessment of a pension on March 9, 1920. He stated that on April 18, 1916, while in Mesopotamia, he was struck on the head at the base of the right mastoid process by a piece of shell casing which he indicated as being about 4 in. by 3 in. in size. He said that he found he was deaf in this ear when the bandages were removed ten days later, and that he has been so ever since. Later, in answer to questions, he said that he was unconscious for two or three minutes after being hit, and that then he was so giddy that he was unable to get up, and that he continued to have severe attacks of giddiness for some time but that these had stopped by the time he arrived in England in June, 1916. His Board papers show that he complained of tinnitus and of giddiness on exertion when before a Board at Poona on June 2, 1916, and another note in October, 1916, says he had been a fortnight without tinnitus.

On examination no scar can be found behind the right ear and the drumhead is intact with no evidence of any old perforation. On the left side the drumhead is normal, the air and bone-conduction are normal; on the right both air and bone-conduction show some shortening with the normal relation one to another. The sound of a fork placed on the mid-line of the head is lateralized to the left side. When the noise-machine is inserted in the left ear and set going he raises the voice distinctly, but with the right ear there is no response. Though he hears the fork in the right ear he has practically no hearing in this ear; with the noise-machine going in the left ear he does not respond to the voice. On syringing the left ear with cold water a very strong normal reaction was obtained, but though the right ear was syringed with water of the same temperature for three times as long no response was got. A turning chair was not available, but the test was imitated by making him walk round a stick on the top of which he had placed the forehead. On walking round four times to the left no reaction was got; on walking round the same number of times to the right a nystagmus to the left was observed and a walking reaction to the right.

DISCUSSION.

Dr. DUNDAS GRANT: In this case there appears to be very considerable destruction of the auditory (cochlear) portion, because when the noise machine was put on the sound side the patient raised his voice distinctly.

Mr. LAYTON (in reply): I brought up the case because it exemplifies a point which, I think, is most important in the diagnosis of these ear injuries, particularly, to my mind, in relation to functional deafness. Dr. Hurst and Dr. Peters have stated you cannot imagine an injury of the ear which has absolutely destroyed the cochlear nerve and left the vestibular nerve intact. That seems to me to be reasonable. Therefore if you find a person who is stone-deaf, or nearly so, but his vestibular reaction is normal, you should look upon the case as one of functional deafness, and give a better prognosis than if it were absolutely destroyed.

I do not think the man has any useful hearing in that ear, but the tests show that he has some hearing. The vestibular nerve, apparently, is destroyed entirely. There is a criticism to my description—which raises the question whether we have any quantitative test for the vestibular nerve, or whether we can, after testing, only say that nerve is reacting or it is not reacting. I could not get any response from this vestibular nerve, therefore I regarded it as entirely destroyed. Such a case supports the view of Dr. Hurst and Dr. Peters that if the vestibular nerve is intact, you cannot consider the auditory nerve as having an organic lesion. I think there are a large number of functional cases about, more than I thought likely before I began work at the Pensions Board.

Case of Facial Paralysis.

By J. DUNDAS GRANT, M.D.

H. B., AGED 7, has now complete left facial paralysis with soft boggy swelling on floor of meatus. There is an enlarged softened gland below the mastoid and a chain of slightly enlarged glands behind the sternomastoid. The taste for sugar is preserved on left side of tongue. Discharge from ear began five weeks ago after a week of pain in the ear. It lasted a fortnight, and on its cessation the facial paralysis developed. A week later pain returned with renewed discharge. ? Caries of postero-inferior wall of meatus.

DISCUSSION.

Mr. J. F. O'MALLEY: It seems to me there is something in the nature of an abscess near the outer part of the sternomastoid foramen, probably pressing on the facial nerve in that region. Dr. Grant said the chorda tympani is active, so the presumption is that it is not in that part of the Fallopian canal in relation to the middle ear that any trouble arises, and also the fact that hearing is good would support this view. I therefore think the trouble is due to peripheral pressure.

Dr. DUNDAS GRANT (in reply): What I propose to do is to make an incision behind the auricle, and work my way towards the soft places in the floor of the meatus, with every expectation of finding some carious focus. I think it is probably a tuberculous lesion. I will show the case again at the next meeting.

Result of Healing of Chronic Middle-ear Suppuration.

By LIONEL COLLEDGE, F.R.C.S.

PATIENT is a girl, aged 18. She had discharge from the left ear for four or five years following scarlet fever. The discharge eventually ceased with simple treatment, and the ear has been dry for nine or ten years. Inspection shows the tympanic membrane replaced by a smooth scar, all the normal landmarks being obliterated. The scar appears to be slightly superficial in position to that of the normal tympanic membrane.

I have seen one other case like it: it was in a medical man aged 35 to 40. When he was a child, he had acute middle-ear disease, and he says that eventually the doctor pulled a piece of dead bone out, and afterwards the ear healed up. He showed me his ear one day, and it has a similar appearance to this: the floor of the meatus was a smooth shining scar, with no sign of ossicles. I think it must be a rare condition.

DISCUSSION.

Dr. DUNDAS GRANT: This is a condition one tries to avoid, because such a cicatricial diaphragm leaves a space communicating with the nasopharynx and Eustachian tube and infection is likely to occur in it. It is however a thin scar and would probably soon yield if suppuration were to take place behind it, so that its removal is not very imperative. In the early days of the Otological Society I showed a case in which a similar condition had resulted after one of my operations, and I removed the diaphragm, preferring to have an open space.

Mr. LAWSON WHALE: After the radical mastoid operation this kind of case is not uncommon: therefore can any member suggest a reason for its happening? Why should it happen in some cases, and not in others?

Dr. KELSON: I think this can hardly be described as replacing the membrane: there may be a cicatricial membrana tympani on the inside of that which we see. It is too external: there is a considerable space between the proper position of the membrana tympani and this scar.

Section of Otology.

President—Mr. HERBERT TILLEY, F.R.C.S.

Acute Suppurative Inflammation of Right Mastoid infected from Chronic Right Maxillary Antral Empyema; Immediate Closure of Mastoid Wound after Application of B.I.P.[1]

By HERBERT TILLEY, F.R.C.S. (President).

MRS. G., aged 42, consulted me on March 26, 1920, for a "bad cold in the nose for a long time," and chiefly noticed on the right side. Examination proved the right antrum to be full of foul pus. She was referred to her dentist because of subacute inflammation in the corresponding first molar. This was removed and pus issued from the socket, but in spite of daily irrigation of the antrum, the discharge continued. During this treatment and about a fortnight after her visit to me, the right middle ear became inflamed and the drum membrane perforated. Rest and frequent cleansing of the meatus failed to check the aural discharge or to relieve pain which, in the course of the following ten days or fortnight, settled in the mastoid and parietal region.

May 1: I opened the right mastoid antrum and cells in the usual way. They were infiltrated with pus. The dura mater was exposed in the roof of the antrum and the lateral sinus in the lower part of the mastoid cell region. The parts were thoroughly cleansed, smeared with B.I.P. and the skin wound sutured in its entire length.

Immediate union has taken place and the hearing is improving every day. It is barely three weeks since the operation. Last week (May 17) I opened the right maxillary antrum by the intranasal method.

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[1] At a meeting of the Section, held May 21, 1920.

Note.—On May 5, i.e., four days after the operation on Mrs. G., her child, aged 6, was sent into the same nursing home with an acute suppurative inflammation of the left mastoid. This child has been operated on in the same way as the mother and is proceeding equally well.

I show the case not because it illustrates anything new in principle: it is the old blood-clot method, in which you have an additional means of avoiding sepsis by the use of a certain type of antiseptic (B.I.P.). It is a great saving of time and expense when you can discharge patients from the home in a fortnight to three weeks, where formerly they were laid up for some six weeks or more after the operation. I shall be glad to hear any criticisms of this case, or of the method which has been used in connexion with it.

DISCUSSION.

Mr. E. M. WOODMAN: The President's result is most successful. . I owe it to his series of cases which he brought forward a year ago that I have had quite a number of exceedingly satisfactory cases. All my hospital cases now go out on the eighth or tenth day, and my private cases are discharged in a fortnight. It seems that the more dirty looking the wounds are the better the cases do. I sew up completely, and there is very rarely any subsequent discharge of serum. There is one type of case, more or less obvious, in which one would not advise the method, and that is where there is an infection of the lateral sinus, not necessarily accompanied by thrombosis.

Sir CHARLES BALLANCE: I think the result in this case is so excellent that it could not have been better. I do not think anybody has ever had a better result from a very bad acute mastoid. Both the lateral sinus and the dura over the temporo-sphenoidal lobe were exposed. To get healing in a fortnight in such a case is a great triumph of surgery. I was much interested, some years ago, in the blood-clot method. I had been visiting Baltimore, and Halsted was the first who wrote about it in the treatment of acute osteomyelitis. I saw some cases in New York, and elsewhere. I had some successes with this method, but I also had one great disaster, which upset me very much. It was that of a child aged 12, who had been ill thirteen days, and was very ill when I saw her. I sewed up the wound, and I have regretted it ever since, though I believe the child had meningitis when I operated. Perhaps I ought to have suspected the meningitis, but even if I had, I should have hoped to save life by operating. She died forty-eight hours afterwards. Except in very septic cases, I cannot imagine the method which the President has brought before us could ever be a bad one. Therefore I am glad that I have seen this case, which is a splendid example of how, even in a serious septic illness, the septic process may be arrested at once.

Tumours of the Eighth Nerve.[1]

By J. S. Fraser, F.R.C.S.E.

In view of the recent publication by Professor Harvey Cushing of his interesting and elaborate work on "Tumours of the Nervus Acusticus," it may appear a work of supererogation to investigate this subject again. In justification of the present contribution, however, it may be pointed out that Cushing has approached the subject from the viewpoint of the general surgeon rather than that of the otologist. In the vast majority of, if not in all, cases the growth starts in the internal auditory meatus and only later invades the cranial cavity, and Cushing states that we have still a great deal to learn about the early diagnosis. He admits that at the stage at which his patients came under observation he was unable in most cases to remove the tumour entire.

The researches of Continental otologists, and especially those of Bárány, have thrown considerable light on the method of diagnosing tumours of the eighth nerve in the "early or otological" as opposed to the "late, neurological or general surgical" stage. In spite of this the great majority of otological text-books in the English language contain nothing about the symptoms, diagnosis and treatment of acusticus tumours. Cushing himself points out that in the past responsibility for the diagnosis and treatment of tumours of the eighth nerve has been shared by the neurologist, the ophthalmologist, the otologist, and the general surgeon. Surgical failures were so frequent that utter discouragement prevailed. During the past fifteen years, according to Cushing, the subject has advanced from the diffuse presentation of tumours of the posterior cranial fossa to the clinical differentiation of intra-cerebellar from extra-cerebellar growths, and particularly of those of the cerebello-pontine angle, from all others. Finally, in the group of cerebello-pontine tumours, those originating in the eighth nerve have come to stand out with comparative clearness from those which secondarily involve this and other nerves through pressure. Owing to the characteristic chronology of their symptoms, acoustic tumours may, as a rule, be sharply distinguished from all others.

The present writer hopes that in the future otologists may be able to diagnose these cases in the *early* stage, when operative treatment holds out a much better chance of recovery than exists at the later periods when, according to Cushing, all we can effect is an extensive decompression operation combined with removal of "as much as possible" of the growth.

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[1] The microscopic work for this paper was carried out at the laboratory of the Royal College of Physicians, Edinburgh.

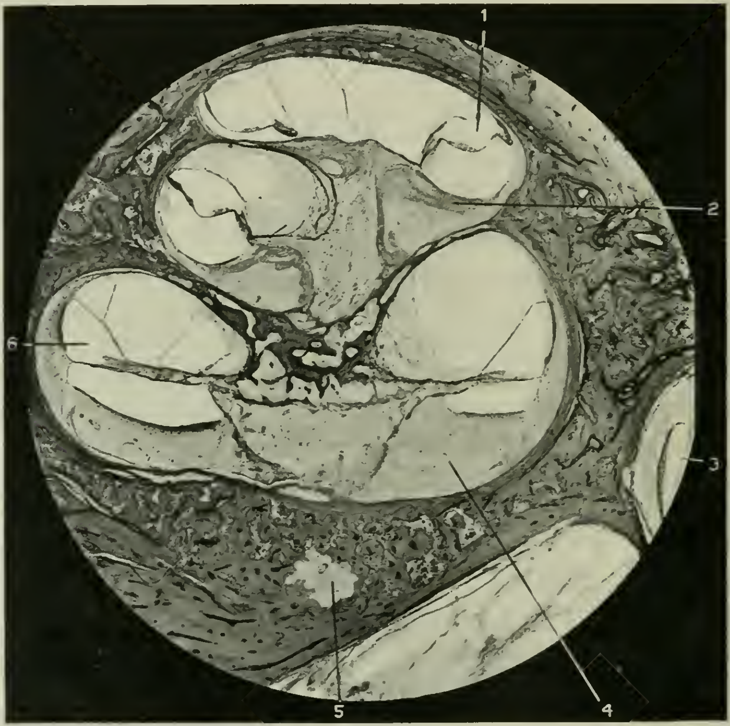

Fig. 1 (Case I).

M. McQ., female, aged 75. Tumour of right nervus acusticus. Horizontal section of *right* ear, No. 170, × 17 diam. Axial section through cochlea and internal meatus. 1, scala media of upper part of middle coil, showing Corti's organ; 2, new bone formation in scala tympani; 3, saccule; 4, new connective or tumour tissue in scala tympani of basal coil; 5, erosion of cartilage capsule of cochlea by tumour tissue; 6, cochlear canal of lower part of basal coil, showing absence of Corti's organ and degeneration of spiral ligament.

Before referring to the literature of eighth-nerve tumours the writer wishes to place on record three original cases in which he has been able to examine the ears microscopically by the celloidin method.

Case I.—M. McQ., female, aged 75, was admitted to a poorhouse hospital on April 30, 1909, complaining of giddiness and deafness of five years' duration. (Unfortunately the clinical notes are very incomplete.) The patient also complained of noises in the *left* ear. On admission it was found that she could not walk without assistance on account of giddiness. Rombergism marked. No note as to nystagmus. Knee-jerks exaggerated. Babinski's sign absent. Pupils react to light and accommodation. Patient is almost completely deaf and has well-marked arteriosclerosis.

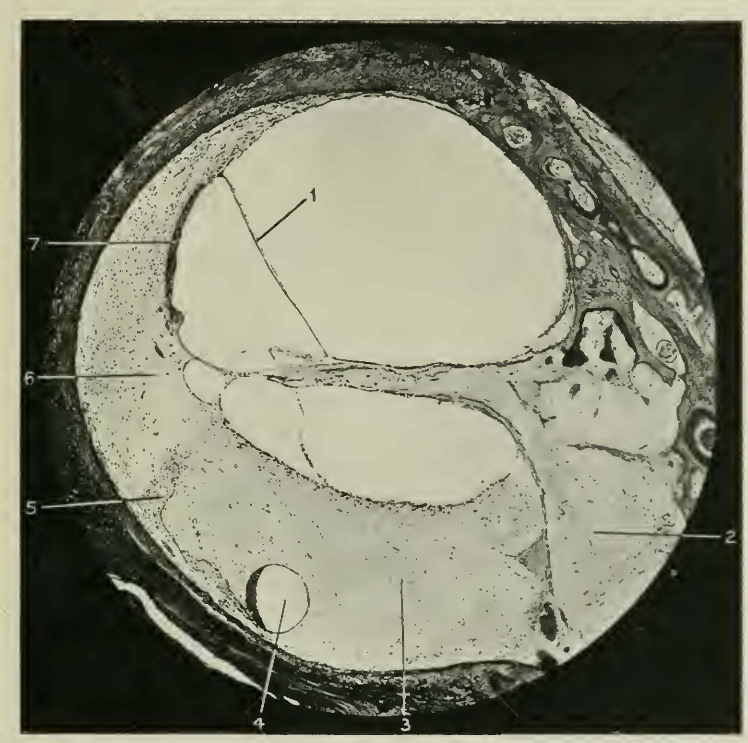

FIG. 2 (Case I).

Horizontal section through basal coil of cochlea (right ear), No. 205, × 48 diam. 1, Reissner's membrane; 2, spiral canal with marked atrophy of ganglion cells; infiltration by delicate connective tissue; 3, delicate connective or tumour tissue in scala tympani; 4, large vein; 5, new-formed bone; 6, myxomatous or dropsical degeneration of spiral ligament; 7, stria vascularis: note absence of Corti's organ.

Functional Examination (J. S. F.).—C_{32} and C_{64} not heard by either ear. C_{128}, C_{256}, C_{512}, and C_{1024} are all heard faintly by the right ear (side of tumour) but not by the left. C_{2048} not heard by either ear. Schwabach shortened. Galton's whistle not heard at all. Vestibular apparatus not tested (1909). *Progress.*—December 15, 1909: Giddiness improved; patient can walk without help; deafness as before. December 17, 1909: Vision very bad; patient still going about. October 12, 1910: Giddiness worse; patient fell and cut her forehead over the right eye. January 9, 1911: Patient better and able to go about by herself. June 9, 1911: Very feeble; complains of indefinite headache.

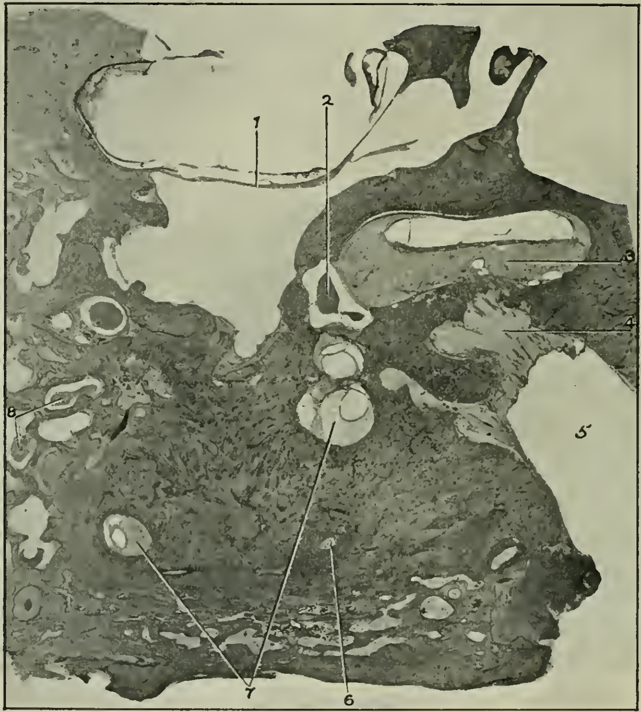

FIG. 3 (Case I).

Horizontal section of right ear, No. 310, × 6 diam. 1, tympanic membrane; 2, serous exudate in niche of round window; 3, connective or tumour tissue in scala tympani of basal coil; 4, cochlear nerve in fundus of internal meatus; 5, site of tumour which has been removed. In the right hand bottom corner a piece of the tumour tissue still remains; 6, aqueduct of cochlea and accompanying vein; 7, two ends of posterior canal (note that the perilymph space at the smooth end is almost filled by connective tissue); 8, serous exudate in air cells.

September 8, 1911: Patient fell again and cut her forehead over the left eye. January 8, 1912: Headache as before; patient completely deaf and bedridden. March 10, 1912: Very restless and noisy at night. October 20, 1912: Sudden paralysis of right arm, leg, and face; patient unconscious. She died four days later without regaining consciousness. Post-mortem showed a tumour the size of a cherry involving the eighth nerve on the *right* side. Bone eroded in cranial end of right internal meatus. Gelatinous fluid present in air cells of mastoid on both sides.

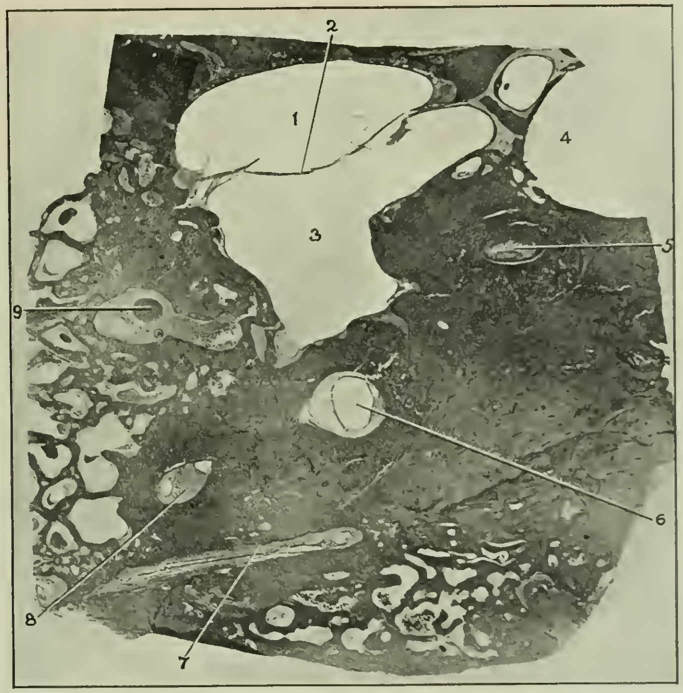

FIG. 4 (Case I).

Horizontal section of right ear, No. 385, × 6 diam. 1, external meatus; 2, tympanic membrane; 3, tympanic cavity; 4, carotid canal; 5, spiral ligament of basal coil of cochlea; 6, ampullary end of posterior canal; 7, aqueduct of vestibule; 8, smooth end of posterior canal, showing perilymph space filled with new-formed tissue; 9, facial nerve.

Microscopic Examination of Right Ear (side of acusticus tumour).—*Middle Ear:* The tympanic membrane is practically normal; the tears in it are artefacts. Malleus, incus and stapes are healthy. The

Eustachian tube and the ossicular joints and muscles are normal. The mucosa of the tympanic cavity and antrum is slightly thickened (fig. 3). There is coagulated fluid in the cells on the inner wall of the antrum and exudate in the border cells between the antrum and the external meatus. The oval window is normal, but the niche of the round window contains some mucoid exudate. The secondary tympanic membrane is thickened as, on its cochlear side, it is incorporated in the newly-formed tissue which fills up the basal coil of the cochlea (fig. 3).

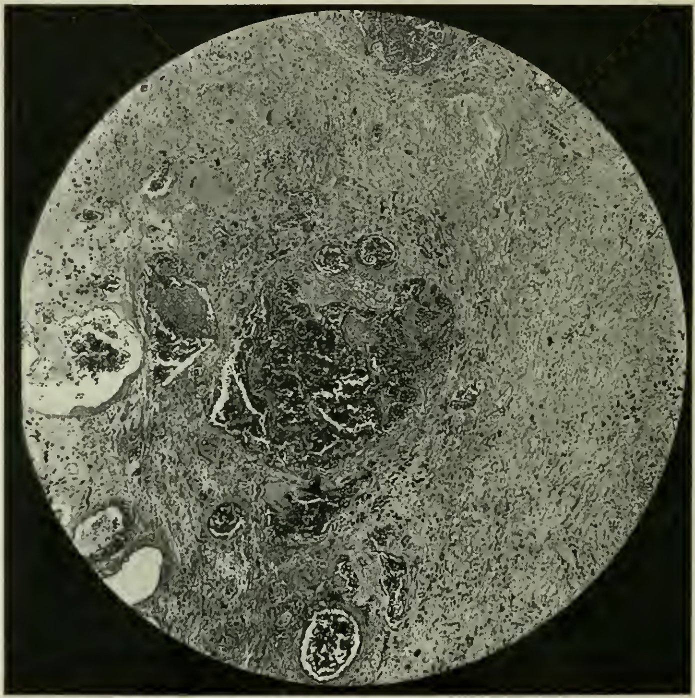

FIG. 5 (Case I).

Section of tumour, × 50 diam. Tumour appears to be about 5 cm. in diam., and is composed of very fibrous cellular structure. In the central portion there are numerous large blood spaces of an angiomatous character, filled with blood. In the stroma surrounding these spaces there are numerous phagocytic cells containing blood pigment.

Cochlea.—Delicate connective (?) tissue, very like tumour tissue, is seen in the scala tympani of the cochlea (fig. 1). There are large vessels in this tissue. The modiolus and spinal canal are infiltrated with the new connective or neuroglia (?) tissue. There are still, however, some fairly well-formed fibres of the cochlear nerve entering the modiolus. In the lower part of the basal coil Corti's organ has quite disappeared, and there is some myxomatous degeneration of the spiral ligament (fig. 2). (*Note.*—The patient was aged 78, and suffered from arteriosclerosis.) The stria vascularis is fairly well formed. In the upper part of the basal coil Corti's organ is recognizable, and there are still some ganglion cells left in the spiral ganglion. (The writer does not attempt to describe the condition of the hair cells of Corti's organ in any of his cases as he does not believe that, with the method employed, such descriptions have any value.) There is some new bone formation in the middle coil in the scala tympani and well-marked new bone formation in the basal coil just in the region of the round window. The cochlear opening of the perilymphatic aqueduct is free from tumour (?) tissue, but the aqueduct is blocked in its middle part.

Vestibule.—The neuro-epithelium of the utricle and saccule is well formed and the otolith membrane is present. There does not appear to have been any secondary degeneration of the neuro-epithelium of the saccule and utricle as a result of the atrophy of the vestibular nerve (fig. 1). There is no dilatation of the endolymphatic structures. Curdled lymph is present in the perilymph space of the vestibule and in the aqueduct near the saccus.

Canals.—Superior : The crista of the superior canal is practically flat, and there is pigment in the position of the epithelium. The atrophy of the crista of the superior canal is remarkable when compared with the well-formed cristæ of the lateral and posterior canals. The smooth end of the superior canal is quite healthy. *Posterior :* There is a little new connective or tumour tissue in the perilymph space at the smooth end of the posterior canal, just beyond the crus commune. The perilymph space is almost filled up with connective tissue towards the convexity. There is also a little new bone formation in the posterior canal in the perilymph space. The crista is well formed, but pigment is again present. The crus commune is healthy. The crista of the *lateral* canal is well formed and the cupula is present, but there is pigment in the epithelium at the base of the crista. Here again some new connective tissue formation is seen in the perilymph space of the lateral canal towards its convexity. There is some swelling and irregularity of the membranous tube in the lateral canal (figs. 3 and 4).

The Internal Meatus and Nerves.—The internal meatus is considerably dilated and its posterior margin is eroded by tumour tissue

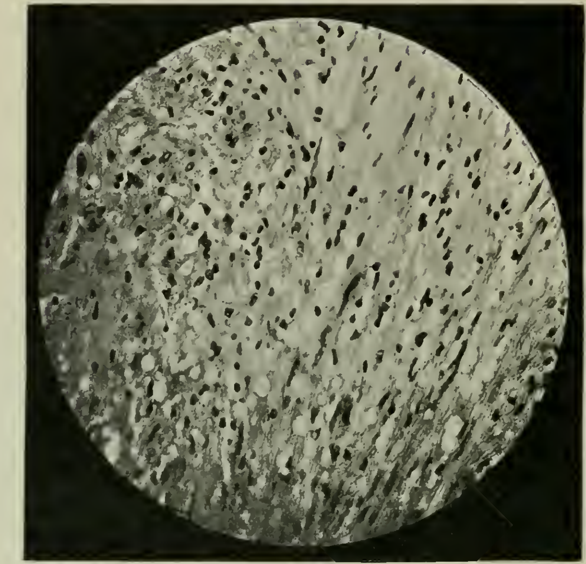

FIG. 6 (Case I).

Section of tumour, × 200 diam. Bundles of fibrous (?) tissue are seen, with well-formed blood-vessels; other parts have a more cellular, myxomatous character, and resemble glial tissue.

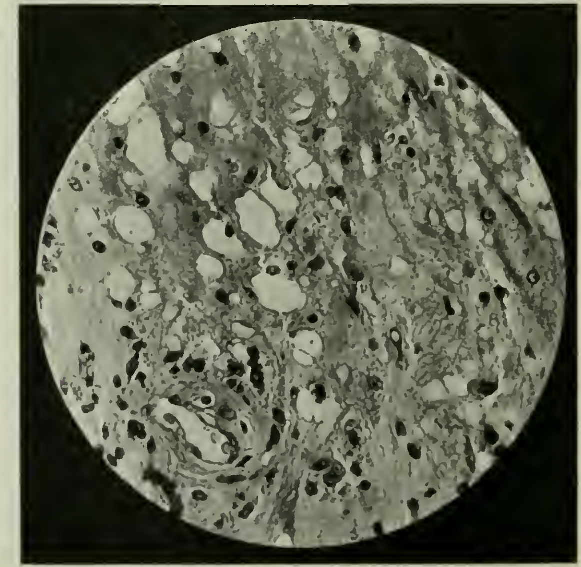

FIG. 7 (Case I).

Section of tumour, × 300 diam.

(fig. 3). The tumour did not reach to the fundus. The cochlear branch can only be recognized at the fundus. The nerve to the ampulla of the posterior canal is fairly well preserved, but there is marked atrophy of other parts of the vestibular nerve in the depth of the internal meatus. (*Note.*—Most of the tumour was removed at the post-mortem for examination by the paraffin method.) The geniculate ganglion is healthy and the facial nerve is well formed.

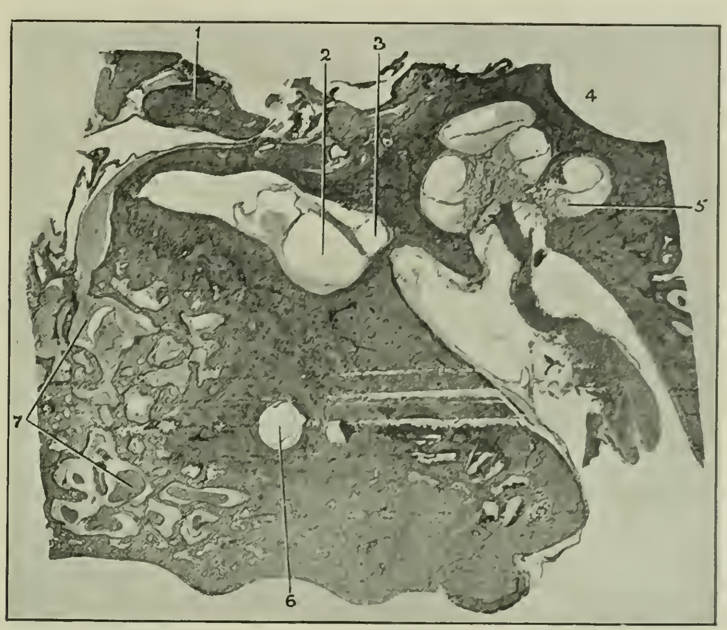

FIG. 8 (Case I).

Horizontal section of *left* ear, No. 80, × 6 diam. 1, incus; 2, utricle; 3, saccule; 4, carotid canal; 5, basal coil of cochlea with new connective tissue and bone formation; 6, smooth end of superior canal; 7, exudate in air cells behind labyrinth.

Tumour.—The tumour, which was cut and stained separately, shows large blood spaces. The main bulk of the tumour is composed of gliomatous or myxomatous tissue (figs. 5, 6 and 7).

Microscopic Examination of the Left Ear.—Middle Ear: The malleus is absent (artefact); the incus and stapes are normal. There is some new bone formation in the anterior part of the attic and the mucosa here is thickened. There is a little hæmorrhage between the numerous folds of mucosa in the tympanic cavity. Serous exudate is present in the cells on the inner wall of the antrum. The vessels of the fossa subarcuata are dilated. The oval window is normal. The membrane of the round window is normal on its tympanic but thickened on its cochlear side (fig. 12).

The labyrinth capsule is normal and there is no sign of a fistula in the lateral canal.

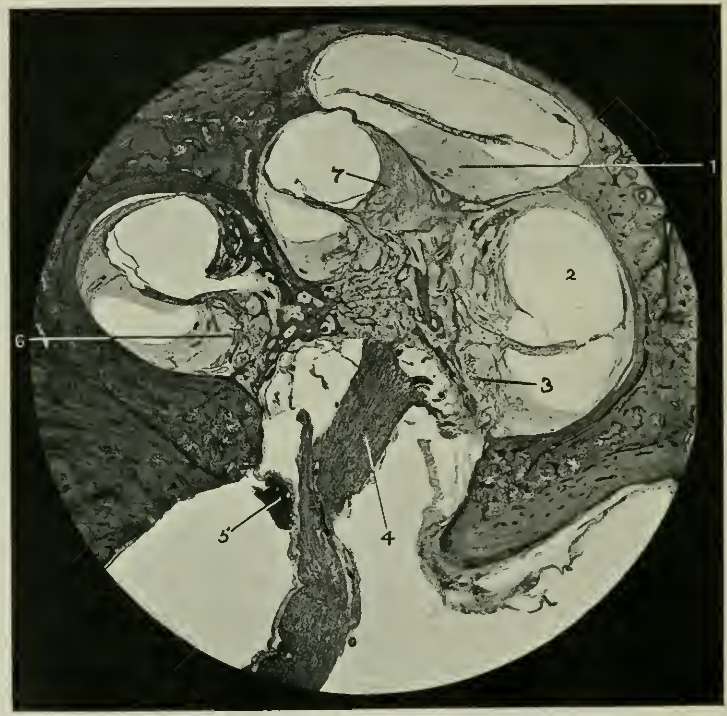

FIG. 9 (Case I).

Horizontal section of left ear, No. 80, × 17 diam. 1, new connective tissue in scala tympani of apical coil; 2, greatly dilated cochlear canal of upper part of middle coil: note new connective tissue and bone in scala vestibuli; 3, spiral ganglion cells still present; 4, cochlear nerve; 5, hæmorrhage between dura mater and bone; 6, scala tympani of lower part of basal coil with new connective tissue and bone formation; 7, scala vestibuli of middle coil filled with new bone formation.

Cochlea.—The basal coil of the cochlea in the region of the round window contains a considerable quantity of new bone and connective tissue (?) formation, but the cochlear opening of the perilymphatic aqueduct appears normal. The middle part of the aqueduct seems to be obliterated by new bone formation, but towards the cranial end the aqueduct is again patent. Corti's organ is present in all coils, even in the lower part of the basal coil. The scala vestibuli also shows new connective tissue (?) and bone formation. There is only slight atrophy of the spiral ganglion cells (figs. 8 and 9).

Vestibule.—The neuro-epithelium of the utricle and saccule is quite normal, as is also the aqueduct of the vestibule. The vestibular nerve, however, appears rather atrophic.

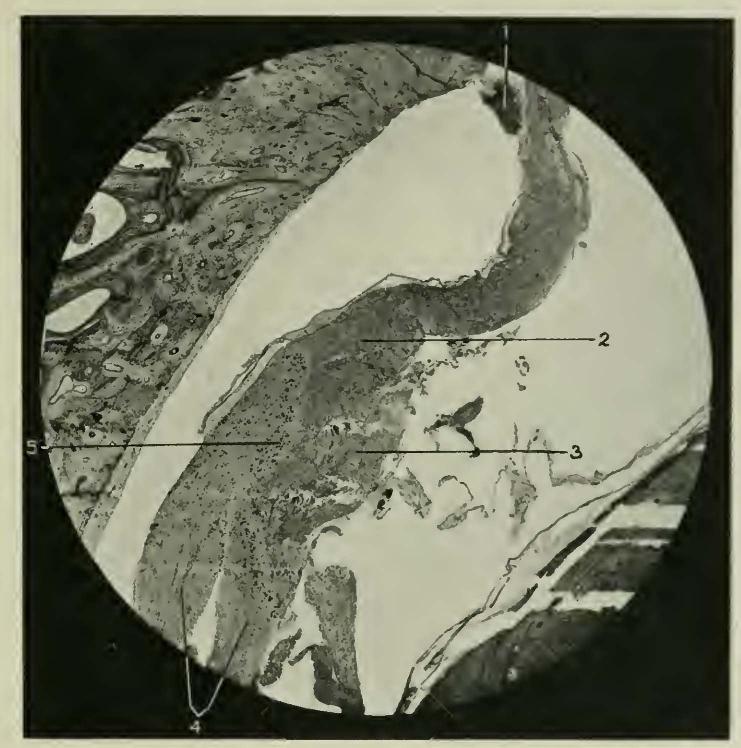

FIG. 10 (Case I).

Horizontal section through left ear, No. 80, × 17 diam., shows two parts of the eighth nerve in the internal meatus; 1, hæmorrhage; 2, distal or medullated portion of cochlear nerve; 3, distal part of vestibular nerve; 4, central non-medullated portion of eighth nerve; 5, zone of corpora amylacea.

Canals.—There is a great amount of pigment present in the crista of the superior canal. The cristæ of the lateral and posterior canals are normal. There is some new connective tissue and bone formation in the convexity of the lateral canal and the opening into the vestibule is practically filled up by new bone (figs. 11 and 12).

Internal Meatus and Nerves.—There is a little hæmorrhage in the internal meatus in the anterior part between the dura and the bone. The cochlear nerve, as is usual in old people, shows well marked corpora amylacea (fig. 10) but is otherwise normal. The vestibular nerve is very atrophic.

Remarks.—The atrophy of Corti's organ in the basal coil on the right side may possibly have been due to senile or arteriosclerotic change.

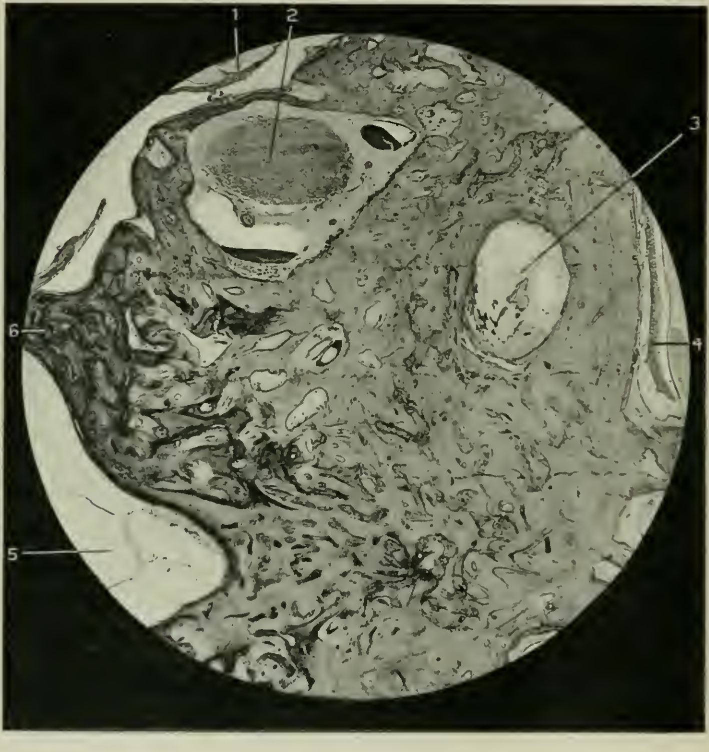

FIG. 11 (Case I).

Horizontal section through left ear, No. 185, × 17 diam. 1, thickened fold of tympanic mucosa; 2, facial nerve; 3, convexity of lateral canal, showing new connective tissue and bone formation; 4, thickened mucosa of antrum with exudate; 5, utricle; 6, posterior margin of oval window.

The interesting thing in this case is the presence on both sides of what appears to have been an old labyrinthitis (formation of new connective tissue and bone within the hollow space of the labyrinth) with very little evidence of middle-ear suppuration. It is true that there is exudate in the mastoid air cells, and that the folds of mucosa in the tympanum are very numerous. (1) The case might possibly be explained by an old middle-ear suppuration which has invaded the cochlea through the round window and resulted in the formation of new connective tissue and bone in the scala tympani, with infection through the helicotrema of the scala vestibuli. It is possible, too, that the lateral canal may have been invaded in the aditus with filling up of the convexity of the canal by new bone and connective tissue, while the oval window remained sound. This theory would account for the superior and posterior canals being normal and the lateral canal being filled up with new bone. As against this view the otitis media appears to have been of a mild variety. The ossicles show no necrosis and the drumhead no chalk patches or scars. In one of Henschen's cases the middle ear showed the same changes as in the present case. Alexander found a leucocytic exudate in the scala tympani in one case, and Sternberg has noted plasma cells in the tumour itself. (2) It is possible, on the other

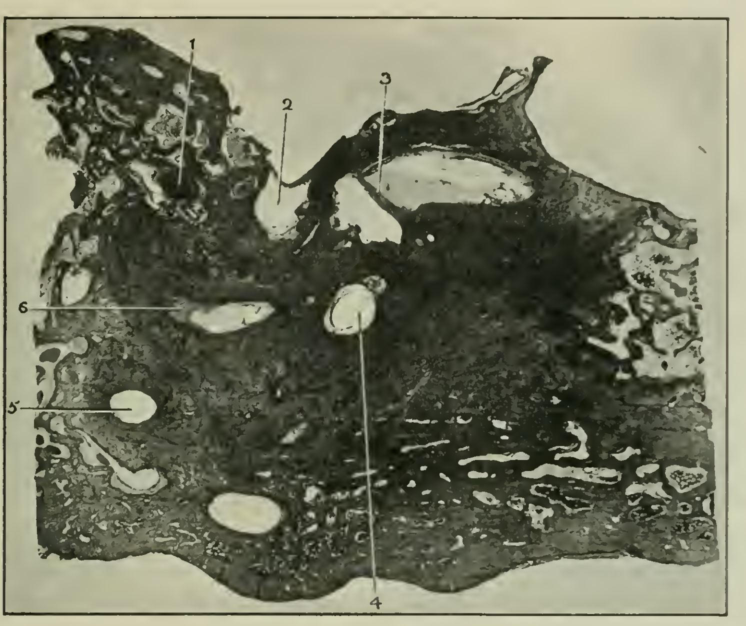

FIG. 12 (Case I).

Horizontal section of left ear, No. 265, × 6 diam. 1, facial nerve; 2, sinus tympani apparently closed by double fold of mucosa; 3, thickened membrane of round window. The scala tympani contains new connective tissue; 4, ampullary end of posterior canal; 5, smooth end; 6, new bone formation in smooth end of lateral canal.

hand, that the labyrinth has been infected from the meninges, as has been suggested by Siebenmann and Nager—i.e., that the case was an old one of meningitic labyrinthitis. (3) Lastly, the presence of a tumour of the eighth nerve on the right side suggests that the condition of the inner ears may be due to an end stage of "choked labyrinth." When the writer saw the patient in 1909 (three years before death) she could hear a little with the right ear (side of the tumour) but not at all with the left ear. This fact does not support the view that the changes in the two labyrinths were the result of the tumour in the right eighth nerve, although, on other grounds, this appears to be the most probable explanation. Giddiness and deafness had been present for eight years before death, and at the post-mortem the acusticus tumour was a comparatively small one. (4) It is of course possible that the conjunction of bilateral otitis media and spontaneous cure of labyrinthitis (if it was labyrinthitis) with tumour of the right eighth nerve was an accident—i.e., that the conditions were not related to one another. In view of the appearances seen in the other two cases this appears unlikely. From the findings in this case it appears to be advisable in cases of solitary acusticus tumour to examine both inner ears microscopically.

Case II.—A. T., female, age 25, was an inmate of a hospital for incurables. Clinical details of the case are almost entirely wanting. The diagnosis was only made at the post-mortem. The patient's face was very expressionless, and she was mentally dull and bedridden for many months before death. She was blind and had double optic neuritis. During the last few days she was very intolerant of touch. Coma supervened before death.

Post-mortem Examination.—Outer (cranial) surface of dura, over almost its entire extent, was covered with small pedunculated tumours, which varied in size from a hay seed to a split pea (fig. 20). On removal of the dura the inner surface of the cranial bones appeared as if deeply pitted by small-pox (fig. 19). In the region of the right internal auditory meatus and angle between pons and cerebellum there was a tumour mass about the size of a walnut, which pressed the pons, medulla and cerebellum towards the left side.

Microscopical Examination of Right Ear. — *Middle ear*, with ossicles, joints and muscles, normal. The membrane of the round window is thickened on its inner or cochlear aspect, as in the previous case.

Labyrinth Capsule.—The tumour is eroding the posterior surface of the petrous bone in the region of the crus commune, and has penetrated posteriorly between the cartilage and lamellar bone (fig. 13). The tumour does not reach as far outwards as the carotid canal.

Cochlea.—The modiolus is not eroded by the tumour, but the spiral ganglion in the lower part of the basal coil is infiltrated with tumour-tissue and the ganglion cells have disappeared (fig. 16). The scala tympani of the cochlea is occupied by tissue resembling that of the tumour, but there is very little myxomatous or tumour tissue in the scala vestibuli. Internal to the round window membrane the scala tympani is full of what appears to be tumour (?) tissue in which there is some new bone formation (fig. 17). The cochlear opening of the perilymphatic aqueduct is filled with this tissue, which also occupies the aqueduct in its inward and downward course. The cranial end of the perilymphatic aqueduct of the cochlea is not in the

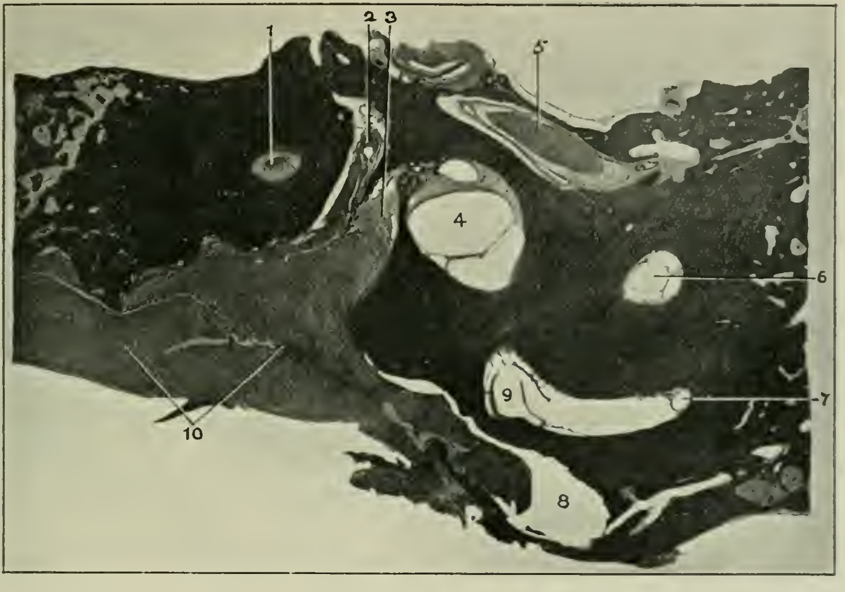

Fig. 13 (Case II).

A. T., female, aged 25. Tumour of eighth nerve. Horizontal section of right ear, No. 5, × 6 diam. 1, upper part of basal coil of cochlea; 2, facial nerve: the tumour tissue is infiltrating as far as the geniculate ganglion; 3, tumour infiltrating vestibular nerve to utricle; 4, utricle; 5, facial nerve; 6, convexity of lateral canal; 7, smooth end of posterior canal; 8, erosion of bone by tumour; 9, crus commune; 10, tumour.

sections, but as far as they extend the perilympathic aqueduct is blocked by connective or tumour tissue. There is some hæmorrhage in the spiral ligament of the middle coil, and also in the scala tympani of the basal coil. Corti's organ is unrecognizable in the lower part of the basal coil (fig. 16), though the patient was only 25 years old. In the middle and apical coils of the cochlea Corti's organ is fairly well formed. The region of the helicotrema contains granular and fibrinous exudate.

Vestibule.—The neuro-epithelium of the saccule and utricle is normal and the otolith membrane is present. The saccule contains curdled lymph. There is also some curdled lymph—fibrinous exudate—in the vestibule on the inner surface of the footplate of the stapes. The vestibular aqueduct appears normal at its vestibular end. Towards its cranial end the ductus and saccus are in contact with the tumour but are not invaded.

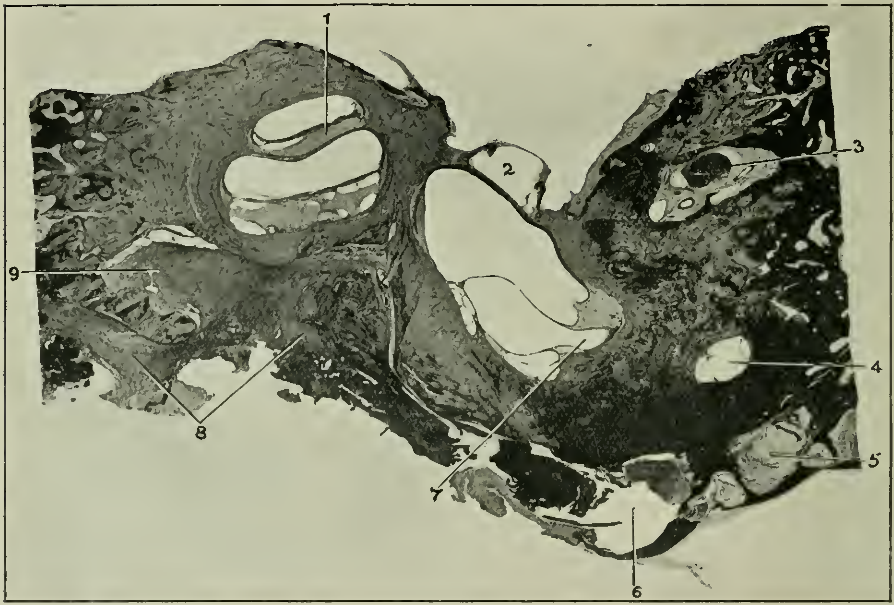

Fig. 14 (Case II).

Horizontal section of right ear, No. 51, × 6 diam. (iron hæmatoxylin staining). 1, new connective tissue and bone in scala tympani of middle coil; 2, hollow of stapes; 3, facial nerve; 4, convexity of posterior canal; 5 and 6, tumour eroding bone internal to labyrinth; 7, junction of membranous lateral canal with utricle; 8, internal meatus greatly dilated by tumour; 9, tumour eroding bone anterior to cochlea.

Canals.—The cristæ of the canals are practically normal. There is some hæmorrhage in the perilymph space at the smooth end of the posterior canal and also in the lateral canal.

Internal Meatus and Nerves.—The internal meatus is greatly dilated by the tumour, especially at its inner end (figs. 14 and 15). The growth consists of interlacing fibres, and contains large vascular spaces. Some areas are more cellular than others. No giant cells are present, although the bone has been distinctly eroded. There is a small process of the tumour extending along the seventh nerve, almost as far as the geniculate ganglion (fig. 13). In its descending portion the facial nerve is normal.

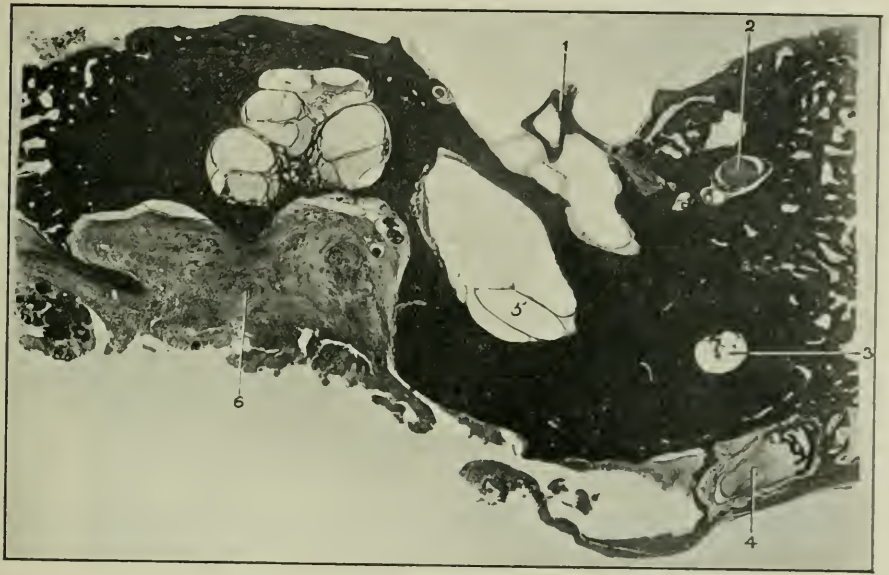

FIG. 15 (Case II).

Horizontal section of right ear, No. 90, × 6 diam. 1, head of stapes with stapedius; 2, facial nerve; 3, posterior canal with hæmorrhage in perilymph space; 4, tumour; 5, lower part of utricle; 6, dilated internal meatus with tumour.

Remarks.—There is no appearance in this case of any otitis media. The tympanic mucous membrane is quite normal and so are the tympanic surfaces of the oval and round windows. The vestibule and canals are quite free from tumour growth, but the cochlea shows marked affection, especially of the scala tympani in the region of the round window. The new tissue in the cochlea is, however, more delicate than the tumour in the internal meatus. There has not been any gross bursting through of the tumour in the internal meatus into the cochlea, but infiltration may have occurred through the porous layer of bone separating the internal meatus from the scala tympani. It is interesting to note in this case the fact that the aqueduct of the cochlea is occupied by delicate connective tissue, apparently the same as that in the scala tympani. The neuro-epithelium of the vestibule and canals was well preserved, but Corti's organ had disappeared in the basal coil. As this patient was only 25 years old, senile or arteriosclerotic deafness can be excluded.

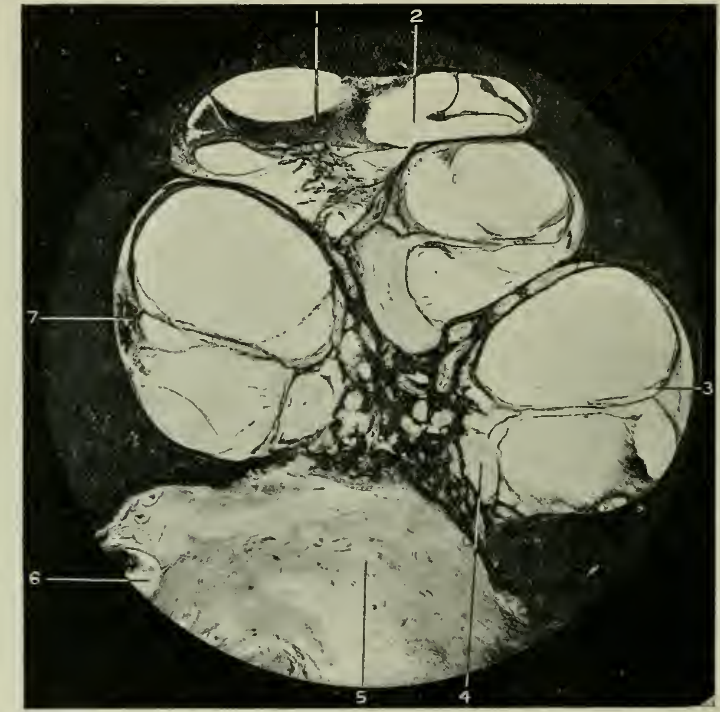

Fig. 16 (Case II).

Horizontal section of right ear, No. 90, × 20 diam. Axial section through cochlea; 1, granular and fibrinous exudate; 2, helicotrema; 3, lower part of basal coil of cochlear canal (Corti's organ has almost disappeared); 4, spiral canal. The ganglion cells are absent and the space is occupied by delicate connective tissue similar to that of the tumour; 5, fundus of internal meatus occupied by tumour; 6, large, thin-walled blood-vessel; 7, hæmorrhage in spiral ligament of upper part of basal coil.

The condition of the dura mater and of the cerebral surface of the cranial bones was very interesting. At numerous spots the brain and pia-arachnoid were adherent to the dura, and on the outer or bony surface of the dura small purplish projections, varying in size from a hay seed to a split pea, were observed. These projections fitted into small pits in the cranial bones (figs. 19 and 20). Cushing and others have noted similar appearances. Cushing holds that the swellings are minor cortical protrusions, which occur at the situation of the arachnoid villi. In 1903 Frankl and Hunt called attention to multiple punctate depressions over the temporal and parietal lobes. This may occur whenever arachnoid villi are to be found, as these structures represent the point of exit for the cerebro-spinal fluid from the subarachnoid spaces. The villi

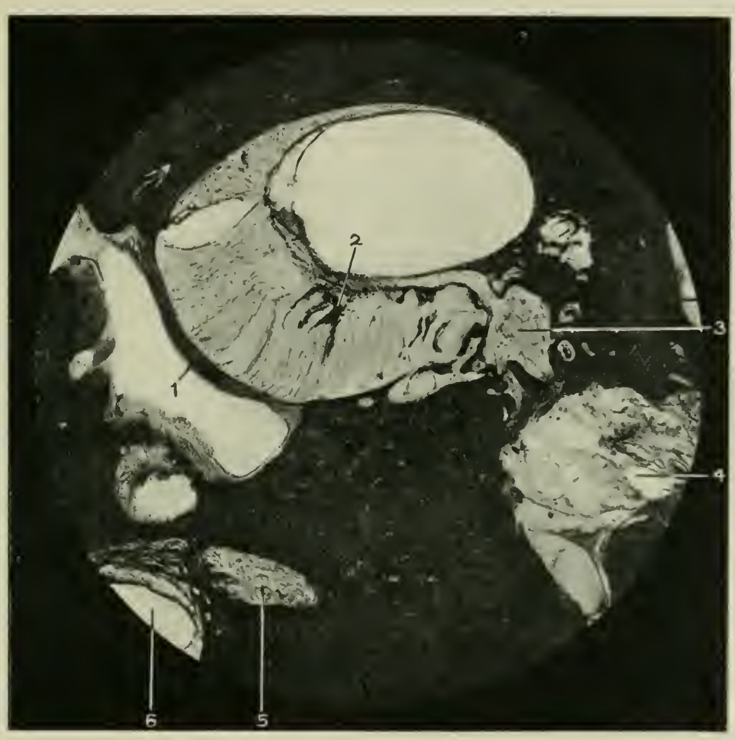

FIG. 17 (Case II).

Horizontal section of right ear, No. 120, × 20 diam. 1, thickened membrane of round window; 2, new bone formation in connective or tumour tissue in scala tympani; 3, spiral canal infiltrated, and ganglion cells absent; 4, remains of cochlear nerve in fundus of internal meatus; 5, canal for nerve to crista of posterior canal infiltrated as above; 6, endolymph space of posterior canal.

project into the layers of the dura, and naturally are more numerous along the parasinoidal expansion of the longitudinal sinus. Cushing holds that these projections are really arachnoid granulations—i.e., enlarged Pacchionian bodies. Certainly the projections are very similar to the arachnoid granulations. On account of the internal hydrocephalus resulting from long standing acustic tumour these bodies may be particularly well developed and give rise to bone absorption at the cranial base. If these small swellings are really Pacchionian bodies one would expect them to be present in other cases with increased intracranial pressure — e.g., in other varieties of intracranial tumour. Professor Shennan, however, has informed the writer that such is not the case.

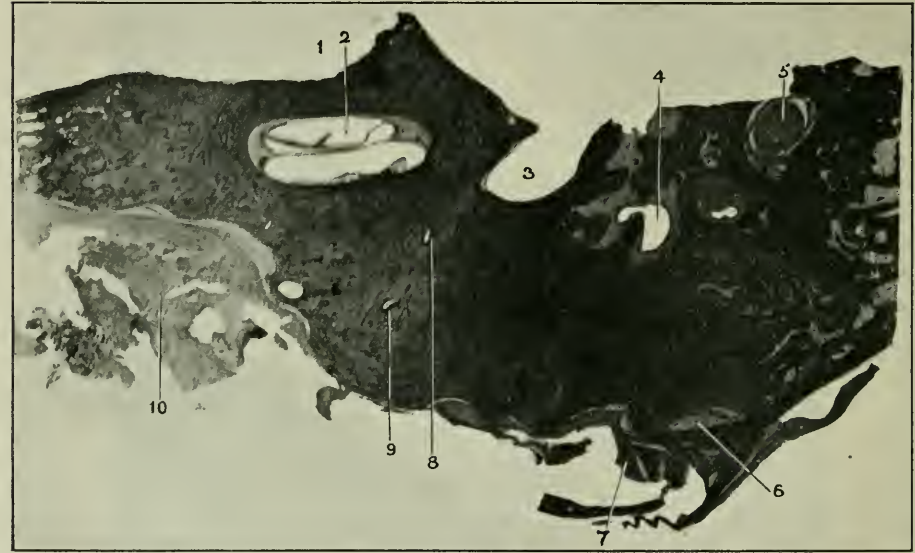

Fig. 18 (Case II).

Horizontal section of right ear, No. 245, × 6 diam. 1, carotid canal; 2, basal coil of cochlea; 3, niche of round window; 4, sinus tympani; 5, facial nerve; 6, endolymphatic aqueduct and saccus in contact with; 7, tumour tissue; 8, vein accompanying; 9, perilymphatic aqueduct; 10, tumour.

A microscopical section of one of the swellings is shown in fig. 20. Professors Lorrain Smith and Theodore Shennan, Dr. J. W. Dawson and Mr. Richard Muir have kindly examined these sections and have given their opinions that the tumours are composed of nerve tissue similar to that seen in gliomata. This tissue certainly has the power of eroding the bone. Key holds that the protrusions are small gliomatous "metastases" in the arachnoid. Without adopting the idea of metastasis or secondary deposits of glioma due to blood infection, it may well be that the small swellings in the brain membranes are due to overgrowth of glia tissue — in other words, that they are caused by a diffuse gliomatosis. This view is strengthened when we remember the relationship of acusticus tumours to general neurofibromatosis.

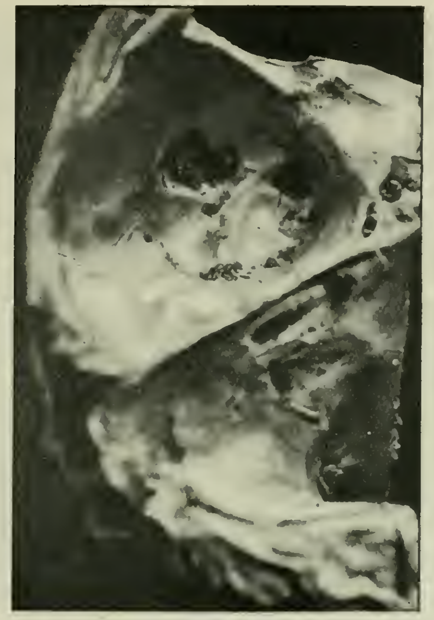

FIG. 19 (Case II).

Shows middle fossa of skull after removal of dura, with deep pits in the bone.

Case III.—(Recorded by kind permission of Dr. Edwin Bramwell.) W. H., male, aged 43, blacksmith, had been deaf in the right ear for six years but was otherwise quite well till two years ago, when he was operated on for appendicitis. Soon after this he began to complain of giddiness and unsteady gait (as if he were intoxicated) and of occasional vomiting. He felt a disinclination to exert himself. The giddiness was always present more or less but there were acute exacerbations. Patient stated that he tended to stagger to the right side. He also noticed slight weakness of the right side when fatigued and occasional right-sided headache (tight feeling) which he could not locate exactly. A little later diplopia was noted.

Examination by Dr. Edwin Bramwell (January 10, 1919) showed no obvious defect of the mental faculties, speech, articulation or phonation, though his doctor reported that the patient had always been a little "slow." There was distinct nystagmus on looking to the right and nystagmoid movements on looking to the left; the pupils were equal and reacted to light; the ocular movements were satisfactory. Dr. Bramwell was not able to satisfy himself as to the weakness of the right external rectus mentioned by the patient's doctor. Dr. Bramwell, however, noted marked deafness in the right ear. There was no definite history of tinnitus. The grasp of the right hand, as taken by the dynamometer, was 85, while that of the left

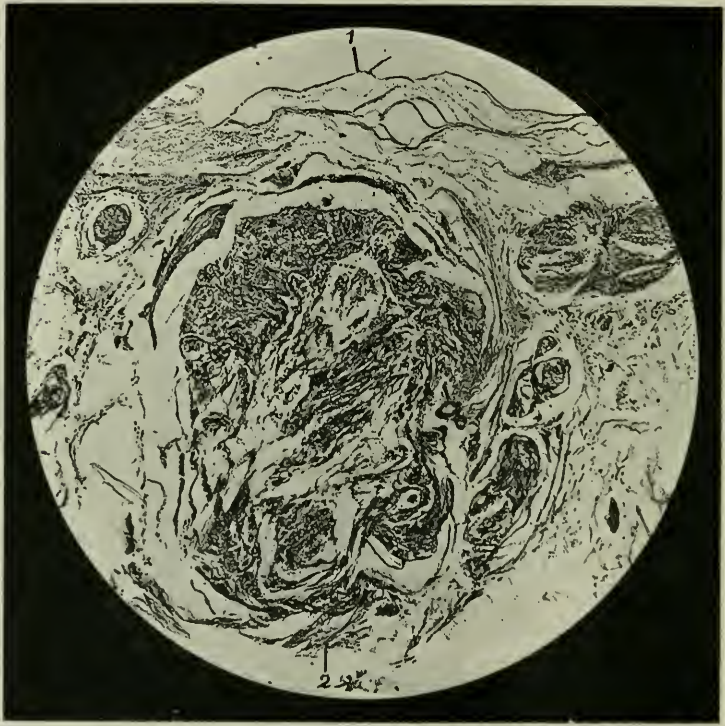

FIG. 20 (Case II).

Shows tumour of the pia arachnoid, which occupied one of the small bony erosions: The section shows a fibrous, cellular growth, apparently composed of neuroglial tissue. The neuroglial fibres appear to run in bundles in various directions. Cushing holds that these swellings are hypertrophied Pacchionian bodies, while Key believes that they are gliomatous metastases in the arachnoid. They certainly appear to be of a gliomatous nature.

hand was 120. (The patient was a right-handed man.) There was no definite asynergy of the right arm and no astereognosis or tremor. The finger-nose test was badly performed on the right side. There was no abnormal position of the head. Ophthalmoscopic examination showed distinct optic neuritis. The right knee-jerk was slightly more active than the left but there was no ankle clonus. The signs of Babinski, Gordon and Oppenheim were negative. The abdominal reflexes were present and active on the two sides. There was no definite weakness in his right leg nor was there any noticeable disturbance of gait. Nothing abnormal was found on examination of the thoracic or abdominal viscera. The blood-pressure was 135. Urine normal. Dr. Bramwell was of opinion that the hemiplegia did not present any pyramidal characteristics and was probably due to disturbance of the cerebellum on the right side. He thought that the patient's condition was probably caused by an extra-cerebellar growth originating in the eighth nerve. The character of the symptoms did

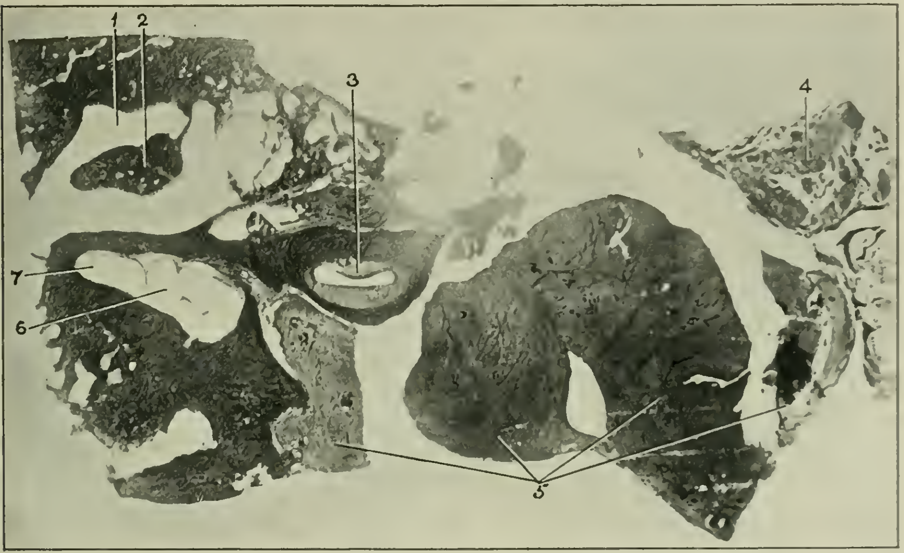

FIG. 21 (Case III).

W. H., male, aged 43. Tumour of right eighth nerve. Horizontal section of right ear, No. 85, × 4 diam. 1, outer attic; 2, joint between malleus and incus: 3, basal coil of cochlea; 4, Gasserian ganglion: 5, tumour which, unfortunately, was incised at the post-mortem in an attempt to remove it entire attached to the brain; 6, ampulla of lateral canal; 7, convexity of lateral canal.

not support the diagnosis of intracranial syphilis suggested by the patient's doctor.

The patient was again seen by Dr. Bramwell on February 19, 1919, when a change in the position of the head was noted. It was held slightly depressed to the left, with the chin pointing to the right—the position assumed by the head of a monkey after one lateral lobe of the cerebellum has been removed. Dr. Bramwell, however, was of opinion that this position could not be regarded as of localizing value in relation to the side of the growth, since the head is sometimes depressed towards the side of the tumour and sometimes towards the opposite side, the posture varying according as the lesion is exerting an exciting or a paretic effect. Dr. Bramwell was of opinion that the tumour was on the right side and based this view mainly on the presence of deafness on the right side for several years before other symptoms appeared. He thought that the growth was probably of considerable size.

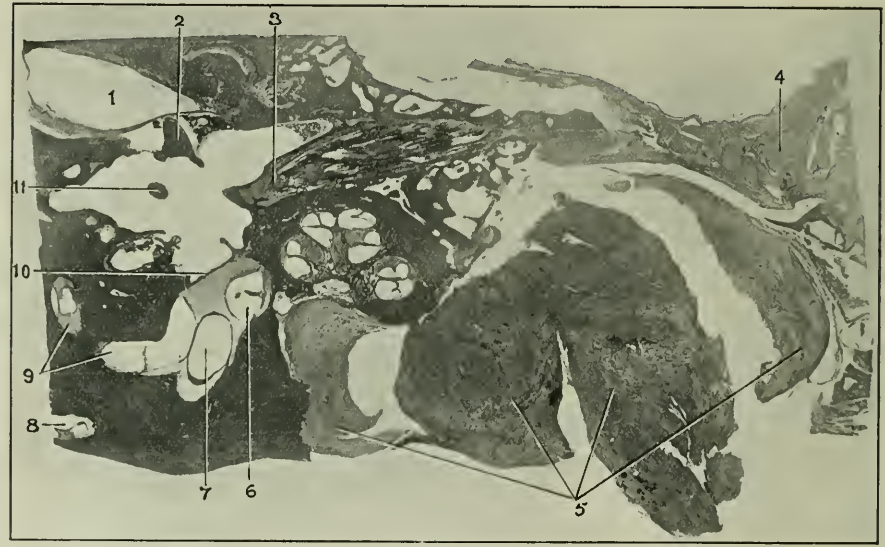

Fig. 22 (Case III).

Horizontal section of right ear, No. 147, × 4 diam. 1, external meatus; 2, malleus; 3, tensor tympani; 4, Gasserian ganglion; 5, tumour; 6, dilated saccule with granular contents; 7, utricle; 8, smooth end of posterior canal; 9, lateral canal; 10, footplate of stapes; 11, long process of incus.

The patient was examined by Dr. Logan Turner (February 22, 1919) who obtained a history of tired feeling and stupidness at work, failing eyesight, nausea, frontal headache, slurring speech, attacks of giddiness two or three times daily during which the patient felt that he could not see. Dr. Turner found the right drumhead slightly opaque but otherwise normal. *Cochlear apparatus*: Whisper was heard by the left ear at 18 in. in the noisy out-patient department, whereas the loud voice was not heard by the right ear when the noise box was placed in the left ear. On Weber's test the tuning fork was lateralized to the left (good) ear. With the noise apparatus in the left ear the patient could not hear the tuning-fork placed on his right mastoid. C_{4096} was not heard by the right ear, nor was Galton's whistle. *Vestibular apparatus*: There was marked nystagmus on looking to the right,

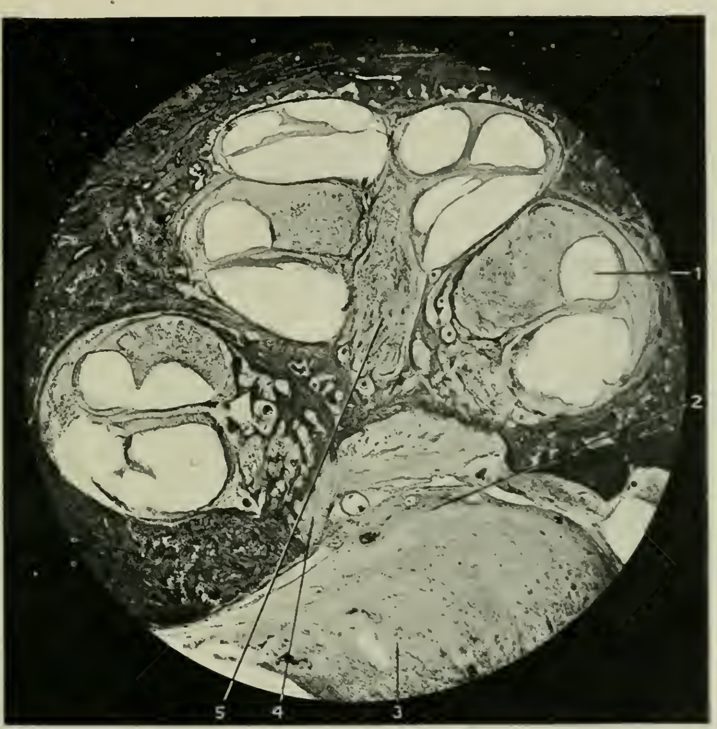

FIG. 23 (Case III).

Axial section through right cochlea, No. 147, × 17 diam. 1, dilated cochlear canal, basal coil; 2, capsule of tumour; 3, tumour tissue; 4, cochlear nerve compressed by tumour tissue; 5, central canal of modiolus.

less marked to the left. Pointing test normal. On cold syringing of the right ear there was no increase in the nystagmus to the left.

The patient was further examined by the writer (J. S. F.) in the beginning of March. It was noted that there was no facial paralysis, that the tongue was protruded straight, and that the movements of the soft palate were normal. The larynx also was normal and the vocal cords moved well. On testing the sense of taste it was found that the patient could appreciate salt on the anterior part of the tongue on the right side. Both drumheads were practically normal. *Cochlear apparatus*: The watch was heard at 4 in. by the left ear in a noisy room, but was not heard even on contact by the right ear. Rinne's test was positive in the left ear but infinitely negative in the right. All the

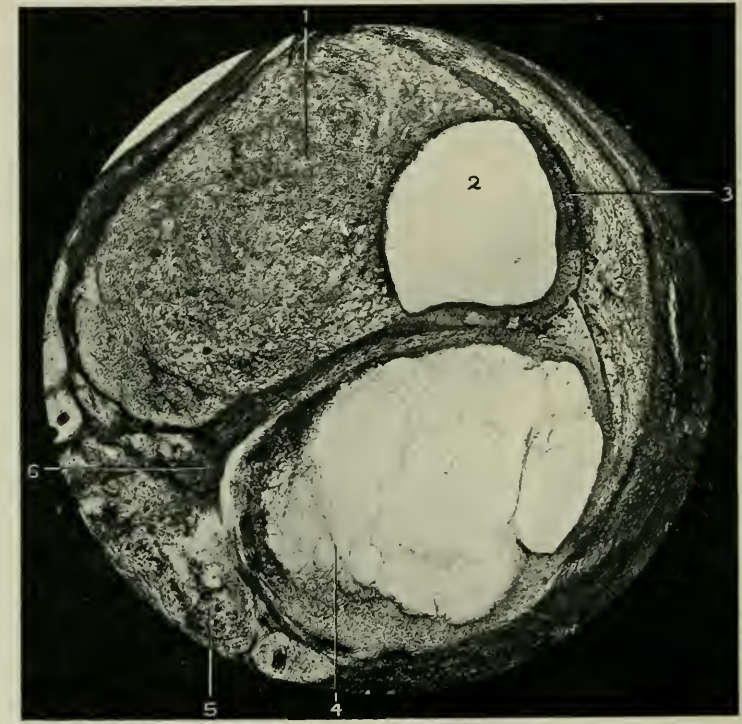

Fig. 24 (Case III).

Axial section through right cochlea, No. 147, × 50 diam. 1, fibrinous and granular exudate in scala vestibuli; 2, dilated cochlear canal; 3, stria vascularis; 4, fibrin threads in scala tympani; 5, spiral canal; some ganglion cells are still to be seen; 6, nerve entering bony spiral lamina.

tuning-forks from C_{32} to C_{1024} were heard by air conduction by the left ear but not by the right. With the left ear closed with the finger he apparently heard the raised voice at 4 in., but with the noise box in the left ear he was quite deaf. *Vestibular apparatus :* There was marked rotatory nystagmus to the right and to the left, and also slight nystagmus to the left on looking straight forward. Further, there was distinct nystagmus on looking up, the quick movement of the eye being to the left. No nystagmus on looking down. The pointing test at the shoulder, wrist and elbow joints on both sides was normal. There was no Rombergism. The rotation tests were impossible because the patient got very giddy in the turning chair on the commencement of rotation. Cold syringing of the left (good) ear for two minutes and ten seconds only resulted in stopping the slight rotatory twitch which was present on looking straight forward. (*Note.*—The difficulty of making exact observations of the results of vestibular tests in a case which presents marked spontaneous nystagmus in all directions, is very great. Numerous examinations by several observers are desirable.) *Galvanic*

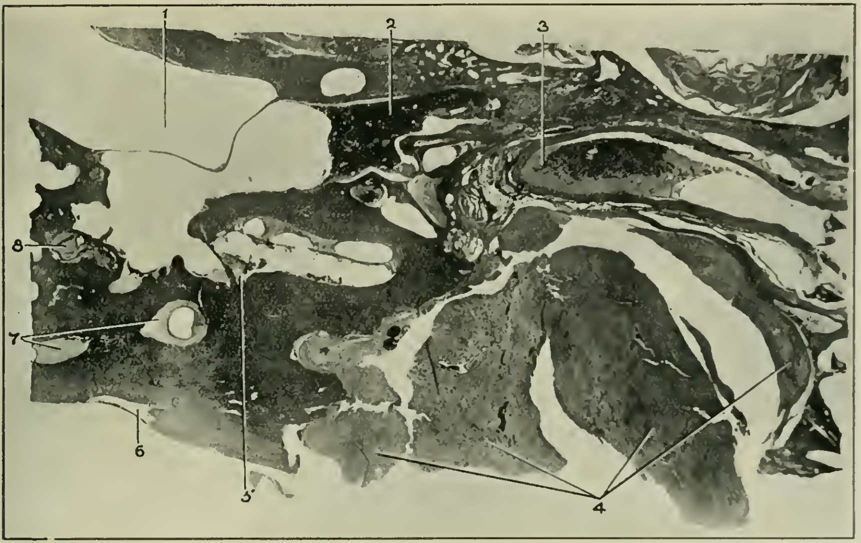

Fig. 25 (Case III).

Horizontal section of right ear, No. 227, × 4 diam. 1, external meatus; 2, hæmorrhage in tubal portion of tympanic cavity; 3, internal carotid artery; 4, tumour; 5, cochlear opening of perilymphatic aqueduct; 6, region of saccus; 7, two parts of posterior canal.

reaction: With the positive pole applied to the right ear it required 14 ma. to make the patient fall to the right, whereas the normal reaction is obtained with 4 ma.; the same finding, however, prevailed on the left side. On ophthalmoscopic examination Dr. Traquair found 5D. of swelling of the disc on each side: visual acuity about normal and only slight contraction of the visual field on the nasal side in one eye.

X-ray Examination.—Bilateral X-ray examination appeared to show that the internal auditory meatus was larger on the affected than on the normal side.

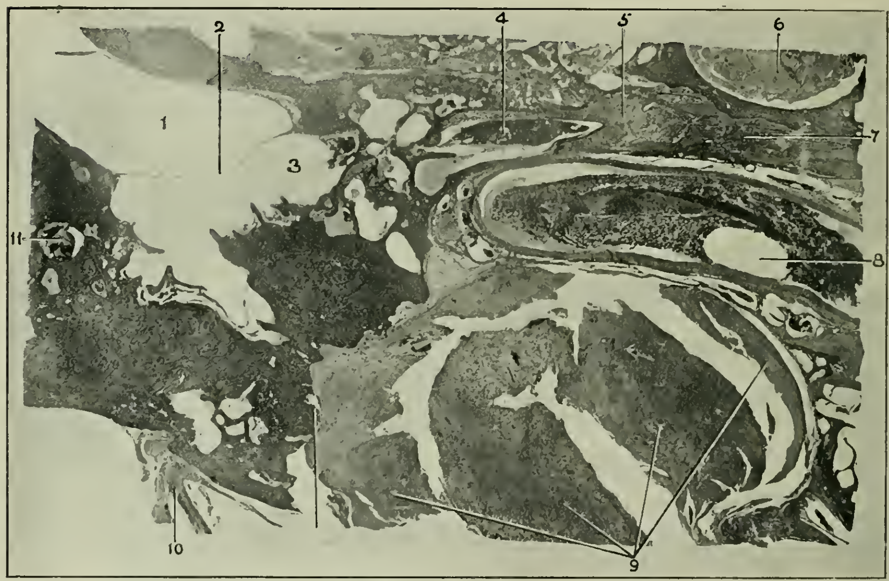

Fig. 26 (Case III).

Horizontal section of right ear, No. 282, × 4 diam. 1, external meatus; 2, tympanic membrane; 3, tympanic cavity; 4, junction of cartilaginous and bony portions of Eustachian tube; 5, cartilage of tube; 6, fifth cranial nerve; 7, tensor palati or dilator tubæ; 8, internal carotid artery full of blood; 9, tumour. Unnumbered line between 9 and 10 indicates the perilymphatic aqueduct in contact with the tumour; 10, dura mater in region of saccus endolymphaticus.

March 11, 1913: Operation by a general surgeon by the unilateral cerebellar route. The tumour, however, was not reached even with the finger. Puncture of the cerebellum was negative.

March 16, 1919: Death.

Post-mortem.—Convolutions flattened. Tumour the size of a large walnut in the cerebello-pontine angle on the right side, invading and partially destroying the right petrous temporal bone.

Microscopic Examination of Right Ear.—The tympanic membrane is healthy. There is blood in the tubal part of the tympanum and in the outer part of the attic, just external to the tensor tympani (figs. 22 and 25). The lining membrane of the attic itself is normal. There is a little hæmorrhage in the region of the sinus tympani. (The origin of the hæmorrhage in the tube and tympanic cavity is not quite clear but may have been caused by concussion at the operation.) The ossicles, joints and tympanic muscles are normal. The oval window is healthy. The niche of the round window shows a little serous exudate but the mucous membrane layer is not thickened (fig. 25).

Labyrinth Capsule.—The growth has distinctly eroded the bone above and in front of the cochlea. Apparently the lamellar bone has yielded much more easily to erosion than the cartilage bone. Tumour tissue also intervenes between the internal carotid artery and the cochlear capsule. The new growth comes right up to the carotid canal and also reaches the base of the modiolus (figs. 22, 23, 25 and 26). The tumour appears rather to have compressed the cochlear nerve (as it emerges from the modiolus) than to have infiltrated it. In this region the tumour appears to be encapsulated. The cranial end of the fossa subarcuata appears to be blocked by the tumour.

Cochlea.—The basal coil of the cochlea shows both scala tympani and scala vestibuli full of fibrinous exudate (figs. 23 and 24). Reissner's membrane is bulged upwards towards the scala vestibuli. In the lower part of the coil Reissner's membrane is ruptured: artefact? Corti's organ is not well formed in the lower part of the basal coil (fig. 24). The membrana tectoria is lying almost on top of the basilar membrane, there being only a low irregular layer of cells between the two. Here also the stria vascularis is atrophic but in other parts it is well formed. In the middle and apical coils Corti's organ is flattened but the supporting cells are still to be made out. The fibrinous exudate in the cochlea is much more marked in the basal than in the middle or apical coils. In all coils there is more exudate in the scala vestibuli than in the scala tympani. There is exudate in the cochlear end of the perilymphatic aqueduct. Further in, the cochlear aqueduct is almost surrounded by the growth (fig. 26). The tumour does not infiltrate the modiolus. The cochlear ganglion cells appear to be shrunken. The helicotrema shows granular exudate.

Vestibule.—The saccule is dilated but the neuro-epithelium is normal.

The otolith membrane appears to be very thick, but this is due to a layer of coagulated lymph on the surface. The neuro-epithelium of the utricle is normal and the otolith membrane is in position. In the perilymph space of the vestibule, between the saccule and the oval window, granular fibrinous material is present (fig. 22). The vestibular end of the endolymphatic aqueduct appears normal, but towards its cranial end the aqueduct contains some red blood corpuscles.

Canals.—The ampullary end of the *superior* canal shows exudate in both endo- and peri-lymph spaces. The crista is well formed. There is a little exudate in the perilymph space at the smooth end of the superior canal. The smooth end of the *posterior* canal, where it joins the crus commune, is almost normal, but here and there one notes a swollen, almost polypoid, condition of the lining membrane of the membranous canal. There is a little granular exudate in the convexity of the posterior canal, which has been opened by the saw cut. The ampullary end of the posterior canal shows the same condition, but the crista and cupula are normal. In the *lateral* canal the perilymph space contains a marked fibrinous network. The crista of the lateral canal is present, with the cupula. In the convexity of the lateral canal the endolymph space is practically normal, it shows only the œdematous swelling of the lining membrane.

Internal Meatus.—The internal meatus is enormously enlarged by the tumour (figs. 21, 22, 25 and 26) which comes right up to the fundus. (Unfortunately at the post-mortem an attempt was made by the pathologist to remove the tumour entire, attached to the brain, and this has to some extent spoilt the appearance of the growth in the internal meatus (figs. 25 and 26)). The nerve from the utricle is being compressed in an outward direction by the tumour—i.e., the fibres are being pushed into the base of the modiolus. The whole anterior wall of the internal meatus has been eroded and the dural lining is present only in the fundus.

Saccus Endolymphaticus.—The tumour tissue comes right up to the saccus endolymphaticus and in fact invades it. This may account for the hæmorrhage in the lower part of the ductus. The invasion of the saccus may also account for the dilated condition of the utricle and saccule, though one would expect that the pressure so caused would be counterbalanced by that on the perilymphatic aqueduct.

Remarks.—In this case the condition of the labyrinth differed plainly from that seen in Cases I and II. There was no new connective tissue nor bone formation and the hollow spaces of the labyrinth contained only fibrinous exudate. In this connexion we must remember that an operation was performed five days before death. Although deafness in the affected ear had been present for six years when the patient was first seen, it is possible that Case III was of comparatively short duration when contrasted with Cases I and II. If this is so we may look on the fibrinous exudate as the early stage of "choked labyrinth," while the formation of new connective tissue and bone represents the late stage. On the other hand, there is strong reason for supposing that the delicate tissue seen in the spiral canal and also in the scalæ of the cochlea in Cases I and II may be gliomatous—due to infiltration of tumour along the nervous and vascular paths. In all three cases there was definite evidence of degeneration of the nervous structures of the cochlea, whereas the vestibular end organs were comparatively well formed. In the first case it is true the crista of the superior canal was absent on the side of the tumour, but the remaining cristæ, along with the maculæ of the utricle and saccule, were well formed.

ÆTIOLOGY.

(1) *Heredity.*—Neurofibroma of the eighth nerve is intimately related to general neurofibromatosis, which, according to Alexis Thomson, may be inherited and occur as a family disease. Thomson holds that neurofibromatosis is a developmental disease dating from intra-uterine life, and that it may be associated with other congenital defects. It is a form of giantism of the connective tissue elements of the nerves. Herxheimer and Roth believe in the hereditary occurrence of tumours of the eighth nerve and Feiling and Ward have recently published a short paper on a "familial form of acoustic tumour."

(2) *Sex.*—This does not appear to have any important influence.

(3) *Age.*—Henschen has collected 125 cases in which the age was given as follows: Second decade, 9; third, 20; fourth, 26; fifth, 27; sixth, 6; seventh, 1. The average age of Cushing's thirty patients was 38 years, youngest 21, eldest 53. The symptoms of an acoustic tumour rarely occur before the third decade. Cushing holds that in a patient under 20 years symptoms of cerebello-pontine angle tumour probably arise from an intracerebellar glioma and not from an acoustic tumour.

(4) *Trauma.*—In only six out of Cushing's thirty cases was there any definite history of cranial injury, but in all of these the blow was in

the occipital region. In cases recorded by Henschen and by Alexander and Fränkl Hochwart accident appears to have accelerated the progress of the case.

(5) *Infection.*—Hessler has collected nineteen examples of acoustic tumour in patients who had suffered from middle-ear disease. Siebenmann and Nager, on the other hand, believe that tumours of the eighth nerve are associated with intra-uterine meningitis. The present Case I is interesting in this connexion.

PATHOLOGY.

At the cerebello-pontine angle we have the following tissues: (*a*) The petrous pyramid; (*b*) dura mater; (*c*) pia-arachnoid and vessels; (*d*) flocculus, lateral recess and choroid plexus; (*e*) eighth nerve.

Virchow has classified neuromata into (1) true and (2) false. According to Alexis Thomson true neuromata are rare. Some say they do not exist. False neuromata are composed of connective tissue derived from the nerve sheath and include acoustic neuro-fibromata. The relation of the tumour to the nerve varies. The nerve fibres may be spread out all over the tumour or may lie on one aspect. The most marked change is the formation of new connective tissue within the primary bundles of nerve fibres—i.e., the endoneural connective tissue between the fibres is the seat of the pathological process. The perineurium at first remains unchanged and forms the capsule of the tumour. There is apparently no new formation of nerve fibres but degenerative changes may be seen, especially in cases with sarcomatous transformation. Funkenstein states that in one case the growth had dilated the facial canal and penetrated as far as the geniculate ganglion (*see* Case II).

Symmetrical Tumours and General Neurofibromatosis.—Henschen states that five cases of "solitary" but symmetrical tumours of both eighth nerves have been recorded, and, further, that the cases of multiple neurofibromatosis with acusticus tumour number sixteen. The latter occur early in life, or at any rate the first symptoms come on early. In these cases both eighth nerves are always affected. Alexis Thomson's researches, however, do not confirm the opinion that the eighth nerves are always involved.

Macroscopic Appearances.—According to Sternberg the tumours vary in size from a hazel nut to a small apple. They have an uneven nodular surface and a greyish-pink colour. They occupy the cerebello-pontine angle and compress the pons, cerebellum and flocculus. They are not connected to the brain, the dura or the petrous bone. Usually they are hard with softer parts. On section the tumours may show cysts or spaces filled with blood. Cushing states that the tumour is diffuse throughout the endoneurium and cannot be shelled out of a more or less definite arachnoid capsule. This capsule can be clearly seen at operation, when the arachnoid spaces are distended with fluid. As the tumour grows the fifth and seventh nerves are pushed upwards and forwards, while the whole of the vagus group is pressed downwards and backwards. Large growths may envelop the facial nerve but the sixth nerve never comes into direct contact with the growth (Cushing).

HISTOLOGY AND DEVELOPMENT OF THE TUMOURS.

Tumours of the eighth nerve have received various names—e.g., fibroma, fibropsammoma, neurofibroma, endothelioma, spindle-cell sarcoma, fibrosarcoma, glioma, gliofibroma, neuroglioma, neurinoma (nerve thread tumours). Of 107 cases collected by Henschen, eighty-six showed a connective tissue structure and twenty-one appeared to be gliomatous. Henschen, however, believes that the gliomatous appearance may be due to œdema. Greenfield states that the main part of the tumours is composed of cells of the connective tissue type. They have an elongated nucleus and the cytoplasm tails off into a spindle-shaped process at either end; the process may branch. In addition, there are areas of looser texture in which multinucleated cells are common while the ground substance is formed of small rounded cells with scanty cytoplasm—suggesting myxomatous or glial tissue. Myelinated fibres are extremely scanty. All observers are agreed that the tumours do not consist of nerve fibrils. They are probably mixed tumours, gliofibromas composed of germinal tissue—i.e., they are tissue "rests." They probably spring from the crest of nerve tissue lying between the ectoderm and the dorsal surface of the neural tube from which the seventh, eighth, ninth and tenth nerves arise (Sternberg and Cushing). The eighth nerve is loose in structure (portio mollis) and is therefore more disposed to include this germinal tissue.

Solitary tumours of other cranial nerves are practically unknown, whereas Henschen has collected 130 cases of solitary tumour of the acoustic. From whence comes this predisposition of the eighth nerve? Henschen believes that the tumours originate from connective tissue in and around the inferior vestibular branch of the distal portion of the nerve in the internal meatus. He points out that with the exception of the eighth nerve the region of exit of the cranial nerves remains more or less unchanged from the cartilaginous stage at the third month to the bony stage at the sixth month. In the latter cases the cartilaginous exit is simply transformed into the bony one, whereas around the eighth nerve there is considerable new formation of bone directly from embryonic connective tissue. Boettscher and Kölliker have shown that the labyrinth capsule, the modiolus, the bony spiral lamina, and the walls of the internal auditory meatus are made up of bone formed from embryonic connective tissue. Henschen holds that between the branches of the eighth nerve in the internal meatus there is present embryonic connective tissue and that this explains why the eighth nerve is so often the seat of tumour. Orzechowski, on the other hand, thinks that the tumours are gliomatous and have little or nothing to do with the acoustic nerve and holds that they proceed from a malformation of the median wall of the lateral recess. Orzechowski, however, was only judging from one case. Henschen admits that tissue resembling neuroglia is to be observed in the tumours but maintains that most of the tissue is of a fibroid character.

Ballance states that Balfour, Beard, Dohrn, Hoffmann and others have demonstrated that, during the first days of embryonal development, neuroblasts migrate from the nerve centres into the mesoderm. It is possible to distinguish three kinds of neuro-epithelial elements which, primarily identical, later become differentiated into cells each of which elaborates a specific substance: (*a*) ganglion cells; (*b*) glia cells; (*c*) neuroformative cells, or peripheral neuroblasts. These last alone give origin to nerve fibres. Bruce and Dawson came to the conclusion that fusiform nucleated cells, linked on to one another, form embryonic nerve fibres. They believe that their researches have confirmed the theory of the multicellular structure of the peripheral nerve fibre. New nerve fibres are formed by the same process which gives rise to neuromata in the repair of divided nerves. A neuroma thus arises from the perverted function of the neurilemma sheath.

The eighth nerve consists of two parts: (*a*) a central non-medullated, and (*b*) a peripheral medullated portion (*see* figs. 8 and 10). The junction of these two parts occurs just within the internal auditory meatus at its cranial end. In the central part the fibres have no neurilemma and the endoneurium consists of a glial tissue just as in the spinal cord. Henschen states that in all cases of small tumour which were thoroughly described (Toynbee, Wolff, Habermann, Panse, Alexander, Henschen, Quix), only the distal part of the nerve was affected. The tumour was situated at the cranial end of the internal meatus—i.e., just beyond the junction of the two parts of the nerve. In case of large tumours there is always a cone-shaped portion of the growth in the internal meatus. Henschen holds strongly that many cases regarded as cerebello-pontine angle tumours—e.g., those which have caused marked erosion of bone, are not true tumours of the acusticus. Cushing believes, and the present writer agrees, that the tumours arise from tissue rests near the transition zone. In any case the tissue seems to be transitional—e.g., the fibrous elements do not stain quite like fibroblastic tissue, nor are the glia fibrils, if such they are, in the reticular areas often fully formed. Cushing holds that the tumour is a true nerve tumour, either a neuro-fibroma or a neuro-glioma, or a neuro-fibro-glioma. It arises from nerve tissue elements even though these are merely supporting structures. This was Virchow's original view. Verocay regards all these tumours as types of glioma. Berlstein and Nowicki think the growth is composed of nerve tissue like glia, as in neurofibromatosis. They use the term neurinomata.

Mallory reported on one series of Cushing's cases without knowing their clinical history, and always gave a diagnosis of fibrosarcoma or endothelioma, more often the latter. Councilman, reporting on the Boston series, made a diagnosis of glioma at first, but later shifted to endothelioma. The tumours show two main types of tissue: (1) Dense interlacing fibrous bands, and (2) loose reticular tissue. The two are as a rule fairly sharply defined. (1) In the fibrous parts the nuclei show a striking tendency to a palisade arrangement. Whorl formation may also be apparent. In some the cells are so numerous that they may be called fibrosarcoma, though there is no evidence of malignancy in the manner of growth, and mitotic figures are very rare. The tissue does not stain like a fibroma—e.g., it does not stain brilliant blue with Mallory stain, nor does it stain bright red with van Gieson. (2) The reticular tissue appears œdematous and glial fibres, or what appear to be glial fibres, may be detected. Occasionally cells suggesting ganglion cells have been found. Some of the tumours are extensively impregnated with fat cells. Pigmentation also is frequently seen. Cysts may occur, though less commonly than in true gliomas. At times the hyaline formations about the blood-vessels assume a whorl arrangement resembling psammoma, though there is no deposit of lime salts. Occasionally nerve fibres, probably pre-formed acoustic fibres, may be detected, usually at the periphery. Councilman states that the tumours have a remarkable sameness of structure. They have the histological characteristics of slow growth, the nuclear material being relatively small in amount. Hyaline degeneration of the vessels is common. This may lead to closure of the vessels and degeneration of portions of the growth. Hæmorrhages are rare. The basic structure of the tumour is fibrous and has the characteristics of white fibrous tissue, though it does not give the usual staining reactions.

PATHOLOGICAL CHANGES IN THE LABYRINTH.

Toynbee found evidence of old bilateral otitis media with fixation of the stapes in a case in which there was a tumour the size of a small pea connected to both cochlear and vestibular nerves. Nager has examined the ears of a microcephalic deaf-mute idiot. The promontory showed exostoses and the round window niche was filled with fat. The long process of the incus was adherent to the facial canal. The footplate of the stapes was thickened. On one side there was a tumour in the spiral canal of the lower whorls of the cochlea. The tumour had broken through into the scala tympani. The conditions found, except for the tumour, corresponded to those seen in cretins. Nager thinks that the neuroma was probably the result of a former inflammation—i.e., of an intra-uterine meningo-encephalitis that had been recovered from.

Alexander and Fränkl-Hochwart have examined the labyrinth in one case of acoustic neuroma and found degenerative atrophy of the cochlear nerve and spiral ganglion; atrophy of Corti's organ and the stria vascularis; dropsical degeneration of the spiral ligament. Alexander in another case found the round window membrane much thickened by a layer of connective tissue on its cochlear side. The perilymph spaces contained a network of fibrin threads. (Alexander thinks that this fibrin network becomes organized into connective tissue.) The cochlear nerve in the modiolus was atrophied and the bony spiral lamina filled with connective tissue. The endolymph space was free from fibrin. All the labyrinth nerve endings were degenerated —cristæ flat, cupulæ and hair cells of Corti's organ absent. The maculæ showed the same change. The spiral ligament was infiltrated with small cells and there were some leucocytes in the perilymph space—evidences of inflammation (?). The stria vascularis was degenerated. Corti's organ was absent in parts. Berlstein and Nowicki also found fibrin present in the labyrinth. The spiral ganglion and nerve fibres had disappeared in parts but the vestibular ganglion was fairly well formed. The tumour did not reach the fundus of the meatus. Corti's organ in the basal coil had disappeared. Panse found the spiral ganglion cells greatly reduced and replaced by round or spindle cells. Funkenstein in a case of multiple neurofibromatosis found the facial canal dilated to the knee and filled with tumour. The hollow spaces of the modiolus were filled with tumour which had passed into the bony spiral lamina. The scala tympani of the lower coil was also filled with tumour: above this there was exudate. Only traces of Corti's organ remained. The nerve canals and vestibule were filled with tumour. Folke Henschen found the bony walls of the internal meatus eroded, especially the anterior wall. There was no infiltration by the tumour.

The microscopic examination of the labyrinthine nerve endings in cases of tumour of the nervus octavus is of interest in connexion with the question of secondary degeneration of sensory nerve endings after they have been cut off from their ganglia. Wittmaack has cut the seventh and eighth nerves in animals before entrance into the internal meatus and has produced degenerative changes in the cochlear nerve ending in the labyrinth. These results are in conflict with the hitherto accepted idea regarding secondary degenerations. Waller's law states that after supra-ganglionary division of a sensory nerve there is no degeneration of the peripheral neurone. In cases of slow compression of the eighth nerve by neurofibroma, Panse (and also Alexander, as has been stated above) found atrophy of the spiral ganglion, slight degeneration of vestibular ganglion, and loss of the sensory cells of Corti's organ. Zange found the vessels in the labyrinth dilated, Corti's organ absent in the basal coil with absence of the nerves to this coil. The spiral ganglion cells elsewhere were reduced in number. The vestibular nerve, however, was well preserved. Zange's results agree fairly well with the experimental findings of Wittmaack—i.e., cochlear degeneration but vestibular nerve and apparatus normal. Quix, in one case, found the internal meatus free from tumour, which had grown into the temporal bone in the neighbourhood of the cochlear aqueduct and had blocked the aqueduct. The perilymph space was dilated and the cochlear and vestibular ganglia were degenerated. The nerve end organs and the stria vascularis were normal. Quix says the internal auditory artery was normal in his case. The difference between Quix's case and those of Alexander and Panse is that in the former the internal meatus was free and therefore the internal auditory artery and the stria vascularis were normal.

SYMPTOMS.

The clinical history of a typical case may be divided into three stages: (1) Initial or "otological" stage, with auditory and vestibular symptoms, followed by headache; (2) intermediate or "neurological" stage, with involvement of other cranial nerves, pressure symptoms and "cerebellar seizures"; (3) terminal stage. It is, however, difficult or impossible to be arbitrary in describing each stage.

(1) *Initial Stage.*—In the great majority of cases auditory and vestibular symptoms are the first to make their appearance; thus Berlstein and Nowicki found that tinnitus was the first symptom. Hezel mentions deafness and tinnitus. Ziehen noted deafness first of all, followed by giddiness. Some observers have found that the hearing may be normal or almost normal at first. Henschen's first case began with deafness and noises. He noted the initial symptoms in 105 out of his 136 collected cases. In sixty, disturbance of hearing was the first symptom (deafness in fifty and noises in ten). Giddiness was noted in twenty-five. In sixty cases headache appears to have been the first symptom, but, as Cushing points out, unilateral deafness may not be noticed by the patient. Henschen remarks that the order of the symptoms may depend upon the site of origin of the tumour. If it arises in the eighth nerve one would certainly expect cochlear and vestibular symptoms, whereas if it arises from the dura the symptoms may be different. Alexander, Fränkl-Hochwart and Sorgo agree that giddiness with nausea, vomiting and stumbling gait, along with occipital headache and tenderness over the neck muscles, are early symptoms of tumour of the eighth nerve. Cushing states that a diagnosis of acoustic tumour can be made with reasonable assurance only when auditory manifestations definitely precede the evidences of involvement of other structures in the cerebello-pontine angle. It is rare for the eighth nerve to be the only one affected by the time that patients present themselves in the neurological clinic, from which apparently Cushing gets most of his cases. In twenty-five of Cushing's thirty cases the first symptoms were auditory; as a rule tinnitus preceded the onset of deafness. Seven patients showed some remains of hearing, while a smaller number gave some response to caloric tests — i.e., complete vestibular paralysis was more common than complete cochlear paralysis. Vestibular disorders may precede the cochlear symptoms. Nystagmus is an early sign, and it is usually stated that it is present to both sides. There is, however, no unanimity in the observations as

to the direction of the nystagmus, some observers finding it more marked to the sound side and others to the diseased side. Nystagmus on looking up and also on looking down has been frequently observed. Cushing found that horizontal nystagmus was more marked when the patient looked towards the side of the lesion. Patients usually state that the vertigo is relieved if they lie on the sound side and increased by turning on to the affected side (as in labyrinthitis). Bárány states that he has never obtained a caloric response in the examination of more than thirty cases of acusticus tumour, whereas some hearing may be preserved. Headache may be regarded as a symptom coming between the "otological" and "neurological" stages. It is made worse by lying down or by movements of the head, and is usually much more severe at night. At first the pain is referred to the occipital region on the homolateral side, and later to the frontal region, often on the contralateral side. The pain may also be localized in the temporal area. Cushing notes suboccipital discomfort, made worse by stooping or straining.

(2) *Intermediate or Neurological Stage.*—Henschen states that headache was present in 100 of 105 cases. In two-thirds the headache was occipital and in one-third frontal. Later, there is *difficulty in progression.* The patient at first walks with a broad base. Cushing says that difficulty in walking may vary from a slight tendency to fall on turning to a pronounced drunken reeling. This is often associated with a definite loss of tone in the limbs on the homolateral side, especially in the lower limbs. Statements as to the presence of paresis of the extremities—homo- or contra-lateral—and as to the condition of the deep reflexes vary very much. Patients also find it more difficult to balance on the foot of the affected side. Rapid movements of pronation and supination are performed with difficulty by the homolateral upper extremity. There is a tendency for the patient to hold his head to the side of the lesion and to deviate and fall to this side. These phenomena, however, are not constant. Statements vary as to the position of the patient in bed. Some observers state that he tends to lie on the diseased side. Cushing, however, finds that the patient tends to lie on the sound side, and to look towards the affected side. Gierlich records a case of tumour on the right side, with nystagmus to the right, in which the patient lay on his back with the head and eyes turned to the left. A little later the *sight* begins to fail. Blindness may come on suddenly. On ophthalmoscopic examination choked disc is observed. Henschen states that optic neuritis may be earlier or

more marked on the side of the lesion. The patient may suffer from diplopia associated with paresis of the sixth nerve. In thirty out of thirty-five cases recorded by Henschen the abducens paresis was homolateral. The condition of the pupils varies, but the majority of observers have found that the pupil on the affected side is narrower than that on the healthy side. Ptosis and exophthalmos have also been noted. *Loss of the conjunctival reflex* is also a comparatively early symptom. Later on there is some loss of sensation in the corresponding side of the face, but contralateral affection has been noted. Tenderness over the exit points of the fifth nerve was noted in ten cases (Henschen). The mucous membrane of the mouth may show this loss of sensation before the skin of the face. Paralysis of the homolateral muscles of mastication has been noted in several cases—the jaw passing to the side of the lesion. *Facial paralysis* is by no means always present in the early stages, but, as the case advances, it gradually appears, as a rule first in the lower part of the face. There may be facial twitchings. Facial paresis is usually homolateral, but Funkenstein has recorded a case in which it was on the contralateral side, and Henschen has collected ten similar records. Gibson, Henschen and Rhese have noted hypersalivation (sublingual gland ?) as an additional symptom of involvement (irritation) of the facial nerve. *Disturbance of speech* (dysarthria) comes on comparatively late, and has been variously described as "thickness of speech," "toneless voice," "slow nasal speech," "slurring," &c. This dysarthria may be due to involvement of the seventh, ninth, tenth and twelfth nerves. *Loss of smell and of taste* are comparatively late symptoms, and are usually most marked or only present on the affected side. Paresis of the homolateral side of the palate has been noted. There may also be *return of fluids by the nose*. *Dysphagia* first manifests itself, according to Cushing, by a conscious effort required in the act of swallowing and by a tendency of food to "go the wrong way." Paralysis of the sternomastoid and trapezius have occasionally been noted. Homolateral paralysis of the hypoglossal nerve was noted in thirty of Henschen's cases, the tongue deviating to the side of the lesion.

"Cerebellar Crises," "Vagal Attacks," "Giddy Attacks," "Fainting Attacks."—The seizures which form such a marked feature of the clinical aspect of acoustic neuromas have been variously described by different authors under the above headings. The seizures are sometimes preceded by giddiness, headache, congestion of the face, dimness of vision or diplopia. Conjugate deviation of the eyes to the sound side

has also been recorded at the onset. At first the patient is conscious during the attacks, but later on he is usually unconscious. The attacks may last from a few minutes to an hour, and may occur once or more daily. During the attacks the pupils are dilated, and there may be jerkings of the face. The breathing is irregular and stertorous, and the pulse rapid and irregular. There are jerkings of the head and extremities, followed by general rigidity. The attacks may be accompanied by vomiting and followed by sweating. Cushing pictures these attacks as paroxysms of a most agonizing type, associated with retraction of the head and back, respiratory difficulty, altered pulse-rate, and a sense of impending death. He calls attention to the great variation in the character and severity of the symptoms from time to time. Sternberg believes that a hæmorrhage into the tumour may cause a sudden development of symptoms. In one of Cushing's cases the symptoms entirely disappeared for a few months. He holds that this phenomenon is best explained by circulatory obstruction. The tumour actually lies within the lateral cistern, the proper evacuation of which is more or less interfered with by the growth. Cerebellar crises are probably due to periods of excessive tension in the cistern.

(3) *Terminal Stage.*—The terminal symptoms include disturbance of memory, loss of weight, weakness of sphincter control, periods of excitement resembling those of delirium tremens, dementia, Cheyne-Stokes respiration and coma.

EXAMINATION OF THE EARS.

The tympanic membranes are usually normal. There may be tenderness on pressure on the tip of the mastoid on the affected side.

Functional Examination of the Cochlear Apparatus.—Although in the great majority of cases there is complete deafness in the affected ear, Sorgo states that the patient may in the early stages hear the conversation voice at 4 in. According to Rhese the patient may even hear a whisper with the noise box in the good ear. If such cases be followed up, however, we eventually get complete deafness in the affected ear. The watch is not heard at all by this ear. (Some observers believe that cases with good hearing are not genuine acoustic neuromas.) On Schwabach's test, bone conduction is shortened on the affected mastoid and is not influenced by closure of external meatus (Neumann). Weber's test is lateralized to the good ear. Rinne's test is absolutely negative on the affected side. Rhese says that *nerve deafness with loss of the lower tones* is diagnostic of a lesion in the nerve stem or in the central paths. The upper tones are also lost, and so we get concentric narrowing of the hearing field somewhat similar to the change in the visual field in cases of pressure on the optic chiasma by tumour of the hypophysis.

Vestibular Apparatus.—Spontaneous nystagmus is usually present to both sides. It may be increased by sudden movements of the head. Opinions vary as to whether it is most marked to the opposite side or to the side of the lesion. Rhese, Ziehen, Henschen, Neumann and Cushing think that it is most marked to the homolateral side. Apparently it is usually of the rotatory variety. In addition, at any rate in fairly advanced cases, there is usually spontaneous nystagmus on looking up and even on looking down. Many observers have noted that the spontaneous nystagmus changes from time to time, as in cases of cerebellar abscess. *Spontaneous vertigo:* The patient may state that during the giddy attacks external objects seem to rotate to the side of the lesion. *Pointing reaction:* Ziehen, Berlstein and Nowicki found the pointing reactions normal, while Neumann obtained uncertain results from the pointing test. *Rombergism* has already been noted. The patient tends to fall to the side of the lesion and tends to deviate to this side on walking. *Rotation:* Most observers have found rotation to that side, which tests specially the healthy ear, gives a longer "after-nystagmus" than rotation in the other direction—e.g., if the tumour be on the left side rotation to the left, which mainly tests the right vestibular apparatus, produces longer "after-nystagmus" than rotation to the right, which mainly tests the left inner ear. We must remember, however, Ruttin's views with regard to compensation in cases in which one vestibular apparatus has been functionless for a considerable period. Further we have the difficulty of estimating after nystagmus in cases in which spontaneous nystagmus is present. *Caloric test:* The caloric test on the healthy side gives a normal result, or even produces an excessive reaction—the "lasting nystagmus" of Neumann—which may continue for five minutes. (Neumann explains this phenomenon by supposing that Deiter's nucleus on the affected side has lost its inhibitory power owing to the pressure of the tumour. It is remarkable that there is no nausea or giddiness during this "lasting nystagmus.") On the other hand, there is no reaction on applying the caloric test to the affected ear. On using Ruttin's apparatus for the simultaneous cold caloric test of both labyrinths we only get a reaction from the healthy ear—

e.g., if the tumour be on the left side and the right ear be healthy, on using Ruttin's apparatus with the cold caloric test we get nystagmus to the left. *Pointing error after the caloric test:* Syringing the healthy ear produces the usual pointing error, whereas syringing the affected ear has no result. *Galvanic reaction:* Here again there is diminution or loss of the galvanic reaction on the diseased side. This test is said to be useful to distinguish a tumour of the eighth nerve from a case of spontaneous cure of labyrinthitis in which the galvanic reaction is retained, though the caloric reaction is absent. If, for example, a patient suffers from neurofibroma of the eighth nerve on the left side, and if the kathode is applied to the left ear and the anode held in the right hand, we may require to use a current of 5 ma. before we obtain nystagmus to the left. On the other hand, with the kathode applied to the right ear and the anode held in the left hand, we get nystagmus to the right with only 2½ ma. In Hezel's case there was no reaction from the nerve on the affected side and the patient did not feel giddy. On stimulation of the healthy side both giddiness and nystagmus were produced. Lastly, in the case mentioned above, with Ruttin's apparatus for applying the galvanic test, if the divided kathodes are applied to the ears while the anode is in contact with the forehead, we get nystagmus to the right with a current of 3 ma. (modified from the description of a case by Berlstein and Nowicki).

DIFFERENTIAL DIAGNOSIS.

If solitary tumours of the eighth nerve are to be diagnosed at the early stage it is of the utmost importance that otologists should thoroughly examine all cases of unilateral nerve deafness. The writer has found very little in the literature dealing with the differential diagnosis of acusticus tumours at the "otological" stage. Conditions which may simulate unilateral eighth nerve neuroma appear to be as follows: (1) Acquired syphilitic neurolabyrinthitis. Here the history of syphilitic infection and the result of the Wassermann reaction from the blood and cerebro-spinal fluid are the important considerations. (2) Neuritis of the cochlear or vestibular (or both) divisions due to (*a*) exposure to cold wind; (*b*) toxæmia in cases of pyorrhœa alveolaris, chronic tonsillitis or appendicitis; (*c*) quinine, salicylate, arsenical or other forms of poisoning. A careful history of the case and thorough physical examination should eliminate such conditions. Further, one does not find complete deafness and certainly not total loss of the vestibular reaction in the above conditions. (3) Hæmorrhage into the labyrinth in the bleeding diseases should be eliminated by an examination of the blood. (4) Senile or arteriosclerotic nerve deafness is bilateral, and, as a rule, is not accompanied by giddiness. Further, the low tones are retained, along with the vestibular reactions. (5) Unilateral congenital deafness is rare and is not associated with giddiness or nystagmus. In the writer's experience the vestibular reaction is present in such cases. (6) Circumscribed labyrinthitis and labyrinth fistula are of course associated with otitis media, and usually with cholesteatoma. (7) Recent diffuse labyrinthitis, whether of middle ear or meningitic origin, is not likely to be confused with acusticus tumour. The history and progress of the case differ distinctly in the two conditions (labyrinthitis and tumour). Further, the galvanic test is said to be of service in differential diagnosis. (8) Otosclerosis is occasionally associated with giddiness, but functional examination of the cochlear and vestibular apparatus should clear up any difficulty. (9) Serous meningitis in the lateral cistern (Bárány's symptom complex). Here we have as a rule a history of otitis media. Further, there is a well marked pointing error which is not usually found in cases of acusticus tumour. Lumbar puncture as a rule resulted in improvement in the cases reported by Bárány, and was followed by a return of normal caloric reaction.

Cushing states that the ideal we have to aim at is the recognition of the lesion when the eighth nerve alone is involved, though it must be admitted that many other disorders give rise to tinnitus, deafness and labyrinthine vertigo. When, however, the primary auditory and vestibular symptoms are followed by numbness of the corresponding side of the face, by periods of diplopia and lowered vision, by occipito-frontal pain and stiffness of the neck, by unsteadiness of gait, and, finally, by difficulty in swallowing and articulation we can, according to Cushing, be reasonably sure of the diagnosis on the history alone. Cushing gives the following list of possible errors in diagnosis : (*a*) The tumour syndrome is misinterpreted and the symptoms ascribed to another lesion—e.g., Ménière's disease, Bell's palsy, trigeminal neuralgia, multiple sclerosis, multiple neuritis of cerebral nerves, occipital neuralgia, torticollis, bulbar paralysis, locomotor ataxia, tuberculous or syphilitic basal meningitis, circumscribed serous meningitis in the lateral cistern. (*b*) A tumour is correctly diagnosed but incorrectly localized—e.g., tumours of the corpora quadrigemina, psammoma of the third ventricle, tumours of the temporal lobe, of the Gasserian ganglion, of the cortical facial area, or of the frontal lobe. (*c*) A tumour is correctly diagnosed and localized in the cerebello-pontine angle, but the relation of the growth to the eighth nerve is not appreciated. Here an accurate chronological history of the case is very important.

There may be difficulty in diagnosing the side of the tumour. In Henschen's first case and in Ziegenweidt's case a right-sided acusticus tumour was wrongly diagnosed as left-sided. (The trouble arises from the size of the tumour, which presses the brain stem over to the other side.) The best method is to judge by the deafness—e.g., in both the cases referred to above there was right-sided deafness. Vestibular examination would further confirm the diagnosis, but was not known at the time of these two cases. Henschen's views may be summarized as follows: (1) The discovery of choked disc on ophthalmoscopic examination serves to distinguish tumour of the eighth nerve from Ménière's disease. (2) In the diagnosis between cerebello-pontine angle tumour and intracerebellar tumour the late occurrence of choked disc favours the latter. (3) In distinguishing acoustic tumours from those of the petrous pyramid or the dura mater Henschen holds that the X-rays should help. He lays stress upon the otological examination of the case. Bárány claims to be able to make the diagnosis from the history of deafness and giddiness and the ear examination before other cranial nerve symptoms arise. Henschen gives details of a case of tumour of the right temporal lobe which was diagnosed by the neurologist as one of cerebello-pontine angle tumour, although Holmgren's examination of the ear showed that there was not complete deafness on the right side and that the caloric reaction was still present. Holmgren believed that the deafness was due to "choked labyrinth" and that the case was not one of cerebello-pontine angle tumour, and turned out to be right. Further, in this case X-ray examination showed the internal meatus to be equal on the two sides. Henschen points out that the portion of the tumour in the internal meatus dilates the meatus, and that this can be seen in a good X-ray. In one of his cases the left (affected) meatus had a diameter of 5 mm., while the right meatus was only 2·5 mm. (4) Regarding the diagnosis between acusticus tumour and acusticus neuritis Henschen says that the latter is usually bilateral, whereas, except in the rarest cases, the tumour is unilateral. Further, the tumour affects both cochlear and vestibular divisions, whereas neuritis shows a tendency to affect the former alone.

Ziehen states that the presence of choked disc is opposed to intrapontine tumour, as also is the unilateral character of the cochlear and vestibular trouble. According to Hezel, pontine tumours are characterized by paralysis of the eye muscles. On the other hand, the oculomotor nerves, with the possible exception of the sixth, are affected late in cerebello-pontine angle tumours. Gordon Holmes and Grainger Stewart state that in intracerebellar cases the subjective rotation and the apparent rotation of external objects are in the same direction—i.e., towards the contralateral side. In extracerebellar cases these two are sometimes opposed. This observation is doubted by Oppenheim, who states that ocular paresis, unilateral loss of the corneal reflex and of the function of one eighth nerve make up the characteristic triad of an acusticus tumour.

PROGNOSIS.

Sorgo gives one and a half to two years as the duration of life from the first symptoms till death. One of Alexander's cases was very rapid—the symptoms only lasting eight months. On the other hand Toynbee's case had deafness for twenty years, and one of Henschen's cases for sixteen years before death. In Fränkl-Hochwart's and Ramsay Hunt's case there was tinnitus and deafness for six years (*see* Cases I and III). Henschen gives the average duration of illness as three and a half years. Cases occurring in comparatively young subjects are apparently more rapid in their progress than those in older people. Cushing distinguishes (1) an initial stage of auditory and vestibular manifestations lasting one year; (2) an intermediate stage, lasting from six months to a year, with occipito-frontal pain, cerebellar inco-ordination and involvement of adjacent cerebral nerves; (3) a terminal stage, with choked disc, difficulty of speech and swallowing, cerebellar crises and respiratory difficulty.

Funkenstein states that only solitary unilateral tumours are suitable for operation. Bilateral cases are associated with more or less general neurofibromatosis.

TREATMENT.

It is not the object of this paper to discuss the details of surgical treatment. It may, however, be stated that there are two routes by which the tumour may be reached: (1) Occipital—either (*a*) unilateral (Krause), or (*b*) bilateral (Cushing); (2) translabyrinthine (Panse and Quix). (These two routes may be combined.) (1*a*) Henschen's statistics from forty-two collected cases show that the mortality by the

Krause method was 80 per cent. (1*b*) Cushing's statistics show a great improvement on the above, as the mortality was only 20 per cent. (2) In 1904, Panse proposed that acusticus tumours should be removed through the middle and inner ear, and in 1911 Quix of Utrecht was the first to operate by this route. As the labyrinth is functionally dead in these cases one need have no hestitation in removing it. Schmiegelow remarks that in healthy people the tympanum and antrum are sterile, and for this reason there ought to be no danger of meningitis. The only objection to the translabyrinthine route is the risk to the facial nerve and, although this nerve withstands destruction better than the auditory, by the time the patient is seen it has already been destroyed by the tumour in many cases. If, however, the facial nerve be healthy, it can be entirely exposed throughout its course in the petrous bone and turned to one side, though this adds greatly to the difficulty and length of the operation. In order to remove the growth, which may be as large as a bantam's egg, it is necessary to perform a free radical mastoid operation and thereafter to obtain space reaching in front to the carotid canal, below to the jugular bulb, behind to the sigmoid sinus, and above to the dura of the temporo-sphenoidal lobe. The danger of injuring the carotid is small, as this vessel runs in a canal which one can break open without difficulty in the removal of the petrous pyramid. (Ruttin had already shown that it was possible to remove the petrous pyramid during life.) Bleeding from the sigmoid sinus can be stopped by plugging, but the inferior petrosal sinus and jugular bulb are much more likely to be injured and to give rise to troublesome hæmorrhage. In fact, the operation may have to be broken off on account of severe bleeding. This need not prevent a successful result after a pause of a day or two (Quix original case). The escape of cerebro-spinal fluid is not likely to give rise to trouble. After removal of the petrous pyramid, one raises the temporo-sphenoidal lobe and splits the dura beneath the superior petrosal sinus as well as that lining the internal meatus, and thus completely exposes the tumour. After the tumour has been removed, in one piece or in bits, the wound cavity is packed with iodoform gauze or worsted, and a plastic operation is performed on the membranous meatus as at the end of an ordinary radical mastoid operation. Forselles records a case operated on by this method and states that the patient was free from recurrence two years later.

Schmiegelow states that he only knows of four cases which have been operated on by the translabyrinthine route, but as yet there have been no deaths. He thinks that if a tumour cannot be removed by the translabyrinthine route, it cannot be removed by the paracerebellar one, though the adoption of the former method does not prejudice a subsequent operation by the latter. He holds that the translabyrinthine is the only method which one can adopt with success in a case of true tumour of the acoustic nerve within the internal auditory meatus. Schmiegelow admits, however, that the translabyrinthine route is the more difficult, especially for the general surgeon who is not accustomed to the topography of the parts.

In conclusion the writer desires to tender his sincere thanks, first, to Dr. Edwin Bramwell for permission to record Case III, and also for his assistance in regard to the symptomatology of the affection; secondly, to Professors Lorrain Smith and Theodore Shennan for help concerning the histological changes; thirdly to Mr. Richard Muir for the photomicrographs; and lastly to the Carnegie Trust for providing the illustrations.

BIBLIOGRAPHY.

Alexander. *Zeitschr. f. klin. Med.*, 1907, lxii, p. 447.

Alexander and Fränkl Hochwart. *Arb. a. d. Neurol. Inst. in Wien*, 1904, xi, p. 385.

Balfour, Beard, Dohrn, Hoffmann. Quoted by Ballance.

Ballance. "Surgery of the Temporal Bone," 1919, ii, p. 536.

Bárány. *Monatsschr. f. Ohrenheilk.*, 1906, xl, p. 193; *Zeitschr. f. Ohrenheilk.*, 1908, lv, p. 414; "Handbuch der Neurologie," Berl., 1910, i, p. 919; iii, p. 811; *Trans. Internat. Cong. Med.*, Lond., 1913, Sect. XI, pt. ii, p. 53.

Bruce and Dawson. *Trans. Roy. Soc. Edin.*, 1913, xlviii, p. 697.

Berlstein und Nowicki. *Monatsschr. f. Ohrenheilk.*, 1913, xlvii, p. 415.

Boettscher und Kölliker. Quoted by Henschen.

Cushing. "Tumours of the Nervus Acusticus" (W. B. Saunders Company, Philad. and Lond., 1917).

Councilman. Quoted by Cushing, "Nervus Acusticus," p. 204.

Feiling and Ward. *Brit. Med. Journ.*, 1920, i, p. 496.

Forselles. *Nord. Tidskr. f. Oto-Rhino-Laryng.*, Bd. 1, No. 1, p. 29.

Fraenkel and Ramsay Hunt. *Ann. of Surg.*, 1904, xl, p. 293.

Funkenstein. *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1905, xiv, p. 157.

Gibson. *Edin. Med. Journ.*, 1896, xli, pt. ii, p. 689.

Gierlich. *Deutsch. med. Wochenschr.*, 1908, xxxiv, p. 1800.

Greenfield. *Brain*, 1919, xlii, p. 29.

Habermann. Quoted by Sorgo.

Henschen, Folke. *Centralbl. f. Ohrenheilk.*, 1910, viii, p. 221; "Ueber Geschwülste der hinteren Schädelgrube, insbesondere des Kleinhirnbrückenwinkels: Klinische und Anatomische Studien," Gustav Fischer, Jena, 1910; *Arch. f. Psychiat.*, 1915, lvi, p. 21; *Fortschr. a. d. Geb. d. Roentgenstrahlen*, 1912, xviii, p. 207.

Herxheimer und Roth. Quoted by Nager.

Hessler.

Hezel. *Zeitschr. f. Laryngol.*, 1912, v, p. 973.

Key. Quoted by Folke Henschen.

Krause. *Centralbl. f. Ohrenheilk.*, 1914, xii, p. 111.

Mallory. Quoted by Cushing.

NAGER. *Zeitschr. f. Ohrenheilk.*, 1917, lxxv, p. 349.
NEUMANN. *Centralbl. f. Ohrenheilk.*, 1911, ix, p. 354.
OPPENHEIM. *Berl. klin. Wochenschr.*, 1890, xxvii, p. 38.
ORZECHOWSKI. *Arb. a. d. Neurol. Inst. a. d. Wien. Univ.*, 1908, xiv, p. 406.
PANSE. *Arch. f. Ohrenheilk.*, 1904, lxi, p. 251.
QUIX. *Arch. f. Ohrenheilk.*, 1911, lxxxiv, p. 252; *Centralbl. f. Ohrenheilk.*, 1911, ix, p. 478, and 1912, x, p. 392.
RHESE. *Passow's Beiträge*, 1914, vii, p. 262.
RUTTIN. *Centralbl. f. Ohrenheilk.*, 1912, x, p. 537.
SCHMIEGELOW. *Zeitschr. f. Ohrenheilk.*, 1915, lxxiii, p. 1.
SIEBENMANN. *Monatsschr. f. Ohrenheilk.*, 1914, xlviii, p. 278.
SORGO. *Monatsschr. f. Ohrenheilk.*, 1901, xxxv, p. 285.
STERNBERG. *Zeitschr. f. Heilk.*, 1900, xxi, p. 163.
STEWART, T. GRAINGER and HOLMES. *Brain*, 1904, xxvii, p. 522.
STEWART, PURVES. "The Diagnosis of Nervous Diseases," Lond., 1916, p. 480.
THOMSON, ALEXIS. "Neuroma and Neurofibromatosis," Edin., 1900.
TOYNBEE. *Trans. Path. Soc.*, 1853, iv, p. 259.
VEROCAY. *Beitr. z. path. Anat. u. z. allg. Path.*, 1910, xlviii, p. 1.
VIRCHOW. "Die Krankhaften Geschwülste," Berl., 1863-65, i, p. 180; ii, p. 116; iii, p. 295.
WITTMAACK. Quoted by ZANGE.
WOLFF. *Passow's Beiträge*, 1912, v, p. 464.
ZANGE. *Arch. f. path. Anat.*, 1912, ccviii, p. 297.
ZIEGENWEIDT. *Pysch. en Neurol. Bl.*, Amst., 1899, iii, p. 36.
ZIEHEN. *Med. Klin.*, 1905, i, pp. 847, 874.

DISCUSSION.

The PRESIDENT: I need scarcely say the thanks of the Section are once more due to Mr. Fraser for the exhibition of his beautiful slides which we have seen with such interest and profit. Obviously they represent an extraordinary amount of research and of excellent work done, and our Section should be proud that one of its members can bring forward such work as Mr. Fraser has done. Sir Charles Ballance asked me to express his regret that he was unable to stay longer, as he wished to thank Mr. Fraser and congratulate him on his work. He also said he was particularly interested in the "choked labyrinth" theory, which he regards as a very good explanation of some of the appearances which Mr. Fraser has demonstrated. What interested me particularly were his remarks about the small excavations or pits in the bone. I have only once seen anything which corresponds to that, and it was a puzzling case in a lady who had had her breast removed for carcinoma seventeen years before she came under my care. She was sent to me because she had pain over the left frontal sinus and a swelling of the left upper eyelid. We thought she had probably some infection in her sinus. When we exposed the front surface of the frontal bone we were puzzled by a number of translucent little swellings the size of a split pea on the surface of the frontal bone. When scraped off these left small eroded pits in the bone. The pathologist reported that these small nodules were nests of epithelial cells, obviously malignant. We then remembered the old trouble in her breast. Eventually other growths appeared in different regions of the body and the patient died. If any members have had experience of tumours of the auditory nerve, I hope they will give us the benefit of it.

Mr. W. M. MOLLISON: I have operated upon one case of acoustic nerve tumour. The patient had, I think, the symptoms which Mr. Fraser has described: she was completely deaf in the left ear, had bad attacks of vertigo, and very severe headache. I removed a tumour from the eighth nerve the size of a hazel nut and for the time she made an excellent recovery; after the operation she could hear a whisper 6 in. from that ear; I could never give myself a satisfactory explanation of this. She died with mental symptoms three months later. I went in behind the lateral sinus, in the posterior fossa, and pushed the cerebellum backwards.

Mr. SYDNEY SCOTT: I think all who have had any experience in the kind of work Mr. Fraser has done and the results of which he has laid before the Section to-day, will realize the many hours he must have spent in obtaining and preparing his material. Not only this Section but the Royal Society of Medicine are to be congratulated on the communication which we have received. There is only time to refer to certain details. The changes in the labyrinth which Mr. Fraser describes as "choked labyrinth"—a term which is comparable to the changes seen in the retina—are familiar in sections of the labyrinth in lateral recess tumours. I have some sections showing the same condition. In some cases the auditory neoplasm undoubtedly invades the tympanic coil of the cochlea. I showed one example about ten years ago and regarded it then as indicating the necessity of removing the labyrinth as well as the tumour. There are many intracranial tumours, excluding auditory nerve tumours, which cause a certain degree of deafness difficult to distinguish from that usually assigned to, say "senile nerve deafness," but in the case of brain tumours being associated with high intracranial tension, one has been accustomed to attribute the deafness to high pressure. Using Mr. Fraser's term we may now speak of such conditions as due to "choked labyrinth." In these non-auditory nerve tumours there is only partial deafness and the vestibular reactions are obtainable. On the other hand in assigning the seat of a tumour to the auditory nerve one can only safely do so when the functions of the auditory and vestibular divisions of the eighth nerve are completely defunct. When applied to the investigation of auditory nerve tumours, the real value of the so-called Bárány tests depends upon the certainty with which we obtain either a definitely positive or an absolutely negative reaction. Responses to the caloric tests described as indicative of various degrees of excitability cannot be relied upon in the diagnosis of an eighth nerve tumour. I have now seen nearly thirty cases of tumour involving the auditory nerve in twelve years, and find that complete negativity is the most valuable sign to rely upon so far as auditory and vestibular reactions are concerned. It is true that in the earlier stages, before the true state of affairs can be diagnosed, auditory and vestibular responses are present, and I have known more than one patient who had previously been treated for months by systematic

Eustachian catheterization. This was tolerated the more easily because the fifth nerve became involved and there was total loss of sensation in the nasal cavity. Seeing that defective hearing, tinnitus, and dizziness are common to Eustachian disabilities and auditory nerve tumours, it is well to be guarded in the expression of opinion too soon, and such cases require to be kept under medical observation from time to time. In several cases the tumours have been bilateral, and in some there have been other tumours in the nervous system. I have previously referred to the man with bilateral facial paralysis, absolute deafness to air and bone conduction for all tones, who could tolerate a galvanic current of 20 ma. transcranially without feeling giddy or showing any sign of vertigo or forced movements, owing to total loss of the vestibular functions of both eighth nerves. Mr. Fraser quotes Wittmaack's findings that after division of the eighth nerve Corti's organ degenerated, but the vestibular apparatus was unchanged. I had a case which I have described elsewhere as that of Mrs. C., who had a tumour of the sheath of the auditory nerve which was flattened into a thin band, but the apical coils of the cochlea contained perfect Corti's cells. This is especially interesting because during the two months preceding her death she became progressively deaf to the low tones in the scale. I tested her every ten days up to the day before she died, and the most notable feature was the definite raising of the low limit to aërial conduction in the affected ear. The low limit on each occasion being approximately and successively 30-60-90-120 and so on up to about 400 d.v. I refrain from saying more at present, but the subject is one of great interest and I again express tribute to Mr. Fraser for his admirable work.

Dr. JOBSON HORNE: Can these cases be so rare after all? Mr. Scott says he has seen thirty of them. It may be we see so few because they are not diagnosed. I remember a case the specimen from which was shown in the Otological Congress Museum at the International Meeting in 1898. The patient was a woman, aged 54, and she had a tumour at the internal auditory meatus, partly cystic and partly solid. It puzzled us much at the time, and that is the reason of my recollection of it. The tumour was not much larger than a pea, and the fibres of the nerve were stretched over the tumour. However, she, like the other patients, succumbed, if not to the treatment to the disease. She was insane four years before she died. She was quite deaf on that side and suffered considerable pain. I wish to add my thanks to Mr. Fraser for having brought this subject before us. I would like to hear more about the ætiology of the disease, though we are presented with an excellent summary in these notes; I wish we could always have particulars served in this modern and concise form. I would like to know, for instance, whether these tumours are regarded as congenital in origin. If they are congenital why is it they take so long to develop and eventually to destroy life? I think there is something to be said for the view that meningitis acts as a factor. Until we know more about the origin of these tumours we shall not make progress in their treatment.

Mr. E. D. D. DAVIS: It would be interesting to know how many early cases of auditory nerve tumour one fails to diagnose. In ordinary practice it must be a rare condition, and there is only one specimen of the tumour I have seen in the Museums of the London hospitals, namely, in University College Hospital. Cushing, in his book in 1917, quotes thirty-four cases, and Mr. Sydney Scott tells us he has seen thirty. My experience consists of two doubtful cases in fifteen years in my clinic. With regard to the early diagnosis, a case of unilateral nerve deafness with involvement of the fifth nerve would suggest an acoustic tumour. Occasionally it is difficult to be certain that the loss of hearing is a pure nerve deafness or a middle-ear deafness. I was asked to see a case of extra-cerebellar tumour in the ward this morning. The patient was deaf in the left ear, had nystagmus with possible weakness of the face and muscles of mastication. On doing the rough hearing tests it was difficult to say whether it was a middle-ear or nerve deafness, as in some of these cases there has been middle-ear catarrh or suppuration, and, owing to the existence of nystagmus, the caloric test is inapplicable. I would like to hear from Mr. Fraser, Mr. Scott, or others, what are the early signs of these tumours, as I have the uncomfortable feeling of having missed many acoustic tumours.

Mr. J. S. FRASER (in reply): I wish to express my thanks to the President, and to the meeting generally, for the kind way in which my communication has been received. I was interested in what the President said about the condition of the frontal bone in your case of malignant disease. The erosions in my Case II were caused by what appears to be gliomatous tissue. There were no giant cells in it. Professor Lorrain Smith, to whom I showed the specimens, could not explain why this gliomatous tissue was eating out deep pits in the cranial bones. I congratulate Mr. Mollison upon his case. I think the reason Mr. Sydney Scott has seen so many of these cases is that he is connected with a very large Hospital for Nervous Diseases: in other words, these cases are only diagnosed by the neurologists who then call in the otologists. It ought to be the other way about. Otologists abroad are diagnosing such cases. Quix, of Utrecht, operated by the translabyrinthine route on one of these tumours, while it was the size of a cherry, and was still in its capsule in the internal auditory meatus. If we can diagnose them at that stage we can do something for the patient. If you read the works of Cushing and Henschen you will realize what a hopeless business it is to operate at the "neurological" instead of at the "otological" stage. Cushing hardly pretends to do more than a decompression operation. The patient, if he survives, lives for a year or two, but seldom more. If we could find these cases of unilateral deafness, with giddiness, nystagmus and loss of caloric and galvanic reaction, and if we could have skiagrams so perfected that we could see the dilated internal auditory meatus, we might be able to diagnose them at the "otological," as apart from the "neurological or general surgical," stage. I agree that these cases of unilateral deafness with noises and giddiness should not be sent away and told that nothing can be done for them: they should be kept under observation and asked to report every three or four months if they come from a distance—oftener if they live near the hospital. We may thus learn to diagnose these cases at an earlier stage. I am sorry I am not able to claim "choked labyrinth" as my own phrase: the credit is due to a foreign observer, whose name I do not at the moment remember. I agree with Mr. Sydney Scott in his difficulty regarding such expressions as "hyper- or hypo-excitable labyrinth." We know where we are when we have to deal with an absolutely normal labyrinth or a quite functionless one. Well-recorded cases of tumour of the auditory nerve, however, show that all such patients are not necessarily totally deaf, and that there is not of necessity a total loss of vestibular irritability. Therefore it is not going to be an easy matter to diagnose these tumours in the very earliest stage. Loss of the lower tones is very important. If you have a case of unilateral nerve deafness with raised lower tone limit and diminution or loss of vestibular reaction the presumption in favour of acoustic tumour seems to be pretty strong. In the ordinary case of nerve deafness the patient can hear the C_{32} fork—e.g., cases of senile nerve deafness. I have gone carefully into the literature of most of the recorded cases of eighth nerve tumour and find that in several this raising of the lower tone limit is reported. It was present in two of my cases. There is also a lowering of the upper tone limit, producing a concentric narrowing of the auditory field. As to the pathology of the labyrinthine affections I have shown on the screen, there are three possibilities: (1) There is first the question of an infective process—a labyrinthitis coming from the middle ear or from the meninges. In the first case the histological changes were very like those produced by a former labyrinthitis. There was some evidence of former otitis media on both sides. On one side there was filling up of the hollow spaces of the inner ear by new connective tissue and bone formation, while on the other side there were similar changes in the labyrinth and also an auditory nerve tumour. (2) The tissue present in the labyrinth may be tumour growth due to infiltration along the nerve or vascular paths. If this is so operation by the translabyrinthine route is the only method of curing the case. (3) The third possibility and the most likely one is that we have to deal with a labyrinth "choked" in the early stages by a fibrin network, and, later on, by a new formation of connective tissue and bone.

Atresia of Left External Auditory Meatus.

By H. Bedford Russell, F.R.C.S.

(Introduced by Mr. H. Buckland Jones).

Male, aged 24. Right ear used to run in childhood. Operations at the ages of 7 and 12 years. Left ear has never run. Hearing in the left ear is remarkably good: conversational voice is heard at 6 ft.

Weber's test is referred to right. Rinne's test negative both sides. Loss of bone conduction very slight.

I should be glad of opinions on the origin of the condition and on the advisability of treatment, if any.

DISCUSSION.

The PRESIDENT: Five years ago I showed a stout woman with a bilateral condition of the same kind, which dated from the time she had scarlet fever as a child. There were concentric pin-point openings at the junction of the bony and cartilaginous meatuses. Even after the length of time referred to there was great improvement in her hearing and relief from the tinnitus by inflation of the ears with a catheter.

Mr. LAYTON: At the Pensions Aural Board I saw an exactly similar case of atresia with pin-hole orifices on either side, with suppuration beneath on one side but not on the other. The skin appeared to be quite normal down to the hole, and showed no evidence of scarring. From touching it with a probe it seemed that in one area there were little masses of bone, as though underlying osteomata were present. I thought the condition was a congenital one.

Mr. SYDNEY SCOTT: I think this case is one of atresia due to cicatrization of the external auditory canal, for the skin is puckered at the isthmus and is almost insensitive to the probe. That the condition is acquired seems the more likely from the fact that the patient has had middle-ear suppuration on the other side for which the radical mastoid operation has been performed.

Mr. W. M. MOLLISON: I have tackled one of these cases. I opened the mastoid and took away bone, then cut a flap as for a mastoid, and skin-grafted the whole area. The patient did fairly well.

Mr. LAWSON WHALE: I tried to follow the treatment adopted by the French in two or three cases, and do an operation through the meatus for traumatic cases immediately after the war. I do not think they can be satisfactorily dealt with through the meatus. I find that with a flap they do all right.

Mr. BEDFORD RUSSELL (in reply): I am grateful for having been given the opportunity to show the case. I brought it forward in the hope of obtaining advice as to treatment, or, indeed, as to whether treatment is indicated. He can hear ordinary conversation at 6 ft., and he says he is well content with it. I feel he could be improved by having the diaphragm removed from the meatus. And I believe it could be attacked through the meatus and the raw area covered by inserting a skin-graft over a wax mould of the meatus. I have used that method in two cases of almost complete atresia following gunshot wound, and it was successful. I agree that this is the result of a pathological change, and that it is not congenital.

Case of (?) Vertigo.

By W. M. Mollison, M.Ch.

J. L. F., aged 32, was referred to me by Dr. Cohen, of Aldershot, in May, 1919, on account of attacks of vertigo. He has had attacks of vertigo since 1918 when he was in the Army; usually two a week but has had five in a week. Objects appear to move; the patient cannot stand; he has vomited. Attacks less frequent lately. Deafness in left ear, perhaps coinciding with onset of vertigo. Tinnitus, throbbing. Membranes dull but normal inclination. Tubes clear, Wassermann reaction negative.

May, 1919: Examination showed the left ear almost completely deaf, though the highest notes of the monochord were heard.

June, 1919: Examination of the labyrinth showed left labyrinth was functional, caloric test produced slight reaction with past pointing.

August, 1919: Lumbar puncture; fluid clear but under considerably increased pressure. Re-testing of labyrinths: Caloric right normal response, vertigo, nystagmus and past pointing; left vertigo, very little nystagmus, marked past pointing with both arms. Examination of the nervous system; no abnormality found (Dr. Johnson). The lumbar puncture improved the condition and the patient was free from attacks for six weeks.

May, 1920: Attacks have recently been more severe; three weeks ago a bad attack; fell in his shop. Examination, left ear, hears whisper at 1 in. (with noise apparatus); hearing varies. Labyrinth, left, syringing in cold water—vertigo, nystagmus—past pointing, left arm, not right. Response sluggish. Right the same, vertigo, nystagmus, past pointing with right arm, not left.

Should the left labyrinth be operated on?

Destruction of the Tympanic Membrane by Hot Fluid.

By Lionel Colledge, F.R.C.S.

The patient upset a cup of hot tea over her right ear four months ago. When first seen two months ago there was a great deal of discharge and swelling of the meatus. This condition has improved under treatment with liquor carbonis detergens, leaving a perforation including almost the whole of the membrana tensa. In Weber's test the sound is referred to the left ear. Rinne's test is positive and there is diminished bone conduction. Nystagmus is produced almost immediately by irrigation with cold water. Patient complains very much of tinnitus in the right ear since the accident. Bromide has little or no effect on the tinnitus.

DISCUSSION.

Mr. J. F. O'MALLEY: I think there is an old perforation in the right ear, one unlikely to be caused by something hot entering the ear, and that idea is supported by the fact that if you look at the membrane on the other side you see changes which suggest that at an earlier period there was inflammatory or suppurative trouble. The ear is doing fairly well, but tinnitus is the symptom most complained of, and in the presence of that our armamentarium is limited. One tries fairly large doses of acid hydrobrom. dil., but I do not think I have ever been convinced that it does much good for the condition. Sometimes I have had better results from blistering the skin over the mastoid, but that is not an infallible cure either.

Mr. E. M. WOODMAN: I would like to repeat a remark Mr. Somerville Hastings made to me when we were examining this case—namely, that he had had a similar case in which he had syringed the ear with too hot a fluid, and he concluded that the auditory nerve is peculiarly sensitive to heat. His patient became stone deaf soon after that accident.

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Section of Pathology.

President—Dr. W. Bulloch, F.R.S.

A Bodo-like Flagellate persisting in the Urinary Tract for Five Years, the Urine remaining Bacteriologically Sterile throughout.[1]

By Arthur Powell and A. J. Kohiyar.

During the past seventy years four observers have described as a human parasite a flagellate organism found in the urine. The first three found the flagellate in decomposing urine only, never in urine freshly drawn with aseptic precautions. The fourth, Sinton, on two occasions only, found flagellates closely resembling those now reported. The urine at the first examination was twenty-four hours old and alkaline; the second observation was made on neutral urine six hours after it was passed. He always failed to find flagellates in urine passed with aseptic precautions.

Pathologists were therefore hitherto fully justified in their scepticism as to the parasitic nature of these protozoa which they regarded as probable aërial contaminations. In 1914 we had the fortune to have under observation a patient whose urine constantly contained a large number of flagellates when freshly drawn with all antiseptic precautions. This year, 1919, though the man seems in perfect health his urine still contains these flagellates. He has been examined almost daily in April and May, and at longer intervals up to September. On most days we find two to four parasites in each field of the one-sixth inch objective with centrifuged urine. On a few occasions they are much more numerous. On rare occasions several fields must be searched to find a parasite. The flagellates generally live for four to eight hours after the urine is passed. They die when decomposition sets in.

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[1] At a meeting of the Section, held November 4, 1919.

K., a Parsi clerk aged 35, in good health up to March 29, 1914, felt some pain on that date in the right loin, and found the next urine resembled milk in appearance. He consulted us next day, March 30, when we found he was suffering from chyluria. In addition to a few *Filaria nocturna* embryos and the usual contents of chylous urine we observed numerous flagellates actively moving in the urine. These flagellates continued to be found daily, even when the urine was drawn off or passed with all aseptic precautions into sterile vessels. Urine so drawn, was added to agar or broth with uniformly negative results as regards bacterial or fungal growth. At intervals the urine contained many oxalate crystals. The night blood contained the embryos of *Filaria nocturna*. The patient remained under constant observation till November, 1914, when the chyluria ceased, but the flagellates persisted in undiminished numbers giving rise to no harmful symptoms.

In April, 1919, he appeared in perfect health, but as a matter of interest we again examined the urine and found the flagellates present in large numbers in almost daily examinations for a month, as well as on other less frequent examinations up to September, 1919. At no examination have they been absent.

In 1919 there was a striking change in the activity and perhaps in the morphology of some of the parasites as compared with 1914. In 1914 the parasites were exceedingly lively. There was considerable locomotion with almost constant rotation of the body and rapid lashing of the flagella. The lashing of the flagella was so rapid that both we and many medical men who kindly examined them were in doubt as to their number. In some of the organisms one flagellum at times trailed backward in corkscrew fashion giving rise to an illusory resemblance to an undulant membrane. Most of the parasites were oval or globular, a few were turnip-shaped with a suspicion of a short posterior rod. In 1914 a very common occurrence was to find a congress of three, vigorously jerking or butting against one another—flagellar end towards the centre—perhaps an attempt at conjugation. Both of us completely failed to fix or stain the protozoan in 1914.

We are indebted to many colleagues for examining and attempting to fix the flagellates, but their success was unfortunately no greater than ours. We specially thank Major Taylor, Colonel Glen Liston, I.M.S., Dr. Turkhud and other members of the staff of Parel Laboratory; also Dr. F. D. Bana.

In 1919 the flagellates are almost all globular or oval in shape; only very few show any tapering at the posterior end. They are now much more sluggish than in 1914. Locomotion is almost absent; wriggling and rotation is relatively infrequent. The parasite keeps up a fairly brisk lashing of the flagella but pauses at intervals so that they can be counted without difficulty. As a rule there are two and only two flagella, but on some days a proportion of the parasites possess four, all arising from the same point. These are doubtless in an early stage of fission. In 1919 we can see nothing like an undulant membrane and only very rarely a suspicion of a rod. The body is from 12 μ to 16 μ in diameter; the flagella from 18 μ to 24 μ in length.

Dr. Kohiyar has now succeeded in fixing the animals. Our previous failures were due to allowing the cover-glass preparations to partially dry. Fixation must be attempted at once while the smear is quite fluid. In this process the vast majority of the protozoa are washed away, but the few that become fixed do so without distortion and can be well stained with iron-hæmatin. A rather large and central nucleus is thus demonstrated and the flagella stain well.

We are greatly indebted to Miss Muriel Robertson who has taken much interest in the case. She pointed out that flagellates are common intruders in the intestine. (In India one of us (P.) has found them with great frequency in cases of diarrhœa.) In this case the chyluria is proof of a round-about avenue from the intestine via the lacteals to the urinary tract, along which the flagellates may have originally migrated.

That the flagellates could travel alone unaccompanied by any of the intestinal flora, such as *Bacillus coli*, is a remarkable fact and points to a selective inhibitory action of the tissues.

As regards the variation in the flagellate's habits and motility in the course of five years, we would suggest that if the animals be emigrants from the intestine, a considerable power of locomotion was necessary to enable them to swim round solid particles and seek their food in the far-from-homogeneous contents of the intestine. In the fluid homogeneous urine the faculty of locomotion is little needed and the parasite has in the course of hundreds of generations become adapted to its environment. The apparent morphological variation was partly error in observation. We have little doubt that the undulant membrane was an illusion due to a free flagellum trailing along the margin of the body. Until we saw

Sinton's paper we considered it possible that more than one species of flagellate was present. Sinton, adapting the nomenclature of Fantham, Stephens, Nuttall, Castellani and others, calls his flagellate a *Prowazekia*. He found considerable variation in shape as did we in our earlier specimens. That this variation is not specific was proved by oval mother-cells dividing into one globular and one carrot-shaped daughter-cell. Professor Kohiyar only succeeded in staining our parasite a few days before I left India and I have made only a brief examination of the stained specimens. We observed no contractile nor food vacuole; no cytostome. There was a large, nearly central, nucleus. I cannot recall having seen any kinetonucleus. Sinton found a large pear-shaped kinetonucleus. The above-named authors say that if there be a kinetonucleus the flagellate should be classified as a *Prowazekia*—if absent, as a *Bodo*.

The nomenclature of the flagellates is at present of so unstable a character that we will not go further than calling the one in question a *Bodonid*.

BIBLIOGRAPHY.

HASSALL, A. H. *Lancet*, 1859, ii, p. 503.

SALISBURY, 1868, and KÜNSTLER, 1883, in Blanchard's "Zoologie Medicale," i.

SINTON. *Ann. Trop. Med. and Parasitol.*, 1912, vi, p. 245.

Section of Pathology.

President—Dr. W. BULLOCH, F.R.S.

The Nature of Serum Antitrypsin and its Relation to Autolysis and the Formation of Toxins in Infection and in Anaphylaxis.[1]

By F. H. TEALE and E. BACH.

From the Research Laboratories, University College Hospital Medical School, London.[2]

(I) NATURE AND PROPERTIES OF ANTITRYPSIN.

(*A*) *The Antitrypsin of Egg White.*—(*a*) Relation to lipoids; (*b*) isolation and properties of antitrypsin.

(*B*) *Serum Antitrypsin.*—(*a*) Action of lipoid solvents on (1) dried serum, (2) ordinary serum; (*b*) endeavours to isolate serum antitrypsin; (*c*) soaps as antitrypsin agents; (*d*) inhibition of antitrypsin by (1) acids and alkalis, (2) temperature, (3) incubation; (*e*) adsorption of antitrypsin; (*f*) mode of action of antitrypsin; (*g*) antitrypsin in albumen fraction of serum.

(II) AUTOLYSIS OF THE SERUM.

(*a*) Result of incubation without destruction of the antitrypsin; (*b*) result of incubation after destruction of the antitrypsin; (*c*) sensitiveness of the ferment to (1) de-antitrypsinizing reagents, (2) reaction, (3) temperature; (*d*) adsorption of the ferment.

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[1] At a meeting of the Section, held December 2, 1919.

[2] The expenses of this research were defrayed by a grant by the Medical Research Committee.

(III) AUTOLYSIS OF SOLID TISSUES.

(*A*) *Liver.*—(*a*) Importance of complete bacteriological sterility; (*b*) effect of reaction; (*c*) effect of bacteria; (*d*) effect of phosphorus; (*e*) effect of bacterial toxæmia.

(*B*) *Leucocytes and Marrow.*

(IV) RELATION OF ANTITRYPSIN TO TISSUE AUTOLYSIS.

(*a*) Is there a tissue antitrypsin?

(*b*) Influence of serum antitrypsin in tissue autolysis: (*a*) liver, (*b*) marrow, &c.

The relation of the above experiments to (*a*) bacterial toxæmia, (*b*) anaphylaxis, (*c*) anaphylotoxin, (*d*) parasitism and pathogenesis of bacteria will be discussed later in a following paper dealing with the variations of antitrypsin in disease and anaphylaxis.

SERUM ANTITRYPSIN AND AUTOLYSIS.

The following experiments on antitrypsin and autolysis were carried out preliminarily to an investigation into the variation of antitrypsin in bacterial infection and anaphylaxis and its relationship to the symptoms of toxæmia.

Various investigators have been struck by the uniformity of the main manifestations of bacterial toxæmia in animals of the same species and have come to the conclusion that the symptoms are due not to specific bacterial endotoxins but to a non-specific poison formed (1) either from the bacteria themselves by the action on them of antibodies and complement (Teale and Embleton [1], Vaughan [2]); or (2) from the plasma of the infected animal (Jobling and Petersen [3]). These last authors believe that bacteria can adsorb antitrypsin and so allow the plasma proteolytic enzymes to give rise to proteose-like toxic bodies from the animal's own body fluids. Further Wright [4] in his work claims to have shown that certain bacteria can only grow in the tissues, &c., and produce toxins when the local adsorption of the serum antitrypsin is sufficiently great to allow of auto-digestion of the tissue fluids, and the consequent production of sufficient split products to allow these bacteria to grow.

Lastly, our experiments on the changes in the nitrogen content of the blood in bacterial infection showed a great increase of non-coagulable nitrogen, chiefly, in fact almost wholly, in the form of urea, and so it became necessary to investigate the influence of the serum antitrypsin on tissue autolysis; and also to determine whether a special tissue antitrypsin was present or not.

HISTORICAL.

Hildebrandt [5] made the first observation that the unheated sera of all animals and also egg albumen possess a marked resistance to the action of proteolytic enzymes. Hahn [6] made a similar observation a few years later. According to Levine and Sankey [7] this antitryptic property is also possessed by the various tissues and organs of the body. Weinland [8] demonstrated that certain intestinal worms contained a powerful antitrypsin, to which he attributed their ability to live bathed in the intestinal digestive juices. This observation was confirmed by Hamill [9] who isolated the antitrypsin of intestinal worms and studied its properties. He showed that the antitrypsin was not a lipoid or a lipoidal combination. The antitrypsin did not destroy the trypsin but inhibited its action for a time until it was itself destroyed by the trypsin. Cathcart [10] showed that the antitrypsin is connected with the albumen fraction of the serum.

With regard to the nature of antitrypsin there are various views:—

(1) That it is a true antibody in the sense of Ehrlich—(Achalme [11], Jochmann and Kantorowicz [12], Mayer [13], Eisner [14], and Wiens [15]). Mayer states his belief that the antitrypsin is an antibody provoked by the ferments of the cells as antigens. Eisner [14] and Wiens [15] believe that the antigen is the leucoprotease of the polymorphonuclear cells. Many observers claim to have been able to produce anti-enzymes by inoculating animals with trypsin, pancreas, &c. The results are very contradictory and have to be discarded because of the action of the precipitins provoked against the proteins in the antigens used. Delezenne and Pozerski [16] state that the antitrypsin exerts its effect chiefly by combining with the kinase activating trypsinogen. Bayliss and Starling [17] oppose this view and believe that the antitrypsin acts directly upon the trypsin.

(2) Rosenthal [18] also suggested that the inhibition was due to the presence of proteoclastic cleavage bodies in the sera used. Bayliss [19], Abderhalden and Gigon [20] found that some of the products of tryptic digestion inhibited the further action of the enzyme to some extent. Later Walters [21] showed that this occurs only to a slight extent and

that the inhibition by serum is far too great to be accounted for in this way.

(3) That the antitrypsin is lipoidal in nature.

Schwarz [22] and Sugimoto [23] believe that the antitryptic action is closely related to the lipoids in the serum. They found that by treating sera with ether, benzol, olive oil, &c., the antitryptic power of the serum was greatly diminished or lost. Delezenne and Pozerski [16] found that shaking sera with chloroform destroyed their antitryptic properties. Later Jobling and Petersen [24] and others, observed the same and came to the conclusion that the antitryptic power was due to the serum lipoids and that the power could be restored by saponifying the lipoid extracted with chloroform, &c., and adding this soap to the extracted de-antitrypsinized serum. They further believed that the antitryptic power of the soap is proportional to the degree of desaturation of the fatty acid in the soap.

Cobliner [25] and Mayer [13] showed that extraction of dried sera with chloroform, ether and petroleum ether did not interfere with the antitryptic power. Jobling and Petersen [24] explain this by saying that the extraction is more effective when carried out with wet than with dried sera.

THE METHODS ADOPTED FOR DETERMINING THE ANTITRYPTIC POWER OF A SERUM.

(*a*) It was found after many experiments that the following rapid method gave results closely comparable to the much longer process detailed below, and in addition allowed of expressing a "unit"—a quantity of serum capable of inhibiting the liquefaction of a standard quantity of gelatine by a standard of trypsin.

The unit of trypsin was determined by finding the smallest amount of a 1 per cent. suspension of trypsin, which caused complete liquefaction of 1 c.c. of 15 per cent. gelatine at 37° C. in thirty minutes, all the reagents being at 37° C. before mixing. After incubation, the tubes containing the mixtures were cooled at 10° C. for thirty minutes and the amount of trypsin causing complete liquefaction determined.

The antitryptic titre of a serum was determined by adding graded quantities of the serum (usually diluted 1 in 5) to a series of tubes containing the unit of trypsin, incubating at 37° C. for thirty minutes, then adding 1 c.c. of 15 per cent. gelatine at 37° C. and incubating for thirty minutes longer at 37° C., cooling as above, and reading the unit as the smallest amount completely interfering with the liquefying action of the unit of trypsin.

(*b*) Graded quantities of the serum were added to 1 c.c. of a 1 per cent. trypsin suspension, and made up with sterile normal saline to 2 c.c. and kept for thirty minutes at 37° C., then 3 c.c. of a 2 per cent. solution of casein in 0·2 per cent. sodium carbonate were added, and the whole incubated for three hours.

The controls were (*a*) casein and trypsin incubated without serum, and (*b*) casein and trypsin without incubation made up to 5 c.c.

All the tubes were after incubation brought rapidly to the boiling point. A measured quantity was taken from each tube (2 c.c.) mixed with 15 c.c. of colloidal iron, 10 c.c. of water (N free), and 1 c.c. of sodium sulphate solution, to precipitate the coagulable nitrogen. The mixture was filtered through two wet filter papers, the filtrate was collected. A measured portion of the filtrate was taken, concentrated and then decarbonized with pure sulphuric acid. The nitrogen content was then determined by Nesslerization. The results were then plotted as curves.

This method of estimating non-coagulable nitrogen was recommended by Dr. Plimmer, and we found that it gave results identical with the other methods used, but proved more rapid and far simpler.

(I) THE NATURE AND PROPERTIES OF ANTITRYPSIN.

(*A*) *The Antitrypsin of Egg White.*

Our first experiments were made with the white and yolk of eggs, and from them it will be seen that egg white has very marked antitryptic properties, whereas the yolk, in spite of its great lipoid content, is not antitryptic in the slightest degree. The suspension of egg white was made by dissolving up 1 grm. of vacuum dried egg albumen in 100 c.c. of normal saline. The yolk suspension was made from a fresh egg and was used in a dilution of 1 in 10 in normal saline and pure.

Experiment 1.—The egg white suspension was added to the unit of trypsin in graded quantities, starting with 10 c.mm. of the 1 in 100 dilution. The mixture was then incubated in the water bath at 37° C., for thirty minutes, when the gelatine was added as above.

10	20, 30, 40, 50, 60, 80, 100 c.mm. of 1 per cent. egg white
Almost complete inhibition of liquefaction.	Complete inhibition of liquefaction.

With the egg yolk in similar fashion:—

1 in 10.		Pure.
10, 20, 40, 60, 80, 100, 200, 400 c.mm.	...	60, 80, 100, 200 c.mm.

Complete liquefaction—i.e., no inhibition exercised by the egg yolk.

Several other experiments gave identical results. Thus egg yolk contains no antitrypsin in spite of its great lipoid content, whereas egg white, which has relatively little lipoid in its constitution, is distinctly antitryptic.

Experiment 2.—Finely powdered vacuum dried egg white was extracted with various lipoid solvents. To ensure complete dryness the powdered egg albumen was dried over sulphuric acid *in vacuo*. After extraction the extracted egg albumen was dissolved in normal saline, the solvent having been previously completely removed by decantation and drying *in vacuo*. 0·2 grm. of egg albumen was extracted in each case and the residue then taken up in 20 c.c. of normal saline.

		Complete inhibition of unit of trypsin by
(*a*) Control; unextracted ..	0·2 grm. in 20 c.c. of normal saline; then diluted 1 : 5 ...	60 c.mm. = 12 c.mm. (undiluted)
(*b*) Treated for twenty-four hours with perfectly dry ether ...	ditto ditto ...	60 c.mm. = 12 c.mm. (undiluted)
(*c*) Treated for twenty-four hours with petroleum ether ...	ditto ditto ...	60 c.mm. = 12 c.mm. (undiluted)
(*d*) Treated for twenty-four hours with absolute alcohol and then with absolute ether ...	ditto ditto ...	60 c.mm. = 12 c.mm. (undiluted)

Experiment 3.—Similar to Experiment 2.

	Complete inhibition of tryptic unit by
(*a*) Control; 1 per cent. diluted 1 : 5 ...	80 c.mm. = 16 pure; almost complete by 14 pure.
(*b*) Extracted twenty-four hours with dry ether ...	80 c.mm. = 16 pure.
(*c*) Extracted twenty-four hours with petroleum ether ...	80 c.mm. = 16 pure.
(*d*) Extracted twenty-four hours with absolute alcohol, then ether ...	90 c.mm. = 18 pure; almost complete by 16 pure.

Other experiments yielded similar results, except that occasionally, owing to imperfect drying, the ether or alcohol and ether caused some degree of coagulation of the egg albumen with corresponding loss in the antitryptic power. These experiments show that the removal of lipoids from dried egg albumen does not affect the antitryptic power of the egg white, unless the reagent has caused some degree of coagulation.

Similar treatment of a suspension of egg albumen leads to a very different result, the antitryptic power becomes completely lost. All the lipoid solvents used on a suspension of egg albumen cause a certain degree of coagulation of the protein.

Experiment 4.—A 1 per cent. suspension of vacuum dried egg albumen in normal saline exposed to different lipoidal solvents for three hours at 37° C. The egg suspension and solvent were thoroughly shaken at intervals. The solvent was removed as far as possible by decantation, the residual solvent was finally removed by exposure *in vacuo.*

	Unit of trypsin inhibited by
(*a*) Control	20 c.mm. of the suspension
(*b*) After exposure to ether	500 c.mm. ,, ,,
(*c*) ,, ,, chloroform	not inhibited by 500 c.mm.

The same result was obtained by leaving the mixture at the room temperature for three hours.

Experiment 5.—Same as Experiment 4, only kept at room temperature for three hours.

	Unit of trypsin inhibited by
(*a*) Control	20 c.mm. of the suspension
(*b*) After exposure to ether	400 c.mm. ,, ,,
(*c*) ,, ,, chloroform	not inhibited by 500 c.mm.

Other experiments gave similar results.

Jobling [24] and his co-workers obtained results similar to those of Experiment 4. They concluded that the difference between the results of the action of the lipoid solvent on a watery suspension and the dried powder *re* antitryptic destruction was due to the lipoid solvent being able to break down the lipoid-protein combination in the suspension but not in the dried state, and thus more effectually remove from the suspension the lipoids to which they ascribe the antitryptic power of a serum. They took no notice of the coagulation of the protein which occurs constantly as the result of the action of the lipoid solvent on the watery suspension, and which will destroy the antitryptic power if it occurs even when the egg white has been dried.

Experiment 6: Isolation of Antitrypsin from Egg White.—In this experiment it was endeavoured to extract antitrypsin from carefully vacuum-dried egg white. It was found later that with different commercial specimens of dried egg white the experiment was not always successful, and that this depended on the method of drying adopted.

Hamill [9] showed that the antitrypsin could be extracted from intestinal worms by treatment with 50 per cent. alcohol. We accordingly took vacuum-dried egg white, ground it up in a coffee-mill, and added two volumes of 50 per cent. alcohol, and kept the mixture at 56° C. for twenty-four hours. The supernatant alcohol was then decanted, and the residue squeezed out in a press. The fluids were mixed and filtered. A clear yellowish fluid was then obtained. This was evaporated down at 56° C., and yielded a yellowish resinous-looking residue. It was purified by adding 50 per cent. alcohol, which caused the greater part to go into solution. The solution was filtered, re-evaporated at 56° C., and again treated with 50 per cent. alcohol, and the process repeated several times. Thus the original alcoholic extract was freed from coagulable protein. This purified extract was easily soluble in normal saline, and 20 c.mm. of a 0·1 per cent. solution caused complete inhibition of the unit of trypsin as determined above. This antitryptic material was also easily soluble in distilled water. It was soluble in alcohol up to 60 per cent., above this it was precipitated, but immediately went into solution on dilution. It had the following properties: It was free from coagulable protein; it gave a xanthoproteic reaction with nitric acid, also a rose-red biuret reaction with copper sulphate and caustic soda; it was precipitated by 50 per cent. ammonium sulphate; its antitryptic properties were in no way affected by treatment with lipoidal solvents such as chloroform, ether, alcohol; it was non-dialysable, on dialysis some salt and carbohydrate diffused away, but the antitryptic power was not impaired. The purified fat sugar and salt free antitrypsin was therefore a colloid and had protein characters.

Experiment 7: Action of various Re-agents on Prepared Antitrypsin.—A 0·1 per cent. suspension in normal saline was used in all the tests in this experiment.

(*a*) *Action of Lactic Acid.*

		Made up to			
	Control	n/25	n/20	n/15	n/10
The unit was contained in	20 ...	20 ...	20 ...	20 ...	25 c.mm. of the suspension

(*b*) *Boiling with Acid and Alkali.*

	Control	Control boiled 15 minutes in water bath	Made up to n/20 lactic acid and boiled as before	Made up to n/20 alkali and treated as before
Unit contained in	15 ...	20 ...	20 ...	Not in 200 c.mm.

(*c*) *Adsorption.*—Quantities of 5 c.mm. of the antitryptic suspension were exposed to the various reagents for two hours at 37° C.; the mixtures were periodically shaken. The agar was added in the solid state and then cooled to gelatine before incubation. The mixtures were centrifuged; correction made for dilution (uncorrected figures in brackets).

	Control	200 mg. kaolin	n/20 lactic acid	1 c.c. of 0·25 per cent. starch	0·5 c.c. of 0·5 per cent. agar	*Bacillus coli*	200 mg. blood charcoal
Unit in	20 c.mm.	20 c.mm.	(25 c.mm.) =21 c.mm.	(25 c.mm.) =21 c.mm.	(25 c.mm.) =22 c.mm.	20 c.mm.	20 c.mm.

Exposure to the same re-agents in the cold gave similar results.

From these experiments we conclude that:—

(1) The antitryptic power of egg albumen is not due to lipoids because: (*a*) Egg white does not lose its antitryptic properties when treated with lipoid solvents unless they cause more or less coagulation of the protein; (*b*) prepared dry antitrypsin is unaffected by lipoid solvents; (*c*) egg yolk which is rich in lipoids is absolutely non-antitryptic.

(2) The antitryptic power appears to be due to a body which is (*a*) a non-diffusible protein substance; (*b*) not destroyed by boiling or by the action of dilute acids; (*c*) destroyed by weak alkalis; (*d*) soluble in 60 per cent. alcohol but is precipitated by alcohols of higher concentration, but not coagulated, going into solution directly the concentration is lowered; (*e*) precipitated by 50 per cent. ammonium sulphate; (*f*) not absorbed or adsorbed by charcoal, kaolin, &c., agar starch or bacteria; (*g*) not affected by treatment in the dry state with lipoid solvents.

(*B*) *Antitrypsin of Blood Serum.*

Having thus isolated the antitrypsin from egg, similar means were adopted to endeavour to isolate it from blood serum.

Experiment 8.—For this purpose the serum was dried *in vacuo* over sulphuric acid. This did not affect its subsequent complete solubility. The dried serum was then treated with 50 per cent. alcohol, but it was impossible to extract an antitryptic substance by this method. Similar experiments were made with concentrations of alcohol varying from 40 to 80 per cent. but without any success. The direct method of extraction having failed we next worked by exclusion, investigating the results of the extractions of the lipoids from the sera experimented with, and also the antitryptic effects of these lipoids and their soaps.

Experiment 9.—Treatment of sera with various lipoidal solvents in the wet state destroys the antitryptic power of the serum. (*a*) 20 c.c. of rabbit serum were repeatedly thoroughly shaken up with 40 c.c. of chloroform and kept at 37° C. for one hour. At the end of the time the mixture was centrifuged and the supernatant clear serum withdrawn, and all traces of chloroform removed by exposure *in vacuo*. The deposit was a thick one consisting of chloroform and coagulated protein. The original titre of the serum by the gelatine method was 60 c.mm. After chloroform extraction the serum did not contain this unit in 500 c.mm. Similar experiments gave the same results: destruction of the antitryptic power. (*b*) Ether: 20 c.c. of rabbit serum were thoroughly repeatedly shaken with 40 c.c. of ether and kept at 37° C. for three hours (renewing the ether periodically). At the end of the time there were three layers—a bottom of coagulated protein, a middle of clear serum and a top of ether. The latter was removed, the clear serum pipetted off and exposed *in vacuo* to remove the ether.

Titre before ......60 c.mm. inhibited the unit of trypsin.
Titre after ......500 c.mm. did not inhibit the unit of trypsin.

Experiment 10.—Same experiment as Experiment 9, only the mixture was kept at the room temperature. (*a*) 20 c.c. of rabbit serum were repeatedly shaken up in 40 c.c. of chloroform and kept at the room temperature for three hours. At the end of the time there was a heavy deposit consisting of chloroform emulsion and coagulated albumen and a somewhat turbid upper layer. The latter was pipetted off, the chloroform removed *in vacuo* and the bulk restored by the addition of normal saline. Before treatment, 60 c.mm. inhibited the unit of trypsin. After treatment, 500 c.mm. did not inhibit the unit of trypsin. (*b*) Serum similarly treated with ether. After treatment 500 c.mm. did not inhibit the unit of trypsin. Hence we must conclude that shaking and extracting liquid sera with lipoidal solvents such as chloroform or ether destroys the antitryptic power of the serum, but it must be remembered that these reagents not only dissolve out some of the lipoids but also profoundly alter the proteins, precipitating and coagulating a portion of them. These experiments also show that the antitryptic loss is not proportional to the amount of lipoid extracted since the loss of power is about the same if the extraction be carried on at 37° C. or at room temperature.

Experiment 11: Extracting Dried Sera with various Lipoid Solvents.—Carefully measured quantities of sera were evaporated in vacuo in very thin layers in shallow dishes. The dried serum was finely powdered and again dried *in vacuo*. Extraction was then performed. After extraction the solvent was carefully fiftered off through a small very fine filter paper. The filter paper and residue were dried *in vacuo* and then treated with normal saline. (*a*) 5 c.c. of guinea-pig's serum was used in each test. After extraction the dried residue was made up to 5 c.c. with normal saline.

	Unit of trypsin inhibited by
Control, not evaporated	40 cmm.
,, dried and re-dissolved	40 ,,
Serum dried, extracted with chloroform at 37° C. for three hours	40 ,,
,, ,, ,, ether at 37° C. for three hours	40 ,,
,, ,, ,, petroleum ether at 37° C. for three hours	40 ,,

(*b*) Rabbit serum: 5 c.c. used in each test. After extraction the total residue was dissolved in 10 c.c. of normal saline.

	Unit of trypsin inhibited by
Control; dried only	100 c.mm. = 50 c.mm. pure
Extracted with chloroform	100 ,, = 50 ,, ,,
Extracted with petroleum ether	120 ,, (almost C. I. with 100 c.mm.) = 60 c.mm. pure
Extracted with xylol	100 c.mm. = 50 c.mm. pure

(*c*) Rabbit serum: Quantities as (*b*) only made up to original bulk.

	Unit of trypsin inhibited by
Control; dried	30 c.mm. of serum
Extracted with absolute alcohol at 37° C. for six hours	30 ,, ,,
Extracted with absolute alcohol for three hours, then chloroform for four hours at 37° C.	30 ,, ,,

(*d*) Rabbit serum: 10 c.c. in each test. Carefully dried as above. Residues taken up in 50 c.c. of normal saline.

	Unit of trypsin inhibited by
Control; dried only	48 c.mm. pure
Extracted with absolute alcohol for twenty-four hours, then with chloroform for six hours; after evaporation of the solvent it was found that the solution in the solvent was very turbid	titre 72 c.mm. pure

So that in spite of some coagulation of the protein the antitryptic power was 66 per cent. of the control.

(*e*) Rabbit serum dried *in vacuo* in quantities of 2 c.c. in very large plates so as to get a very thin film of dried serum.

	Unit of trypsin inhibited by
(1) Control dissolved in 10 c.c. of saline	160 c.mm. = 32 pure
(2) Treated with absolute alcohol sealed up for twenty-four hours, alcohol evaporated, then chloroform for four hours, all at 37° C.	160 c.mm. = 32 pure
(3) Treated with alcohol alone for twenty-four hours at 37° C.	160 c.mm. = 32 pure

(*f*) Same as (*e*) only taken up in 5 c.c. of normal saline.

	Unit of trypsin inhibited by
(1) Control; dried serum	70 c.mm. = 28 pure
(2) Extracted with absolute alcohol for twenty-four hours at 37° C.	70 c.mm. = 28 pure
(3) Extracted with alcohol for twenty-four hours, then chloroform for six hours at 37° C.	80 c.mm. = 32 pure
(4) Chloroform extraction only, twenty-four hours ...	70 c.mm. = 28 pure

These experiments undoubtedly show that lipoidal solvents at 37° C. do not remove the antitryptic power from dried sera. Further, some of the methods used, alcohol followed by chloroform extraction, not only dissolve out the uncombined lipoids but also the bound lipoids. This disposes of the objection put forward by Jobling [24] to explain the difference between the results obtained when fresh and dried sera are extracted. Jobling states that in the wet state the combined lipoids are split off from their combination by chloroform or ether and so extraction in this state leads to a greater removal of lipoids than when the serum is dried. Besides being contrary to usually accepted facts the following experiment shows the assumption to be wrong.

Experiment 12.—Equal quantities of ox serum were taken. (1) One portion was dried *in vacuo*, ground up finely, extracted with chloroform at 37° C. for twenty-four hours. (2) The other was repeatedly shaken up with 2 vols. of chloroform and kept at 37° C. for twenty-four hours. The mixture was centrifuged, the deposit taken up—treated with absolute alcohol and ether to clear and precipitate the albumen. The extracts (1) and (2) were then evaporated and boiled with 20 c.mm. of N/10 alcoholic potash, and excess of alkali determined.

(1) 20 − 3·5 c.c. = 16·5 c.c. N/10 alkali were required to saponify the fat extracted.
(2) 20 − 8·0 c.c. = 12·0 c.c. N/10 alkali were required to saponify the fat extracted.

Showing that more fats are dissolved out by chloroform extraction of dried serum than fresh serum.

Finally the method of Cathcart [26] for completely removing the lipoids from a serum was carried out.

Experiment 13.—(*a*) 5 c.c. of guinea-pig's serum were cooled to 0° C., then poured into 40 c.c. of absolute alcohol at −14° C. and left for one hour, filtered in cold filter at the same temperature, washed with absolute dry ether at −8° C., then extracted with boiling dry ether for twenty-four hours.

Titre of serum (50 per cent.)	50 c.mm. = 25 pure
Titre of extracted serum (50 per cent.)...	50 c.mm. = 25 pure

(In spite of small loss of material during the process.)

(*b*) Similar experiment to last.

Titre before	...	30 c.mm.	Titre after	...	70 c.mm.

(But here there was great loss of precipitate during transference to filter paper and there was some coagulation.)

(*c*) Similar experiment.

Control	...	...	20 c.mm.	After extraction	...	25 c.mm.

(Here again there was loss of material due to manipulation.)

(*d*) 5 c.c. of rabbit serum was treated as above. The residue was taken up in 10 c.c. of normal saline.

Titre before extraction (50 per cent.) = 100 c.mm. = 50 pure.
Titre after extraction (50 per cent.) = 100 c.mm. = 50 pure.

(*e*) 10 c.c. of guinea-pig's serum was treated as above, very carefully; there was very little loss of precipitate during the experiment and the precipitate after complete extraction readily went into solution. The serum and precipitate were made up to 50 c.c.

Before treatment the serum inhibited unit of trypsin in 120 c.mm. = 24 pure.
After treatment the precipitate inhibited unit of trypsin in 120 c.mm. = 24 pure.

Hence although we have not succeeded in isolating serum antitrypsin we have been able definitely to prove that it is not lipoidal in nature contrary to the results of nearly all previous workers. Complete removal of the lipoids without interfering with the solubility of the protein in no way affects the antitryptic titre. Hence we must conclude that the antitryptic power of the serum is not due either to the free or combined lipoids.

(*C*) *Soaps as Antitryptic Agents.*

Jobling [24] in support of his view that the serum lipoids are antitryptic shows that the extracted lipoids when saponified are antitryptic. He further states that the antitryptic power of a soap depends upon the degree of desaturation of its fatty acids. Our experiments go to prove that all soaps destroy trypsin, no matter how unsaturated the fatty acids are. Their action is quite different from that of true antitrypsin. As will be seen later there is not much difference in the amount of inhibition exercised by a given quantity of serum if the experiment is performed in two stages or in one, viz., if the serum is allowed to act on the trypsin for some time before the gelatine is added or if all three are added simultaneously. In the case of the soaps there is a great difference in the amounts necessary to produce inhibition if the experiment is performed in two stages or one, i.e., the soaps depend for their antitryptic action on the degree of concentration.

Experiment 14.—Suspensions of the following soaps were prepared and the amounts necessary to inhibit the unit of trypsin were determined: (*a*) with the soap, trypsin and gelatine incubated simultaneously; (*b*) with the soap and trypsin incubated together for thirty minutes at 37° C. first, and then the gelatine added.

	(*a*)	(*b*)	Iodine number of soap
5 per cent. sodium oleate	400 c.mm. inhibit unit of trypsin	30 c.mm. inhibit unit of trypsin	86
5 per cent. sodium linoleate soap	500 c.mm. inhibit unit of trypsin	50 c.mm. inhibit unit of trypsin	176
5 per cent. sodium stearate soap	500 c.mm. do not inhibit unit of trypsin	10 c.mm. inhibit unit of trypsin	78
2½ per cent. sodium stearate soap	500 c.mm. do not inhibit unit of trypsin	20 c.mm. inhibit unit of trypsin	78
5 per cent. croton oil soap	500 c.mm. inhibit unit of trypsin	10 c.mm. inhibit unit of trypsin	104
5 per cent. sodium linoleate soap (another specimen)	Ditto	30 c.mm. inhibit unit of trypsin	188
Ditto, completely saturated with iodine (none free)	Ditto	Ditto	*Nil*
2½ per cent. soap made from chloroform extract of dried ox serum	Ditto	20 c.mm. inhibit unit of trypsin	—

These soaps were all prepared by Mr. Reeve, B.Sc., of the Department of Physiological Chemistry, University College.

In connexion with this the following experiment is interesting in bringing out the difference between the mode of action of antitrypsin and soap.

Experiment 15.—(*a*) The different constituents in the experiment were incubated together; (*b*) The antitrypsin and trypsin were incubated together first for thirty minutes at 37° C., then the gelatine added.

	(*a*)	(*b*)
(1) Serum; unit of trypsin inhibited by	20 c.mm. ...	16 c.mm.
(2) ,, ,, ,, ,, ...	20 ,, ...	16 ,,
(Very marked inhibition by 16 c.mm.)		
(3) 5 per cent. sodium oleate (iodine No. 86); unit of trypsin inhibited by	100 ,, ...	10 ,,
(4) 5 per cent. sodium linoleate soap; unit of trypsin inhibited by	300 ,, ..	20 ,,

These experiments are at variance with those of Jobling [24] and show: (1) that the soaps act quite differently from the serum antitrypsin—the effect of concentration of the former is very apparent, in the latter this is not apparent to any marked degree; (2) that the desaturation of the fatty acids in the soap makes no appreciable difference; (3) that the soaps from the uncombined lipoids act quite well antitryptically—and according to Jobling it is the combined lipoid which is antitryptic; (4) that the soaps destroy trypsin in virtue of the hydroxyl-ion concentration.

The following experiment, using the casein digestion method, brings out the slight difference in the action of antitrypsin if allowed to act alone in the trypsin first or if all constituents are added together.

Experiment 16.

	50 c.mm serum	100 c.mm. serum
(*A*) Control N.C.N. determined trypsin and casein and serum; no incubation	1·9 mgr.	1·95 mgr. of N.
(*B*) Serum—trypsin and casein incubated together for three hours at 37° C.	4·4 mgr.	4·3 mgr.
(*C*) Serum — trypsin incubated for thirty minutes at 37° C., then casein added and incubated for three hours at 37° C.	3·9 mgr.	3·9 mgr.

(1 c.c. of 1 per cent. trypsin, 3 c.c. of 2 per cent. solution of casein used in each case. All made up to 5 c mm.)

The separate incubation makes a difference of about 13 per cent., thus agreeing with the gelatine experiments.

(D and E).—Inhibition of Antitrypsin, Adsorption of Antitrypsin in Serum.

We have previously shown that lipoidal solvents destroy the antitrypsin in fluid sera, but find that this is due to the precipitation of proteins, and not due to the removal of the fats The following experiments show the effect of acids and alkalis on serum antitrypsin. It was, however, first necessary to find the limits of acids or alkali concentration interfering with tryptic action. We accordingly exposed the trypsin to acids and alkalis of various strengths, and tested the result with gelatine liquefaction.

Experiment 17.—(*a*) 200 c.mm. of 1 per cent. trypsin (the unit causing complete liquefaction of 1 c.c. of 15 per cent. gelatine in thirty minutes at 37° C.), and 1 c.c. of gelatine were put into a series of tubes and the reaction was adjusted as follows. Incubation for thirty minutes at 37° C. was allowed. Then readings taken as usual.

	n/10	n/20	n/50	n/100	n/120	n/200	n/250	n/500
Acid ...	CL	ACI	CL	CL	CL	CL	CL	CL
Alkali ...	SI	CL	CL	CL	CL	CL	CL	CL

Thus n/20 acid interferes distinctly with trypsin.
n/10 alkali only to a slight degree.

(*b*) As before, a mixture of gelatine and 1 per cent. trypsin was made in the proportion of 200 c.mm. of the latter to 1 c.c. of the former. The reaction was then adjusted as below.

	n/5	n/7	n/10	n/11	n/13	n/14	n/16	n/20	n/25	n/33	n/50	n/100
Lactic acid	CI	CI	CI	CI	CI	CI	CI	CI	SI	CL	CL	CL
Alkali ...	CL	CL	CI	ACI	SI	CL	CL	CL	CL	CL	CL	CL

CI = Complete inhibition
ACI = Almost complete inhibition
SI = Slight inhibition
CL = Complete liquefaction
} from action of alkali.

Experiment 18 : Action of Lactic Acid on Serum Antitrypsin.—(1) 2 c.c. of rabbit serum in each test incubated for three hours at 37° C., with acid as follows: then the antitryptic titre was taken as before.

Reaction	n/66	n/50	n/40	n/30	n/30	n/28	n/22	n/20	n/10 acid
Amount of serum inhibiting unit of trypsin ...	50	50	60	60	60	70	200	250	300 c.mm.

(2) Similar experiment. Lactic acid, three hours' incubation at 37° C. as above.

Reaction	n/100	n/80	n/60	n/60	n/20
Amount of serum inhibiting the unit of trypsin ...	60	60	60	60	300 c.mm.

Experiment 19 : Rate of Destruction of Antitrypsin by Lactic Acid.—From Experiment 18 it was seen that n/20 lactic acid concentration destroyed antitrypsin in the serum. The following experiments show rate of destruction.

	Control	SERUM MADE UP TO n/20 LACTIC ACID.					
		Immediately after acidification	Five minutes later*	Fifteen minutes later*	Twenty minutes later*	Twenty-five minutes later*	Thirty-five minutes later*
Quantity of serum inhibiting unit of trypsin	70	90	150	150	250	300	500 c.mm.

* In water bath at 37° C.

Experiment 20 : Action of Alkali.—Rabbit serum made up to the following concentration NaOH incubated for three hours at 37° C., then tested. (1)

	n/100	n/80	n/60	n/40	n/20	Control	
Amount of serum inhibiting unit of trypsin	60	60	60	60	70	60	c.mm.

(2) Another specimen :—

Alkali	n/100	n/50	n/20	Control	
Three hours at 37° C. ...	80	80	200	80	c.mm.
Twenty hours in cold ...	80	80	200.	80	,,

Experiment 21 : The Titre cannot be brought back on Neutralization.—(1) Rabbit serum made up to n/20 lactic acid, incubated for forty-five minutes in water bath at 37° C., then neutralized with N/1 NaOH.

	Control	After ¾ hour's incubation in n/20 acid	After neutralization
Amount of serum inhibiting unit of trypsin	60	400	400 c.mm.

A deposit was formed on neutralization. This was taken up and was found to have no antitryptic power.

(2) Rabbit serum was made alkaline with NaOH to the desired degree, incubated for twenty minutes in the water bath at 37° C., then neutralized.

Amount of serum inhibiting the unit of trypsin—

	Control	n/20	n/10	n/5	
Before neutralization ..	40	80	200	500	c.mm.
After ,, ...	—	80	200	500	,,

Experiment 22 : Effect of Temperature on Antitrypsin.—(1) Rabbit serum incubated in the water bath at :—

Control	Amount of serum inhibiting the unit of trypsin	
	56° C.	60° C.
0 minutes	40 c.mm. ...	40 c.mm.
15 ,, ...	40 ,, ...	80 ,,
30 ,, ...	50 ,, ...	100 ,,
60 ,, ...	60 ,, ...	160 ,,
90 ,, ...	60 ,, ...	200 ,,
180 ,, ...	80 ,, ...	400 ,,

(2) Another experiment. Rabbit serum kept at—

Control	Amount inhibiting the unit of trypsin	
	56° C.	60° C.
0 minutes	30 c.mm. ...	30 c.mm.
30 ,, ...	30 ,, ...	120 ,,
60 ,, ...	30 ,, ...	120 ,,
2 hours	30 ,, ...	300 ,,
3 ,, ...	30 ,, ...	300 ,,
4 ,, ...	30 ,, ...	400 ,,

Experiment 23: Effect of Incubation on Antitrypsin in Bacteriologically Sterile Serum.—(1) Sterile rabbit serum.

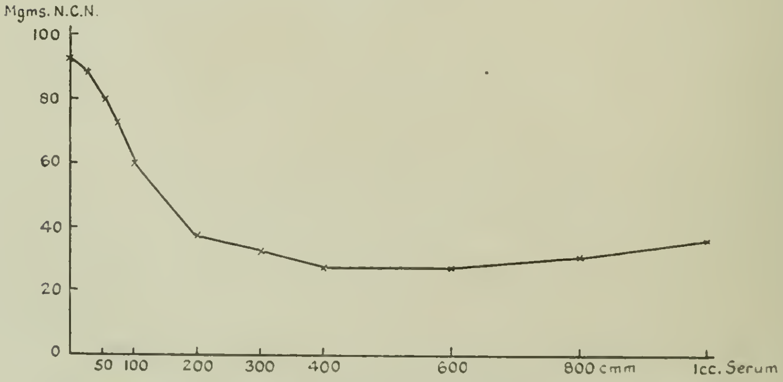
	Control	72 hours	120 hours
Unit of trypsin inhibited by ...	30 ...	30 ...	30 c.mm. of serum

(2) Dog's serum.

	Control	4 hours' incubation	24 hours	14 days
Unit of serum inhibited by ...	60 ...	50 ...	50 ...	50 c.mm. of serum

Experiment 24.—During the course of the previous experiments, where the casein method was used as a control, it came out that the amount of inhibition by serum antitrypsin was not proportional to the amount used, and even when apparently a large excess had been used some digestion still occurred. Thus in the following experiment graded quantities of guinea-pig serum (diluted 1 in 5, and pure as required) were added to 1 c.c. of 1 per cent. trypsin, and all the tubes made up to 2 c.c. with normal saline incubated at 37° C. for thirty minutes, then 3 c.c. of a 2 per cent. casein solution added; digestion two hours at 37° C.

Amount of serum	Non-coagulable nitrogen
0 c.c. ...	= 92 mgr. per cent.
0·025 ,, ...	= 88 ,, ,,
0·05 ,, ...	= 80 ,, ,,
0·075 ,, ...	= 72 ,, ,,
0·1 ,, ...	= 60 ,, ,,
0·2 ,, ...	= 38 ,, ,,
0·3 ,, ...	= 32 ,, ,,
0·4 ,, ...	= 28 ,, ,,
0·6 ,, ...	= 28 ,, ,,
0·8 ,, ...	= 30 ,, ,,
1 ,, ...	= 32 ,, ,,
Control trypsin and casein, no incubation ...	= 24 ,, ,,



Experiment 24.—To show inhibition by varying amounts of antitrypsin.

(*G*) *Experiment 25 : The Protein Fraction of the Serum containing the Antitrypsin.*—Serum treated with Am_2 SO_4.

	Titre		True
(1) 33 per cent. saturation to precipitate euglobulin—euglobulin solution	600 c.mm.	...	300 c.mm.
(2) 50 per cent. saturation to precipitate englobulin and pseudoglobulin, solution of the two	300 ,,	...	150 ,,
(3) Solution of pseudoglobulin and albumen from (1), after precipitation of euglobulin	100 ,,	...	50 ,,
(4) Solution of albumen from (2)	240 ,,	...	120 ,,
(5) Control, diluted 50 per cent.	80 ,,	...	40 ,,

· All made up to double the volume of the original serum.

Experiment 26.—Similar experiment.

	Titre		True titre
(1) Control	50 c.mm.	...	25 c.mm.
(2) Control + equal amount of saturated ammonium sulphate	100 ,,	...	25 ,,
(3) Euglobulin precipitate with 33 per cent. ammonium sulphate	not 400 ,,	...	– ,,
(4) Albumen after precipitation of rest with 50 per cent. ammonium sulphate	200 ,,	...	100 ,,
(5) Euglobulin + pseudoglobulin preparation from (4)	300 ,,	...	150 ,,
(6) Albumen + pseudoglobulin, after (3)	90 ,,	..	45 ,,

Hence the euglobulin fraction is not antitryptic, but the pseudoglobulin and albumen fractions are. These experiments are in agreement with those of Cathcart [10] who stated that the albumen fraction was antitryptic.

(II) AUTOLYSIS OF SERUM.

All the sera used for the following experiments were collected with the necessary aseptic precautions. Sera showing evidence of contamination at any stage of the experiment were rejected. Non-coagulable nitrogen was determined as before (*see* p. 9.)

(*A*) *Incubation of Sera without Previous Destruction of the Antitrypsin.*

Experiment 1.

	Antitryptic titre		Non-coagulable nitrogen
(*a*) Rabbit serum—			
Control	60 c.mm.	...	32 mgr. per cent.
After four hours at 37° C.	50 ,,	...	32 ,, ,,
After twenty-four hours at 37° C. ...	50 ,,	..	32 ,, ,,
(*b*) Rabbit serum tinged with hæmoglobin—			
Control	40 ,,	...	32 ,, ,,
After seventy-two hours at 37° C. ...	40 ,,	...	34 ,, ,,
After 120 hours at 37° C.	40 ,,	...	40 ,, ,,

Experiment I (continued).

	Antitryptic titre	Non-coagulable nitrogen
(*c*) Dog's serum—		
Control	... 30 c.mm.	... 45 mgr. per cent.
After 120 hours at 37° C. ...	... 30 ,,	... 45 ,, ,,
(*d*) Dog's serum—		
Control	... 30 ,,	... 52·5 ,, ,,
Seventy-two hours at 37° C.	... 30 ,,	... 52·5 ,, ,,
(*e*) Sheep's serum—		
Control	... 60 ,,	... 32 ,, ,,
Seventy-two hours at 37° C.	... 60 ,,	... 32 ,, ,,
Fourteen days at 37° C. ...	... 50 ,,	... 32 ,, ,,
(*f*) Sheep's serum, tinged with hæmoglobin—		
Control	... 50 ,,	... 40 ,, ,,
Fourteen days at 37° C. ...	... 50 ,,	... 52 ,, ,,
(*g*) Sterile guinea-pig's serum—		
Control	... 20 ,,	... 34 ,, ,,
Twenty-four hours at 37° C.	... 20 ,,	... 34 ,, ,,
Seventy-two hours at 37° C.	... 20 ,,	... 34 ,, ,,

From these experiments it is obvious that the serum antitrypsin inhibits any proteolytic autolytic ferment in the serum. It, however, does not interfere with the autolytic ferment in the red cells. Antitrypsin is therefore also not destroyed by the proteolytic ferment as it is by trypsin.

(*B*) *Effect of Destruction of the Antitrypsin.*

Experiment 2.—(*a*) Sterile rabbit serum was made N/20 lactic acid by adding the requisite amount of n/1 lactic acid, then incubated.

	Antitryptic titre	Non-coagulable nitrogen
Control, before lactic acid ...	... 60 c.mm.	... 32 mgr. per cent.
Eight days at 37° C., after lactic acid	... not in 500 c.mm.	... 32 ,, ,,
Twelve ,, ,, ,, ,,	... ,, ,,	... 32 ,, ,,

(*b*) Sterile rabbit serum: Destruction of antitrypsin by heat and acid.

	Antitryptic titre	Non-coagulable nitrogen 24 hours	Non-coagulable nitrogen 72 hours
Control	60 c.mm.	... 36	... — mgr. per cent.
Made n/20 lactic acid	not 500 c.mm	... 36	... 36 ,, ,,
Inactivated at 60° C.	,, ,,	.. 36	... 36 ,, ,,
Inactivated and made n/20 lactic acid	,, ,,	... 36	... 36 ,, ,,

(*c*) Sterile dog's serum.

	Non-coagulable nitrogen
Control	45 mgr. per cent.
Incubated five days	45 ,, ,,
Made n/20 acid and incubated five days	45 ,, ,,

(*d*) Sterile guinea-pig serum.

	Antitryptic titre	Incubated 24 hours Non-coagulable nitrogen	Incubated 96 hours Non-coagulable nitrogen
Control...	20 c.mm.	32	— mgr. per cent.
Made n/20 lactic acid	not 500 c.mm.	32	32 ,, ,,
Inactivated at 60° C. for forty-five minutes	,, ,,	32	32 ,, ,,

Hence after destroying the antitrypsin by (*a*) lactic acid, (*b*) heating, there is no evidence of autodigestion.

Experiment 3.—(*a*) Sterile rabbit serum was heated at 37° C. with chloroform as for destruction of antitrypsin. The chloroform was removed and the serum evaporated *in vacuo* sterilely to remove last traces of chloroform, made up to original bulk with normal saline.

	Antitryptic titre	Non-coagulable nitrogen
Control	not in 500 cmm.	32 mgr. per cent.
Incubated three days at 37° C.	,, ,,	,, ,,

(*b*) Similar experiment with guinea-pig serum.

	Antitryptic titre	Non-coagulable nitrogen
Control, after treatment with chloroform	300 c.mm.	34 mgr. per cent.
Incubated three days at 37° C.	300 ,,	34 ,, ,,

(*c*) Sterile sheep's serum was treated with (*a*) chloroform at 37° C., (*b*) ether at room temperature, (*c*) iodine, to destroy antitrypsin. In the case of (*a*) and (*b*) the solvent was removed, and the serum evaporated *in vacuo* to get rid of the last traces, then made up to the original volume with normal saline. Each specimen was sterile by tests before and after incubation.

	Ether serum	Chloroform serum	Iodine serum
Non-coagulable nitrogen—			
Before incubation	40	40	35 mgr. per cent.
Seventy-two hours at 37° C.	40	40	35 ,, ,,
120 hours at 37° C.	40	40	35 ,, ,,

Antitryptic titre not in 500 c.mm. Just in 500 c.mm.

From these experiments it is seen that autodigestion of the serum does not occur when the antitrypsin is destroyed by (*a*) n/20 lactic acid, (*b*) chloroform at 37° C., (*c*) ether at room temperature (sheep serum), (*d*) iodine. This may be due to (*a*) absence of an autolytic ferment, (*b*) destruction of the ferments by the reagents used to destroy the antitrypsin.

(C) (1) To investigate the latter point the following experiments were performed:—

Experiment 4.—(*a*) Sterile rabbit serum was treated with: (i) Chloroform in the cold (room temperature) shaking and leaving for two hours; (ii) ether in the cold. The solvents were removed, the sera evaporated to dryness *in vacuo* to remove the solvents completely, then made up to original volume with sterile normal saline.

	Antitryptic titre	NON-COAGULABLE NITROGEN		Increase
		Incubated 24 hours at 37° C.	Incubated 72 hours at 37° C.	
Control	60 c.mm.	34 mgr. (not incubated)	—	—
Chloroform serum ...	500 ,,	36 mgr.	37 mgr.	10 per cent.
Ether serum	500 ,,	34 ,,	34 ,,	—

(*b*) Sterile guinea-pig's serum, treated as in (*a*).

Control	20 c.mm.	32 mgr.	—	—
Chloroform serum ...	500 ,,	38 ,,	38 mgr.	19 per cent.
Ether serum	500 ,,	36 ,,	36 ,,	12 ,,

(*c*) Sterile dog serum, treated as above.

Control	30 c.mm.	45 mgr.	—	—
Chloroform (room temperature)	not in 500 c.mm.	52 ,,	52 mgr.	15 per cent.
Chloroform (37° C. for one hour)	,, ,,	48 ,,	48 ,,	6 ,,
Ether (room temperature)	,, ,,	48 ,,	48 ,,	6 ,,

(*d*) Human serum, treated as above.

Control	40 c.mm.	28 mgr.	—	—
Chloroform serum (room temperature)	not in 500 c.mm.	29·5 ,,	30 mgr.	—
Ether	,, ,,	30 ,,	30 ,,	—

These reagents only act by destroying the antitrypsin and have no direct activating effect on the ferment.

Experiment 5.—Equal quantities of sterile guinea-pig serum were evaporated *in vacuo* and ground up to a fine powder and extracted with: (*a*) chloroform at room temperature, (*b*) chloroform at 37° C., (*c*) ether, (*d*) alcohol and then chloroform for twenty-four hours. The solvents were removed and the dried extracted residues were taken up in normal saline to original bulk and incubated for twenty-four hours at 37° C.

Antitryptic titre	Non-coagulable nitrogen	
	Before incubation	After incubation
(*a*) 20 c.mm.	32 mgr. ..	32 mgr. per cent.
(*b*) 20 ,, ...	32 ,, ...	32 ,, ,,
(*c*) 20 ,, ...	32 ,, ...	32 ,, ,,
(*d*) 20 ,, ...	32 ,, ...	32 ,, ,,

At the same time similar portions (*e*) and (*f*) of the serum after treatment as (*a*) and (*b*) were dissolved in normal saline and treated with chloroform at room temperature and then the solvent removed in the usual way.

(*e*)	First at 37° C.	not in 500	32	38
(*f*)	First at room temperature	,, ,,	32	38

Hence it is only the destruction of the antitrypsin which activates the serum ferment. Further, the experiment shows that the ferment is not inactivated by chloroform at 37° C. when the serum is dried.

Effect of Reaction on the Ferment.

Experiment 6.—Sterile guinea-pig's serum was treated as above with chloroform at the room temperature for the destruction of antitrypsin. After complete removal of the solvent by drying *in vacuo* the serum was dissolved in sterile saline and the reaction adjusted with standard sterile alkali and acid (N/1, N/10, N/20) to make the following concentrations and then incubated at 37° C. for twenty-four hours.

	Non-coagulable nitrogen									
	Control	n/20	n/40	n/80	n/100	n/200	n/400	n/600	n/800	
Acid (lactic) ...	32	32	32	32	32	32	34	38	38	mgr. per cent.
Alkali (NaOH) ...	32	32	32	32	32	32	32	38	39	,, ,,

From this it will be seen that the autolytic ferment is very sensitive to reaction, acid or alkali. About n/400 either acid or alkali appears to destroy the ferment, hence the reason why the ferment cannot be demonstrated when the antitrypsin is destroyed by acid or alkali; the necessary concentration n/20 is injurious to the ferment.

(*C*) (3) *Effect of Heat.*

Experiment 7.—Sterile guinea-pig's serum deantitrypsinized as above by chloroform in the cold. Heated at 56° C. and 60° C. for various periods and then incubated at 37° C. for twenty-four hours and the N.C.N. determined.

Exposure	30 minutes	60 minutes	90 minutes	120 minutes	4 hours	
At 56° C. ...	34	34	34	34	34	mgr. non-coagulable nitrogen per cent.
At 60° C. ...	34	34	34	34	34	,, ,, ,,

Control 34 mgr. non-coagulable nitrogen per cent. and incubated for twenty-four hours, 39 mgr. non-coagulable nitrogen per cent.

Hence the autolytic ferment shows the slightly greater sensitiveness to temperature than antitrypsin does.

(D) Adsorption of the Enzyme.

It was found that if the deantitrypsinized serum (by chloroform in the cold) was exposed to sterile blood charcoal, 0·5 per cent. suspension of agar solution, or kaolin, that digestion did not take place.

Experiment 8.—Guinea-pig's serum was deantitrypsinized by chloroform in the cold, repeatedly shaken. The chloroform was removed, the serum made up to original bulk by addition of sterile saline.

	Antitryptic titre	NON-COAGULABLE NITROGEN	
		Before incubation	After 24 hours at 37° C.
(*a*) Serum alone	not in 500 c.mm.	32 mgr. per cent.	38 mgr. per cent.
(*b*) Exposed to blood charcoal, then centrifuged (100 mgr. to 10 c.c.)	,, ,,	32 ,, ,,	32 ,, ,,
(*c*) Exposed to agar (1 c.c. of 0·5 per cent. agar solution, 10 c.c. of serum mixed at 37°C., then iced to gelatine)	,, ,,	30 ,, ,,	30 ,, ,,
(*d*) Exposed to kaolin (200 mgr., then centrifuged to 5 c.c.)	,, ,,	32 ,, ,,	32 ,, ,,

This experiment is especially of interest and importance in conjunction with the results obtained *re* the antitryptic adsorption and the production of anaphylotoxin *in vitro* by the same substances.

Jobling's [24] experiments showed adsorption of antitrypsin, rise in N.C.N. and production of anaphylotoxin; our experiments show that although a potent "anaphylotoxin" can be obtained by exposing guinea-pig's serum to agar, &c., there is (1) no adsorption of antitrypsin by the reagent; (2) no rise in N.C.N.; (3) an adsorption of the proteolytic autolytic ferment. Hence these experiments are completely at variance with those of Jobling, and show that the "anaphylotoxin" is produced without any evidence of autodigestion and by substances which do not cause the adsorption of the antitrypsin. Hence we conclude that (1) there is a weak proteolytic autolytic ferment in the serum of the guinea-pig, less in the other animals experimented with (human, sheep, rabbit, dog); (2) the autolytic ferment is easily destroyed by (*a*) slight variation in the reaction (n/400); (*b*) the reagents which destroy antitrypsin; (*c*) and by heating (56° C.). The importance of this lies in the relation of this ferment to "anaphylotoxin" production *in vivo* and *in vitro* and the so-called protective ferments of Abderhalden [27] which will be discussed later.

The following experiment is of great importance *re* the activity of the autolytic proteolytic ferment of the serum *in vivo.*

Experiment 9.—A rabbit was inoculated subcutaneously with 1 c.c. of oleum phosphoratum B.P. Previous experiment had shown that this causes the antitryptic titre to fall almost to *nil* a few hours before the death of the animal. The animal, as already mentioned elsewhere, suffered no pain from the inoculation and continued to eat well till shortly before it was killed. The serum of this animal gave the following results.

		Non-coagulable nitrogen—		
Antitryptic titre		Unincubated	Incubated 24 hours	Incubated 4 days
500 c.mm.	...	78 mgr. per cent.	78 mgr. per cent.	78 mgr. per cent.
Another similar experiment—				
300 c.mm.	...	52 ,, ,,	52 ,, ,,	52 ,, ,, (72 hours' incubation)*

Hence in the rabbit in phosphorus poisoning where the antitrypsin is almost destroyed there is no evidence of activity of an autolytic proteolytic ferment in the serum.

(III) AUTOLYSIS OF SOLID TISSUES, &c.

This investigation was carried out because we desired (1) to determine whether there was any difference in the rate of autolytic degradation of the solid tissues of animals dying from bacterial toxæmia and those of normal animals. The possibility that such differences might occur was suggested by our experiments in which we noted a great increase in the non-coagulable nitrogen in the blood of infected animals, the increase being however almost wholly in the form of urea. The experiments of Jacoby [28] show increased autolytic degradation in phosphorus poisoning; (2) to determine whether serum antitrypsin had any influence on tissue autolysis. The experiments under this head therefore resolved themselves into (*a*) the confirmation or otherwise of the current conception of the rate of autolytic degradation of tissues under conditions of absolute bacteriological sterility; (*b*) to determine whether "antitrypsin" is present in the tissues and if serum antitrypsin has any influence on tissue autolysis.

The great difficulty in these experiments was to obtain the tissues absolutely bacteriologically sterile. In spite of absolute bacteriological sterility in removal and manipulation, it was found that bacteria were present in practically every specimen used. It appears that the liver, especially under normal conditions, contains bacteria, probably in the bile passages only, of the nature of *Bacillus coli* and *Bacillus Welchi*. These bacteria, as will be seen later, have a marked influence on autolytic degradation and account for the current idea of the rapidity of autolytic degradation of the tissues. In order to obtain material bacteriologically sterile, the tissues used were removed as carefully as possible *re* asepsis and were then ground up with sterile normal saline in sterile silica tubes with pebbles, by continued shaking for six hours in a machine shaker—on the principle of the pulverization of bacteria to obtain the "endotoxin." In this way perfectly sterile specimens were obtained for experimentation. N.C.N. was determined by the method previously mentioned.

(*A*) *Autolysis of the Liver.*

Experiment 1.—An emulsion of normal rabbit's liver was made in normal saline. The emulsion was made about 10 per cent. to compare with the experiments of Schryver [29]. It was found to be bacteriologically sterile.

	Non-coagulable nitrogen
Not incubated ...	8·4 mgr. per 100 c.c. of emulsion
Incubated 4 hours	8·4 ,, ,, ,,
,, 6 ,,	8·4 ,, ,, ,,
,, 8 ,,	8 4 ,, ,, ,,
,, 12 ,,	8·4 ,, ,, ,,
,, 24 ,,	9·6 ,, ,, ,,

Other experiments gave similar results. Rise in twenty-four hours, 7 to 14 per cent. N.C.N.

Experiment 2.—An emulsion of normal rabbit liver was made in normal saline, about 20 grm. in 100 c.c. The liver emulsion proved to be sterile bacteriologically—aërobic and anaërobic tests.

	Non-coagulable nitrogen
(*a*) Control not incubated	17·4 mgr. per 100 c.c. of emulsion
(*b*) ,, incubated 24 hours	18·6 ,, ,, ,,
(*c*) ,, ,, 48 ,,	20·4 ,, ,, ,,

Increase in 24 hours = 7 per cent.
,, 48 ,, = 17 per cent.

Experiment 3.—Another emulsion same strength as Experiment 2.

	Non-coagulable nitrogen
(*a*) Control not incubated	24 mgr. per 100 c.c.
(*b*) ,, incubated 72 hours	29 ,, ,, ,,
	= 20 per cent. increase

Acidity at end of 72 hours = n/100 lactic acid

Experiment 4.—A 20 per cent. emulsion of rabbit's liver. Sterile by tests.

	Non-coagulable nitrogen
Control	24 mgr. per 100 c.c.
Incubated 24 hours	27 ,, ,, ,,
,, 72 ,, ...	31 ,, ,, ,,

Increase in 24 hours = 12·5 per cent.
,, 72 ,, = 28·0 per cent.

Experiment 5.—An experiment—one success amongst many failures—where whole pieces of liver were incubated and proved to be bacteriologically sterile. After incubation the pieces, with exuding fluid, &c., were weighed and ground up in normal saline to make emulsions of equal strength (10 per cent.).

	Non-coagulable nitrogen
Before incubation	68 mgr. per 100 gms. of liver
After 30 hours at 37° C.	84 ,, ,, ,,
,, 4 days ,, ...	132 ,, ,, ,,
,, 6 ,, ,, ...	144 ,, ,, ,,

Increase in 30 hours = 24 per cent. of non-coagulable nitrogen = n/50 acidity.
,, 96 ,, = nearly 100 per cent. ,, ,, = n/40 ,,
,, 144 ,, = 110 per cent. of ,, ,, = n/40 ,,

Experiment 6.—An emulsion of liver was made in the usual way with very little saline. It proved to be quite sterile. It was incubated at 37° C. for fourteen days.

Non-coagulable nitrogen
Control, not incubated = 64 mgr. per cent. of the emulsion.
Incubated 14 days = 132 ,, ,, ,,
= over 100 per cent. increase in fourteen days.
Acidity = n/40.

(B) *Effect of Added Lactic Acid.*

Experiment 7.—A 20 per cent. emulsion of rabbit's liver was made in normal saline in the usual way. It proved to be bacteriologically sterile. Sterile N/1 lactic acid was added to make the following concentrations. All tests were made up to same bulk with normal saline. Incubation two hours at 37° C.

(1) Control	Not incubated	24 mgr. non-coagulable nitrogen per cent. of emulsion.	
	incubated	24 mgr.	= 0 per cent. rise.
(2) Made n/100 lactic acid		30 ,,	= 25 ,, ,,
(3) Made n/50 ,, ,, ...		33 ,,	= 37 ,, ,,
(4) Made n/40 ,, ,, ...		36 ,,	= 50 ,, ,,
(5) Made n/20 ,, ,, ...		42 ,,	= 75 ,, ,,
(6) Made n/10 ,, ,, ...		42 ,,	= 75 ,, ,,
(7) Made n/20 lactic acid left at room temperature for twenty minutes, then neutralized and incubated		24 ,,	= 0 ,, ,,

Other experiments gave similar results.

From this experiment it will be seen (1) that the autolytic degradation is proportionate to the degree of acidity and (2) that there is no tissue antitrypsin of the nature of serum antitrypsin since every addition of acid causes increased degradation. There is no degree of acidity below which degradation does not occur and above which it does, as would be the case if a serum antitrypsin were present. Further, the n/20 acid experiment, Experiment 7, No. 7, shows that the autolytic degradation is not due to the destruction of antitrypsin since the degradation has been inhibited by neutralization. Our previous experiments have shown that antitrypsin destroyed by acid cannot be restored by neutralization, so that if the tissue autolysis were solely inhibited by an antitrypsin of this nature, degradation should have taken place, as any such antitrypsin would have been completely destroyed by this treatment. These experiments show further that under absolutely sterile conditions the autolytic degradation of the liver is slow, and this is due to the relatively low concentration of lactic acid in the liver under sterile conditions.

(*c*) The following experiments show how bacterial infection hurries on the process of autolytic degradation, chiefly as the result of the acidity produced by the bacteria from the sugar of the liver and to a less extent due to the proteolytic activity of the infecting organisms.

Experiment 8.—A sterile emulsion of liver 10 per cent. was treated as follows and incubated for four hours :—

	Sterile	n/10 lactic acid	Infected *Bacillus coli* (broth culture ½ c.c. to 10 c.c. of emulsion)
Control ...	5·76	4·8	6·72
Incubated ...	5·76	22·1	9·6

milligrammes non-coagulable nitrogen of emulsion.

Lactic acid causes 500 per cent. rise in four hours.
Bacillus coli ,, 50 ,, ,, ,,

Experiment 9.—Similar to above. Four hours' incubation; 10 c.c. of the emulsion in each experiment.

	Sterile and 1 c.c saline	Staphylococci (1 c.c. active broth culture)	Streptococci (1 c.c. active broth culture)	*Bacillus Welchi* (1 c.c. active broth culture)	*Bacillus coli* (1 c.c. active broth culture)
Control	5·6	5·8	5·8	5·8	5·8
Incubated four hours	5·6	5·8	5·8	11·4	9·2

milligrams non-coagulable nitrogen per cent.

Bacillus Welchi alone gives an increase of 68 per cent. N.C.N. in four hours. The broth used was markedly acid, the reaction of the mixture 10 c.c. of emulsion and 1 c.c. of the *Bacillus Welchi* broth was n/120 at the commencement of the experiment.

(*a*) Phosphorus was primarily used during the course of these experiments in the endeavour to obtain bacteriologically sterile livers, the livers from normal animals, as explained above, practically always containing bacteria. The idea was that the excretion of the drug into the bile passages would exercise a bactericidal effect there. We noted that the livers from phosphorus-poisoned rabbits were more frequently sterile than normal. In addition it was used for the purpose of these and other experiments *re* autolysis, &c. In these experiments the rabbits were given 1 c.c. of the oleum phosphoratum B.P. The drug apparently caused the animal no pain, it ate well, &c., till shortly before the fatal issue. The animals were killed at different periods, bled, and the organs removed sterilely.

Experiment 9.—A 20 per cent. emulsion of the liver of a rabbit poisoned with phosphorus was made in sterile normal saline.

Control	24	mgr.	non-coagulable	nitrogen	per cent.	of emulsion.
One hour's incubation at 37° C.	24	,,	,,	,,	,,	,,
Three hours ,, ,,	24	,,	,,	,,	,,	,,
Four ,, ,, ,,	24	,,	,,	,,	,,	,,
Six ,, ,, ,,	24	,,	,,	,,	,,	,,
Eight ,, ,, ,,	24	,,	,,	,,	,,	,,
Twelve ,, ,, ,,	24	,,	,,	,,	,,	,,

Experiment 10.—Similar to Experiment 9, only continued longer. Animal very ill at time of removal of liver.

Control... ...	34	mgr.	non-coagulable	nitrogen	per cent.	of emulsion.
Twenty-four hours	39	,,	,,	,,	,,	,,
Forty-eight hours	49	,,	,,	,,	,,	,,

Increase in twenty-four hours about 15 per cent.
,, ,, forty-eight ,, ,, 44 ,,

Experiment 11.—Solid pieces of liver from a phosphorus poisoned rabbit were removed sterilely, and remained sterile throughout the experiment. At the end of period of incubation the pieces, &c., were weighed and ground up to 20 per cent. emulsion in normal saline. The organ was very fatty. (Animal killed forty-eight hours after inoculation of oleum phosphoratum.)

Control	42	mgr.	non-coagulable	nitrogen	of emulsion.
Seven hours' incubation at 37° C. ...	42	,,	,,	,,	,,
Twenty-four hours ,, ,, ...	46	,,	,,	,,	,,
				– 10 per cent. increase.	
Seventy-two ,, ,, ,, ...	52	,,	,,	,,	,,
				– 24 per cent. increase.	

Experiment 12.—A liver from a phosphorus-poisoned rabbit was removed sterilely shortly before fatal termination. Pieces were placed in sterile tubes and incubated, then treated as usual.

Control ... ... 108 mgr. non-coagulable nitrogen per cent.
After ninety-six hours ... 108 ,, ,, ,, ,,
Ratio of coagulable to non-coagulable nitrogen before incubation, 0·032.
,, ,, ,, ,, after ,, 0·032.

Here there was no autolysis. Bacteriological sterility during the whole of the experiment. Another specimen of liver gave a similar result.

(C) *Liver of Rabbits with Bacterial Toxæmia.*

Experiment 14.—A rabbit was inoculated intravenously with two slopes of dead *Bacillus coli;* after sixteen hours it became very ill, was killed, and the liver removed sterilely. A control rabbit had no food for the same time, to make the experiment strictly comparable. The liver was ground up in the usual way and a 10 per cent. emulsion made with normal saline. During the process of grinding up the tube was placed in an ice jacket. The control liver was treated in the same way.

	Bacillus coli liver Percentage emulsion	Control liver (no food sixteen hours) Percentage emulsion
Control	5·76 mgr.	6·78 mgr. non-coagulable nitrogen
One hour	5·76 ,,	6·78 ,, ,, ,,
Two hours	5·76 ,,	6·78 ,, ,, ,,
Three ,,	5·76 ,,	6·78 ,, ,, ,,
Four ,,	5·76 ,,	6·78 ,, ,, ,,
Five ,,	5·76 ,,	6·78 ,, ,, ,,
Six ,,	5·76 ,,	6·78 ,, ,, ,,
Eight ,,	5·76 ,,	6·78 ,, ,, ,,
Ten ,,	5·76 ,,	6·78 ,, ,, ,,

Another experiment gave a like result.

Experiment 15.—Similar to Experiment 14.

	Infected rabbit	Control rabbit
Control	6·8	6·2 mgr. non-coagulable nitrogen per cent.
One hour at 37° C.	6·8	6·2 ,, ,, ,, ,,
Two hours ,,	6·8	6·2 ,, ,, ,, ,,
Three ,, ,,	6·8	6·2 ,, ,, ,, ,,
Four ,, ,,	6·8	6·2 ,, ,, ,, ,,
Six ,, ,,	6·8	6·2 ,, ,, ,, ,,
Seven and a half hours at 37° C.	7·2	6·2 ,, ,, ,, ,,
Ten hours at 37° C.	7·9	6·2 ,, ,, ,, ,,
Twelve ,, ,,	8·9	6·2 ,, ,, ,, ,,

But here a growth of *Bacillus coli* was obtained.

Hence it would appear that in phosphorus poisoning and bacterial toxæmia the liver of the infected animals does not autolyse more rapidly *in vitro* than do normal ones, and this in spite of the fact that the blood of infected animals has a great increase in N.C.N. chiefly or almost wholly in the form of urea, which might indicate increased rate of degradation *in vivo*. In fact some of the phosphorus livers do not autolyse at all—it would appear as if the phosphorus had also destroyed the tissue enzymes.

(*D*) *Leucocytes and Marrow.*

Opie [30] in his paper showed that leucocytes easily autolyse in the presence of 0·2 per cent. sodium bicarbonate, less easily in 0·2 per cent. acetic acid. He also showed that the serum antitrypsin exerted an inhibiting influence on the autolysis. He obtained similar results for marrow.

Experiment 16.—We were able to obtain some pus from a case of tubercular psoas abscess. It was quite sterile apart from tubercle bacilli, which were very few. The pus cells were alive, were still capable of active phagocytosis, and were not stained by trypan blue. The cells were broken up into an emulsion (20 per cent.) by shaking as above. Previously they had been centrifuged, the serum removed, and cells washed with sterile normal saline several times.

	Non-coagulable nitrogen
Control ...	18 mgr. per cent.
Incubated alone seven days at 37° C. ...	32 ,, ,,
Made n/20 acid ...	20·4 ,, ,,
Made n/20 alkali ...	20·4 ,, ,,

We got a similar result in several other experiments made with the same pus.

Experiment 17.—Leucocytes from the peritoneal cavities of several guinea-pigs by aleuron and casein injection. Perfectly sterile. Treated as above.

	Non-coagulable nitrogen
Control ...	0 mgr. per cent. of emulsion.
Control, incubated at 37° C. for three days ...	6 ,, ,, ,,
,, made 0·2 per cent. lactic acid ...	9 ,, ,, ,,
,, made alkaline 0·2 per cent. sodium carbonate	9 ,, ,, ,,

From which it will be seen that the sterile autolysis of leucocytes is slow, even if acidified or made alkaline.

Experiment 18.—Rabbit marrow was carefully and sterilely removed from the long bones. Emulsified in saline as above. Sterile bacteriologically after emulsification.

	Non-coagulable nitrogen
5 c.c. emulsion—5 c.c. saline, control	*Nil*
,, ,, ,, incubated at 37° C. for four days ...	9 6
,, ,, ,, made 0·2 per cent. Na_2CO_3 for four days...	9·6
,, ,, ,, made 0·2 per cent. lactic acid	9·6

Similar experiments gave very like results. The marrow of course contained a fair number of red cells.

These results are so at variance with those of other observers that we wondered if there was not some error in our methods. In confirmation, however, of our own results the following experiment can be quoted.

Experiment 19.—A rabbit was killed, bled as much as possible. It was then placed in a strong lysol bath to kill bacteria in the skin; the skin was then drawn off in a room, the air of which was saturated with moisture, and a thigh was amputated sterilely from just below the hip to below the knee; the thigh was then placed in a wide-mouthed glass-stoppered sterile bottle, and the bottle was sealed with paraffin; the thigh was kept in this bottle for eight months: four months at 37° C. in the incubator and four months at summer room temperature (1916). Owing to the above results we examined the thigh at the end of these eight months. The muscle appeared to be perfectly preserved, except that it was a little discoloured and had a steamed appearance. There was very little exuded fluid in the jar. On section and microscopic examination the muscle bundles are all perfect, the transverse striation is generally quite perfect, only the nuclei appear diminished in number and shrunken. The interstitial tissue is a little soft, but nerves, blood-vessels, &c., are perfectly visible and appear undegenerated. There was no smell of decomposition; no fatty acid smell. The bone was then opened, and the marrow was seen to be quite firm, only pale. Sections were then made: the drawings herewith are from them. The marrow is almost like the section of fresh marrow—the main change is the absence of red cells; they have all autolysed and are represented by masses of pigment. The marrow cells, &c., have stained perfectly. This shows that absolutely sterile autolysis is extremely slow. This can further be confirmed by post-mortem observation—sterile infarcts of the spleen or kidney do not undergo autolysis at any great rate, there is nothing like a 50 per cent. degradation of the dead tissue in twenty-

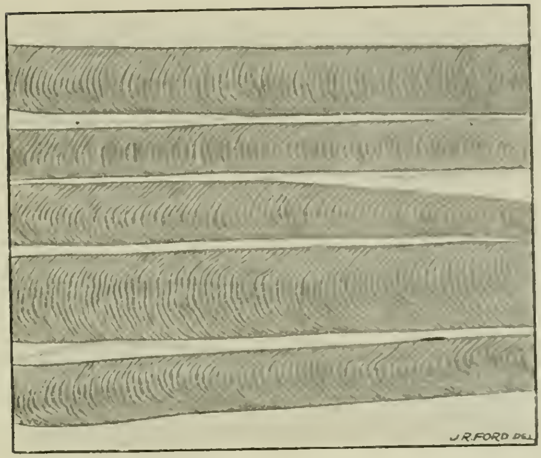


FIG. 1.

Experiment 19. Section of rabbit's muscle incubated for four months at 37° C. and four months at room temperature.

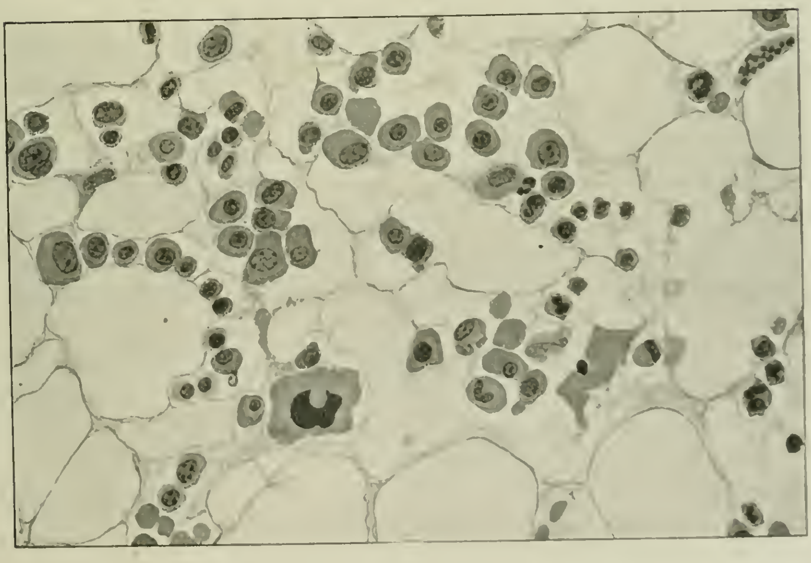


FIG. 2.

Experiment 19. Section of rabbit's marrow incubated at 37° C. for four months and room temperature (summer. 1916) for four months.

four hours (Schryver). Sterile infarcts in fact are only very slowly absorbed. Hence we must conclude from all these experiments that sterile autolysis is very slow, but that autolysis can be greatly accelerated by the presence of bacteria, which act chiefly by the formation of a suitable degree of acidity for the activation of the ferment, and partly in themselves may be capable of digesting the protein.

(IV) EFFECT OF SERUM ANTITRYPSIN ON TISSUE AUTOLYSIS.

In the previous section of this paper it has been shown that serum antitrypsin is only definitely destroyed with an acid concentration equal to n/20. If now the autolytic ferment of the tissues is inhibited by an antitrypsin similar in nature to serum antitrypsin it would have been expected that a similar degree of acidity would be needed to destroy the inhibiting antitrypsin. Experiment 7, Section III, however, shows that the rate of autolysis of liver tissue varies with the acid concentration present, and that there is no apparent degree of acid concentration below which autolysis does not occur and above which it occurs, as might be expected if tissue autolysis were inhibited normally by the presence of an antitrypsin of the nature of serum antitrypsin. It has also been shown that antitrypsin once inactivated by acid cannot be reactivated by neutralization; now Experiment 7, Section III, shows that even when a possible antitrypsin has been destroyed by a concentration of n/20 acid, autolysis of the tissue cannot take place when neutralization has been carried out: the antitrypsin if present would have been destroyed, if it were of the nature of serum antitrypsin. The experimental results lead to the conclusion that there is no tissue antitrypsin of the nature of serum antitrypsin, but that the autolytic ferment is normally inhibited by the normal reaction of the tissues.

Experiment 1.—An emulsion of rabbit's liver was made in the usual way and was found to be bacteriologically sterile. Sterile rabbit's serum was used as antitrypsin. A mixture of the two was made in the proportion of 20 c.c. of liver emulsion and 5 c.c. of serum.

	Non-coagulable nitrogen in milligrammes per 100 c.c.
(*a*) Mixture, not incubated	36
(*b*) ,, incubated	36
(*c*) ,, made n/60 lactic acid	36
(*d*) ,, made n/40 ,, ,, ...	36
(*c*) ,, made n 20 ,, ,, ...	48

Incubated four hours at 37° C.

Experiment 2.—An emulsion similar to Experiment 1 was used; same proportions of emulsion and serum or saline used.

	Non-coagulable percentage of emulsion
(*a*) Liver emulsion alone + 5 c.c. saline not incubated, made n/40 lactic acid	36
(*b*) Liver emulsion, incubated	54
(*c*) Liver emulsion + 5 c.c. serum, no acid, not incubated	36
(*d*) ,, ,, ,, ,, ,, incubated	36
(*e*) ,, ,, made n/40 lactic acid, incubated	36
(*f*) ,, ,, but the serum used was first deantitrypsinized by making n/20 with lactic acid, incubating for one hour at 37° C., then neutralized; the whole afterwards made n/40 lactic acid, incubated	36

Incubation four hours at 37° C.

Experiment 3.—Another experiment on similar lines to Experiment 2. In each test was 20 c.c. of a thick liver emulsion, then saline, serum, &c., added and all made n/20 lactic acid by calculation.

	+5 c.c. saline	+ 5 c.c. serum	+ 5 c.c. deantitrypsinized serum	+ 5 c.c. (10 per cent. egg white, fresh)
Control	36	42	42	36
Incubation twenty hours	102	54	54	54
,, seventy-two ,,	138	78	78	72

Non-coagulable nitrogen milligrammes per cent. of the emulsion.

(1) Experiment 1 appears to show an inhibiting influence of the antitrypsin on the autolytic ferment, since with n/20 there is autolysis and below that n/40, &c., which does not destroy antitrypsin, there is none.

(2) Experiment 2 shows that in spite of previously destroying the antitrypsin (*f*) there is no greater degree of autolysis than when the antitrypsin is unaffected (*e*), whereas with n/40 lactic acid alone the liver undergoes marked autolysis.

(3) Experiment 3 shows that when serum is added there is much less autolysis when the reaction is adjusted to n/20 by calculation, and hence antitrypsin inhibited, than when no serum is added. The same is noted even when the antitrypsin of the serum has been previously destroyed. It therefore appears that the restraining action of serum on tissue autolysis is not due to the inhibiting action of serum antitrypsin but to the reaction of the serum affecting the H ion concentration and the consequent differences in this explaining the various results. Further, in the work of previous observers carried out under conditions which were not absolutely bacteriologically sterile the germicidal action of serum greatly helped the idea of an inhibitory activity. We accordingly carried out experiments where the amount of hydrogen ion

was the same in all the experiments. A series of preliminary tests were made. An emulsion of liver was made in normal saline and

(*a*) 4 c.c. and 1 c.c. of saline were made calculated n/20 lactic acid.
(*b*) 4 ,, ,, 1 ,, ,, ,, ,, n/40 ,, ,,
(*c*) 4 ,, ,, 1 ,, of serum ,, ,, n/40 ,, ,,
(*d*) 4 ,, ,, 1 ,, ,, ,, ,, n/40 ,, ,,

To each was added the same amount of a 0·5 per cent. solution of neutral red and a titration made with n/40 NaOH. The amounts required to just make the tubes alkaline to neutral red[1] to the same degree were determined, the differences obtained indicated the amounts of acid bound by the serum and its salts. This amount was added to the amounts previously calculated in each case and new mixtures were thus adjusted till each pair, saline and serum, required the same amount of alkali to produce the same tint in the neutral red in each series. Thus—

Experiment 4.—Rabbit liver emulsion in the proportion of 20 c.c. of emulsion with 5 c.c. of saline or rabbit serum. Sterile experiment.

	(*a*) Saline added calculated n/20	(*b*) Serum added reaction = to (*a*)	(*c*) Saline added calculated n/40	(*d*) Serum added reaction = to (*c*)
Not incubated ...	30	36	30	36
Incubated six hours ...	60	69	48	55
,, twenty-four hours	108	120	84	96
	milligrams non-coagulable nitrogen per cent. of emulsion.			
	Per cent.	Per cent.	Per cent.	Per cent.
Increase in six hours ...	100	91	60	53
,, twenty-four hours	260	250	180	166
	as just determined, using neutral red as indicator.			

Experiment 5.—An emulsion of freshly killed chicken liver was made and found to be sterile. It was mixed as follows in proportion of 20 c.c. of the emulsion to 5 c.c. of the other constituents.

	Non-coagulable nitrogen milligrams per cent.		
	Control	Incubation	Percentage rise
(*a*) Liver + saline, made nominal n/20 lactic acid ...	25·2	60·0	= 140·0
(*b*) Liver + 5 c.c. egg antitrypsin titre, made nominal n/20 ...	27·6	50·4	= 80·0
(*c*) Liver + chicken's serum, made nominal n/20 ...	27·6	42·0	= 50·0
(*d*) Liver + chicken's serum, made equal in free acid to the liver and saline tube (*a*) ...	27·6	67·2	= 140·0
(*e*) Liver + antitrypsin, made equal in free acid to the liver and saline (*a*) ...	27·6	64·0	= 130·0

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[1] This was used since it was the only indicator available.

Hence here the egg antitrypsin does not inhibit autolysis of chicken liver. Again the result depends on the degree of ionization.

Thus serum antitrypsin exercises no restraining influence on the autolysis of tissues such as the liver. From Opie's [30] experiments, however, it appears that the serum antitrypsin inhibits the leucocyte ferments since he shows that if an effusion containing leucocytes is incubated no autolysis occurs, but if the effusion is first centrifuged, the serum removed and the leucocytes washed, autolysis occurs. In the following experiments the influence of serum antitrypsin on leucocyte ferment is shown.

Experiment 6.—Sterile pus from a psoas abscess. The pus was centrifuged, the supernatant serum removed and put aside, the leucocytes repeatedly washed in sterile normal saline. A 20 per cent. emulsion was made in normal saline : 15 c.c. were used in each test. Mixtures incubated for twenty-four hours.

			Milligrams non-coagulable nitrogen per cent.
(*a*)	Leucocytes	+ 1 c.c. of normal saline (Control)	= 16
(*b*)	,,	+ 1 c.c. of normal saline, incubated	= 22
(*c*)	,,	+ 1 c.c. of supernatant fluid, incubated	= 18
(*d*)	,,	+ 1 c.c. of supernatant fluid, heated to 60° C. for thirty minutes, incubated	= 20
(*e*)	,,	+ 1 c.c. of supernatant fluid (Control)	= 17
(*f*)	,,	+ 1 c.c. of human serum, incubated	= 17
(*g*)	,,	+ 1 c.c. of human serum, heated to 60° C. for thirty minutes, incubated	= 21
(*h*)	,,	+ 1 c.c. of human serum (Control)	= 17

Control = not incubated.

Conclusions.

This paper deals with the nature of serum antitrypsin showing :—

(1) That it is protein in nature and not lipoidal.

(2) That it is not adsorbed by bacteria or colloids.

(3) That there is a weak proteolytic ferment present in the serum.

(4) That it is destroyed by 56° C. and by weak acids and alkalis to N/400.

(5) That it does not influence tissue autolysis.

Also the paper deals with tissue autolysis, and shows that this is quite slow under conditions of absolute bacteriological sterility. Further the paper deals with the question of autolytic degradation occurring in infection and anaphylaxis and is opposed to the work of Jobling bearing on these points.

REFERENCES.

[1] Teale and Embleton. *Lancet*, 1913, i, p. 234.
[2] Vaughan. *Journ. Amer. Med. Assoc.*, 1914, lxii, p. 583.
[3] Jobling and Petersen. *Journ. Exper. Med.*, 1914, xix, pp. 459 *et seq.*
[4] Wright. *Lancet*, 1917, i, p. 6.
[5] Hildebrandt. *Virchow's Archiv*, 1893, cxxxi, p. 5.
[6] Hahn. *Berl. klin. Wochenschr.*, 1917, p. 499.
[7] Levine and Sankey. *Journ. Med. Research*, 1903, x, p. 127.
[8] Weinland. *Zeit. f. Biol.*, 1903, xliv, p. 45.
[9] Hamill. *Journ. Physiol.*, 1906, xxxiii, p. 429.
[10] Cathcart. *Journ. Physiol.*, 1905, xxxi, p. 491.
[11] Achalme. *Ann. de l'Inst. Pasteur*, 1901, xv, p. 737.
[12] Jochmann and Kantorowicz. *Münch. med. Wochenschr.*, 1908, lv, p. 728.
[13] Mayer, K. "Folia Serologica," 1911, ii, p. 472.
[14] Eisner, G. *Zeit. f. Immunitätsf. O.*, 1908-9, i, p. 650.
[15] Wiens. *Münch. med. Wochenschr.*, 1907, liv, p. 2637.
[16] Delezenne and Pozerski. *Comptes rend. Soc. Biol.*, 1903, lv, p. 327.
[17] Bayliss and Starling. *Journ. Physiol.*, 1905, xxxii, p. 129.
[18] Rosenthal. "Folia Serologica," 1910, vi, p. 285.
[19] Bayliss. *Archives de Soc. Biol.*, 1904, xi (suppl.), p. 261.
[20] Abderhalden and Gigon. *Zeit. f. physiol. Chemie*, 1907, liii, p. 251.
[21] Walters. *Journ. Biol. Chem.*, 1912, xii, p. 43.
[22] Schwarz. *Wiener. klin. Wochenschr.*, 1909, xxii, p. 1151.
[23] Sugimoto. *Arch. f. Exp. Path. u. Pharm.*, 1913, lxxiv, p. 14.
[24] Jobling. *Journ. Exper. Med.*, 1914, xix, pp. 239 and 459.
[25] Cobliner, S. *Biochem. Zeitschr.*, 1910, xxv, p. 494.
[26] Cathcart. *Journ. Physiol.*, 1904, xxxi, p. 497.
[27] Abderhalden. Abwehrfermente, 1913.
[28] Jacoby. *Zeit. f. physiol. Chemie*, 1900, xxx, p. 149.
[29] Schryver. *Journ. Physiol.*, 1905, xxxii, p. 159.
[30] Opie. *Journ. Exper. Med.*, 1905, vii, p. 316 and 795.

The Relation of the Antitryptic Titre of the Blood to Bacterial Infection and Anaphylaxis.[1]

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IN this paper we propose recording our observations on the variations in the antitryptic titre of the blood of animals during various stages and degrees of bacterial infection and anaphylactic shock. In addition we include observations on the alteration in the antitryptic titre of the serum when anaphylotoxin is produced *in vitro*. So far we have been unable to find any records of systematic observations regarding variations in the antitryptic titre of the blood in infection, &c., the observations are chiefly scattered clinical observations, there are no records of continued observations.

In these experiments we have investigated the changes in the antitryptic power of the blood in—

(I) Normal animals infected with or inoculated with—
  (A) Non-pathogenic bacteria.
  (B) Heterogeneous non-toxic protein.
  (C) Pathogenic and virulent bacteria.
  (D) Exotoxins.
  (E) Sensitized bacteria.

(II) Immunized animals reinjected with the homologous bacteria.

(III) Sensitized or immunized animals reinjected with the homologous antigen.

(IV) Variations due to (*a*) food; (*b*) phosphorus poisoning, &c.; (*c*) agents destroying antitrypsin *in vitro*; (*d*) drugs affecting the temperature; (*e*) drugs damaging blood-forming tissue.

(V) Variation in the *in vitro* preparation of anaphylotoxin.

(VI) Influence of antitrypsin on the growth of bacteria.

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[1] At a meeting of the Section, held December 2, 1919.

[2] The expenses of this research were defrayed by a grant from the Medical Research Committee.

METHOD.

It has been shown in a previous paper (1) that antitrypsin is not affected by keeping, especially at 0° to 3° C. In order to reduce errors due to strength of trypsin, &c., the samples of serum obtained from a given animal were all stored in this way till the series was complete, and all were tested together, using for the experiment the same gelatine, trypsin, &c.

The method adopted was as stated previously (2). The unit of trypsin was determined as that amount of a 1 per cent. solution which caused complete liquefaction of 1 c.c. of 15 per cent. gelatine in thirty minutes at 37° C. in the water bath. The tubes after incubation were kept at 10° C. for thirty minutes before the reading was taken. The unit of trypsin thus determined was placed in a series of small tubes and graded quantities of the serum to be tested added (the serum was diluted 1 : 2 or 1 : 5 according to its titre; which in some cases was determined roughly first). These mixtures were incubated for thirty minutes at 37° C. in the water bath, then 1 c.c. of 15 per cent. gelatine at 37° C. added, and a further period of thirty minutes' incubation in the water bath at 37° C. allowed—then cooled at 10° C. for thirty minutes. The titre was the smallest amount of serum completely inhibiting liquefaction by the trypsin.

(1) NORMAL ANIMALS INFECTED WITH OR INOCULATED WITH—

(A) NON-PATHOGENIC BACTERIA.

Experiment 1: Sarcina lutea.—A rabbit of 1,920 grm. inoculated intravenously with one slope of *Sarcina lutea.*

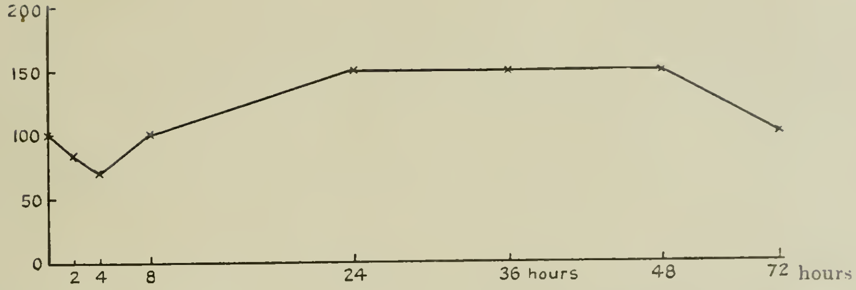
Antitryptic titres		Before inoculation	3	6	12	24	38	48	60 hours after inoculation
Unit in	...	80	100	100	80	60	55	55	80 c.mm. of serum

Experiment 2: Sarcina lutea.—A quarter slope of a twenty-four hours' laboratory strain was inoculated intravenously into a rabbit.

Titre		Before inoculation	3	6	9	24	48	72 hours after inoculation
Unit in	...	60	60	80	80	40	30	40 c.mm. of serum

Experiment 3: Bacillus mycoides.—Growing at 22° C. Two slopes were inoculated into a rabbit of 1,080 grm.

Antitryptic titres		Before inoculation	2	4	8	24	36	48	72 hours after inoculation
Unit in	...	60	70	80	60	40	40	40	60 c.mm. of serum



Experiment 3. Rabbit inoculated with two slopes of *Bacillus mycoides*, 22° C. intravenously.

Experiment 4: Bacillus smegma.—Two slopes were inoculated intravenously into a rabbit of 1,860 grm.

Titre		Before inoculation	3	6	9	18 hours (death) after inoculation
Unit in	...	60	55	50	45	35 c.mm. of serum

Experiment 5: Bacillus prodigiosus.—Two slopes of *Bacillus prodigiosus* were inoculated intravenously into a rabbit of 1,960 grm.

Antitryptic titres		Before inoculation	3	6	10	24	48	72 hours after inoculation
Unit in	...	60	80	80	60	40	40	50 c.mm. of serum

(B) PRIMARILY NON-TOXIC PROTEINS.

(1) *Sheep's Red Corpuscles, washed Sterile with Sterile Normal Saline.*

Experiment 6.—10 c.c. of sheep's sterile red cells were inoculated intraperitoneally into a rabbit.

Antitryptic titre		Before inoculation	3	6	9	24	36	48	72 hours after inoculation
Unit in	...	60	80	80	60	35	35	30	30 c.mm. of serum.

Experiment 7.—Sterile washed sheep's red cells: 10 c.c. were inoculated intraperitoneally into a rabbit of 2,480 grm.

Antitryptic titre		Before inoculation	3	9	24	48	90 hours after inoculation
Unit in	...	60	60	40	30	30	60 c.mm. of serum

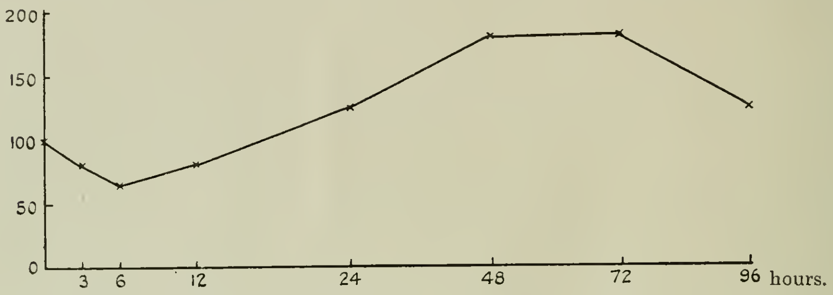
Experiment 8.—5 c.c. of sterile sheep's red cells were inoculated intravenously into a rabbit of 2,020 grm.

Antitryptic titre		Before inoculation	2	4	6	20	36	54	72	96	120 hours after inoculation
Unit in	...	60	60	80	80	40	30	30	30	60	60 c.mm. of serum

(2) *Sterile Egg Albumen.*

Experiment 9.—5 c.c. of a 10 per cent. suspension of sterile egg albumen was inoculated intravenously into a rabbit of 2,860 grm.

Antitryptic titre		Before inoculation	3	6	12	24	48	72	96 hours after inoculation
Unit in	...	50	60	80	60	40	30	30	40 c.mm. of serum



Experiment 9. Rabbit inoculated with 5 c.c. 10 per cent. egg albumen intravenously.

Experiment 10.—Similar experiment to No. 9, only 5 c.c. of a 1 per cent. suspension was injected. Rabbit of 1,800 grm.

Antitryptic titre		Before inoculation	3	6	10	24	36	48	72 hours after inoculation
Unit in	...	60	70	70	60	50	40	40	50 c.mm. of serum

Experiment 11.—Similar experiment to No. 9, only 1 c.c. of 0·1 per cent. solution was used. Rabbit of 1,960 grm.

Antitryptic titre		Before inoculation	3	6	9	20	36	48 hours after inoculation
Unit in	...	60	60	80	80	40	40	60 c.mm. of serum.

(3) *Sterile Gelatine.*

Experiment 12.—Rabbit of 2,000 grm. was inoculated with 20 c.c. of 30 per cent. gelatine intraperitoneally.

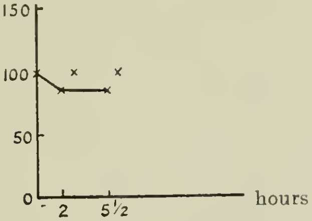
Antitryptic titre		Before inoculation	3	6	12	24	48	72 hours after inoculation
Unit in	...	80	100	100	80	60	40	60 c.mm. of serum

(C) PATHOGENIC BACTERIA.

(1) *Experiments with Bacillus coli.*

Experiment 13.—Four slopes of virulent *Bacillus coli* were inoculated intravenously into a rabbit of 2,500 grm. Death occurred in five and a half hours.

Antitryptic titre of serum	Before inoculation	2 hours after	After death 5½ hours
Unit in	70	80	80 c.mm. of serum



Experiment 13. Rabbit inoculated intravenously with four slopes of *Bacillus coli*; death in five and a half hours.

Experiment 14.—Four slopes of virulent *Bacillus coli* were inoculated intravenously into a rabbit of 1,800 grm. Death occurred in two hours. The animal soon showed signs of toxæmia.

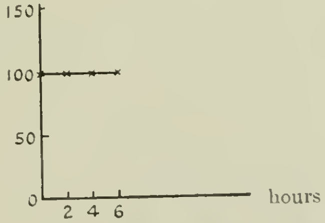
Antitryptic titre	Before inoculation	30 minutes	1 hour	2 hours after inoculation
Unit in	60	60	75	75 c.mm. of serum

Experiment 15.—A rabbit of 2,200 grm. had six slopes of non-virulent *Bacillus coli* inoculated intravenously. Death occurred in six hours.

Antitryptic titre	Before inoculation	3 hours after	Immediately after death, 6 hours
Unit in	60	60	60 c.mm. of serum

Experiment 16.—A rabbit of 1,800 grm. was inoculated with four slopes of non-virulent *Bacillus coli* intravenously. Death occurred in six hours.

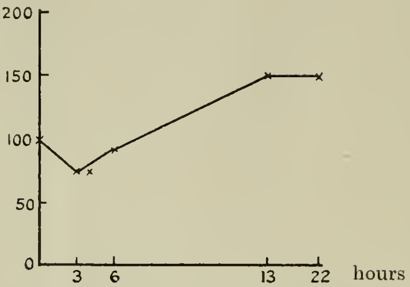
Antitryptic titre	Before inoculation	2	4	6 hours after inoculation
Unit in	60	60	60	60 c.mm. of serum



Experiment 16. Rabbit inoculated intravenously with four slopes of *Bacillus coli*; death in six hours.

Experiment 17.—A rabbit of 2,080 grm. was inoculated with four slopes of an ordinary laboratory strain of *Bacillus coli* intravenously. Death occurred in twenty-two hours.

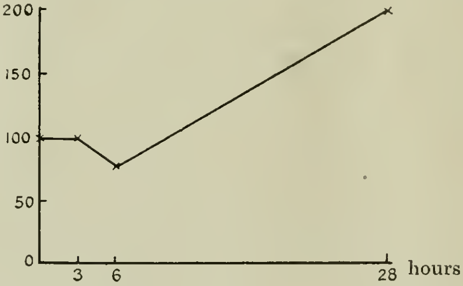
Antitryptic titre	Before inoculation	3	6	18	22 hours after inoculation
Unit in ...	60	80	70	40	40 c.mm. of serum



Experiment 17. Rabbit inoculated with four slopes of *Bacillus coli* intravenously; death in twenty-two hours.

Experiment 18.—A rabbit of 1,720 grm. was inoculated intravenously with four slopes of dead *Bacillus coli*, the same strain as Experiment 15. Recovery.

Antitryptic titre	Before inoculation	3	6	28 hours after inoculation
Unit in ...	80	80	100	40 c.mm. of serum



Experiment 18. Rabbit inoculated with four slopes of dead *Bacillus coli* intravenously; recovery.

Experiment 19.—A rabbit of 2,210 grm. was inoculated with quarter slope of very virulent *Bacillus coli.* Death in thirty-six hours.

Antitryptic titre	Before inoculation	3	6	12	24 hours after inoculation (death)
Unit in	60	60	80	60	40 c.mm. of serum
Bacteria in circulation	—	+	—	—	+

(2) *Experiments with Bacillus typhosus.*

Experiment 20.—A rabbit of 1,980 grm. was inoculated intravenously with two slopes of a twenty-four hours' growth of *Bacillus typhosus* on agar. Recovery.

Antitryptic titre	Before inoculation	2	4	8	10	24 hours after inoculation
Unit in	40	60	60	60	55	55 c.mm. of serum
Bacteria in peripheral blood		+	+	−	+	−

Experiment 21.—A rabbit of 2,180 grm. was inoculated with two slopes of *Bacillus typhosus* as in Experiment 20, intravenously. Recovery.

Antitryptic titre	Before inoculation	3	6	10	24	48 hours after inoculation
Unit in	60	80	80	60	40	40 c.mm. of serum
Bacteria in peripheral blood		+	+	—	—	

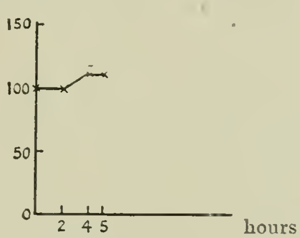
(3) *Experiment swith Varieties of Bacillus mycoides of Varying Virulence* [3].

Experiment 22.—A normal rabbit of 2,018 grm. was inoculated intravenously with two slopes of slightly virulent *Bacillus mycoides* (37° C). Death occurred in sixty-six hours.

Antitryptic titre	Before inoculation	2	4	8	9	24	48	60	66 hours after inoculation
Unit in	60	60	80	55	55	50	40	40	50 c.mm. of serum
Bacteria in peripheral blood		—	+	—	+	—	—	+	+

Experiment 23.—A rabbit of 1,096 grm. was inoculated intravenously with one slope of naturally warm growing *Bacillus mycoides* (37° C.) isolated from a wound. The rabbit died in five hours.

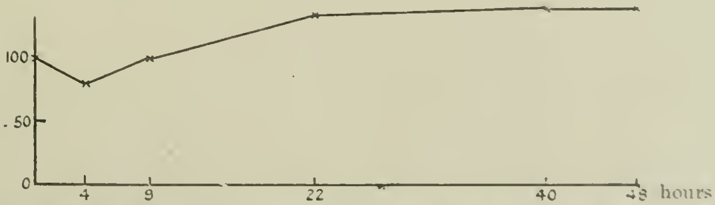
Antitryptic titre	Before inoculation	2	4	5 hours after inoculation
Unit in ...	60	60	55	55 c.mm. of serum



Experiment 23. Rabbit inoculated with one slope of *Bacillus mycoides* (37° C.); death in five hours.

Experiment 24.—A rabbit was inoculated with four slopes of *Bacillus mycoides* educated to grow at 37° C. The animal died in seventy-two hours. Organisms were present in the tissues post-mortem in small numbers.

Antitryptic titre	Before inoculation	4	9	22	48	72 hours after inoculation
Unit in	40	50	40	30	28	28 c.mm. of serum
Bacteria in peripheral blood		—	+	—	—	+ (very few)



Experiment 24. Rabbit inoculated with four slopes of *Bacillus mycoides* (37° C.) intravenously; death in seventy-two hours.

(4) *Experiments with Bacillus anthracis.*

Experiment 25.—A rabbit of 2,080 grm. was inoculated with one slope of *Bacillus anthracis*. Death occurred in 60 hours.

Antitryptic titre	Before inoculation	3	5	10	24	30	48 (death)	hours after inoculation
Unit in	60	80	80	70	50	40	40	40 c.mm. of serum
Bacteria in peripheral blood		+	+	+	−	−	+	
			(very few)				(very numerous)	

(5) *Virulent Streptococci.*

Experiment 26.—A rabbit was inoculated intravenously with two slopes of virulent streptococci. Death occurred in twenty-two hours.

Antitryptic titre	Before inoculation	3	6	10	22 hours after inoculation
Unit in	60	80	60	40	40 c.mm. of serum
Bacteria in peripheral blood	−	+	+	+	+
			(very few)		

Experiment 27.—A rabbit was inoculated intravenously with ¼ c.c. of a twenty-four hours' broth culture of virulent streptococcus. Animal very ill, but recovered.

Antitryptic titre	Before inoculation	3	10	24	30	48	72 hours after inoculation
Unit in ...	60	60	80	50	40	40	50 c.mm. of serum

(6) *Pneumococci.*

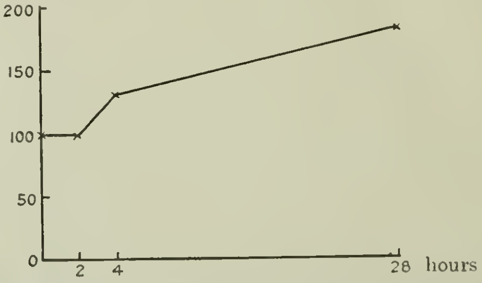
Experiment 28.—A rabbit was inoculated with a virulent broth culture of the pneumococcus. Death occurred in thirty-six hours.

Antitryptic titre	Before inoculation	3	6	9	24	30 hours after inoculation (at death)
Unit in ...	60	80	80	60	40	40 c.mm. of serum

(7) *Bacillus enteritidis Gaertner.*

Experiment 29.—A rabbit was inoculated intravenously with one slope of *Bacillus enteritidis* of Gaertner. Death occurred in twenty-eight hours.

Antitryptic titre	Before inoculation	2	6	28 hours after inoculation
Unit in ...	90	90	70	60 c.mm. of serum



Experiment 29. Rabbit inoculated with one slope of *Bacillus enteritidis Gaertner*; death in twenty-eight hours.

(D) EXOTOXINS.

Experiment 30.—A rabbit of 2,200 grm. was inoculated intravenously with 10 c.c. of tetanus toxin. Death in eighteen hours.

Antitryptic titre		Before inoculation	2	4	7	18 hours after inoculation
Unit in	...	60	60	70	60	40 c.mm. of serum

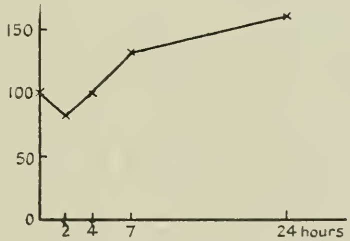
Experiment 31.—A rabbit was inoculated with 7 c.c. of diphtheria toxin, intravenously. Death in twenty-four hours.

Antitryptic titre		Before inoculation	3	6	17	24 hours after inoculation
Unit in	...	80	80	120	80	80 c.mm. of serum

(E) SENSITIZED BACTERIA.

Experiment 32.—A rabbit was inoculated intravenously with two slopes of sensitized typhoid bacilli. Recovery. The typhoid bacilli were exposed to a large quantity of high grade agglutinating serum for twenty-four hours at room temperature.

Antitryptic titre		Before inoculation	2	4	7	24 hours after inoculation
Unit in	...	40	50	40	35	25 c.mm. of serum



Experiment 32. Rabbit inoculated intravenously with two slopes of fully sensitized *Bacillus typhosus.*

Experiment 33.—Experiment as above. A rabbit of 1,920 grm. was inoculated intravenously with two slopes of fully sensitized typhoid bacilli.

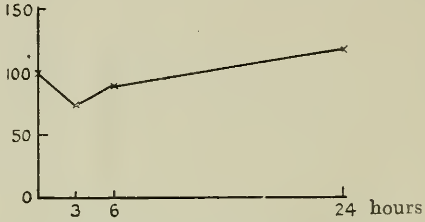
Antitryptic titre		Before inoculation	3	6	10	24	48	72	96	120 hours after inoculation
Unit in	...	60	60	60	50	40	40	40	50	60 c.mm. of serum

Experiment 34 : Sensitized Pneumobacilli.—A rabbit was inoculated intravenously with one slope of sensitized pneumobacilli. Recovery. Sensitization as before.

Antitryptic titre		Before inoculation	3	5	10	24	48	72	96 hours after inoculation
Unit in	...	40	50	45	40	30	25	30	40 c.mm. of serum

Experiment 35: Sensitized Bacillus enteritidis Gaertner.—A rabbit was inoculated intravenously with one slope of highly sensitized *Bacillus enteritidis Gaertner.* It died in twenty-four hours.

Antitryptic titre	Before inoculation	3	6	24 hours after inoculation
Unit in ...	60	80	70	50 c.mm. of serum



Experiment 35. Rabbit inoculated intravenously with one slope of fully sensitized *Bacillus enteritidis Gaertner;* death in twenty-four hours.

(II) INOCULATION INTO IMMUNE AND SENSITIZED ANIMALS.

Experiment 36.—A rabbit of 1,840 grm. with an agglutinin titre against *Bacillus typhosus* of 1 : 10,000 was inoculated intravenously with two slopes of *Bacillus typhosus.* Recovery.

Antitryptic titre	Before inoculation	2	4	8	10	25	48	72 hours after inoculation
Unit in	40	45	40	50	45	30	30	40 c.mm. of serum
Bacteria in peripheral circulation	—	+	+	+	—	—	—	—

Experiment 37.—A rabbit of 2,010 grm. was immunized against the pneumobacillus. Later two slopes of pneumobacilli were inoculated intravenously. Recovery.

Antitryptic titre	Before inoculation	3	6	9	24	48	72	96 hours after inoculation
Unit in	40	40	45	40	30	25	25	30 c.mm. of serum
Bacteria in peripheral circulation	—	+	+	—	—	—	—	—

Experiment 38.—A rabbit was immunized against the *Bacillus mycoides:* 200 mgr. dead bacteria were first inoculated intraperitoneally then two slopes of the organism grown at 37° C. ten days after. Finally, three weeks after last injection two slopes of virulent *Bacillus mycoides* were inoculated intravenously. Death in ninety-six hours.

Antitryptic titre	Before inoculation	3	7	24	30	45	60	96 hours after inoculation
Unit in	50	60	60	50	40	30	30	50 c.mm. of serum

Experiment 39.—Another rabbit was immunized with 200 mgr. dead *Bacillus mycoides,* intraperitoneally. Ten days later two slopes of

virulent *Bacillus mycoides* intravenously, and seven days later two slopes of virulent *Bacillus mycoides* intravenously. Death in seventy-two hours after last injection.

Antitryptic titre	Before inoculation	2	5	9	24	48	72 hours after inoculation
Unit in	40	60	50	50	40	30	30 c.mm. of serum
Bacteria in peripheral circulation	–	+	+	+	–	–	+

Experiment 40.—A rabbit had 200 mgr. dead *Bacillus mycoides* inoculated intraperitoneally. Ten days later two slopes of virulent *Bacillus mycoides* intravenously. Death in eight hours.

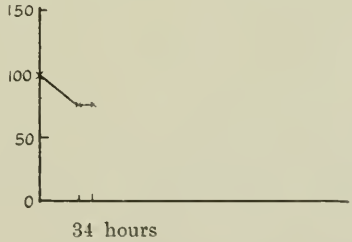
Antitryptic titre	Before inoculation	1	3	5 hours after inoculation
Unit in ...	70	72	72	72 c.mm. of serum

Experiment 41.—A rabbit was inoculated intraperitoneally with 200 mgr. dead *Bacillus mycoides*, followed in ten days by a further 200 mgr. Then ten days later two slopes of non-virulent but 37° C. growing *Bacillus mycoides* were inoculated intravenously. Recovery.

Antitryptic titre	Before inoculation	2	4	6	24	48	72	96 hours after inoculation
Unit in	70	70	70	60	50	45	40	45 c.mm. of serum
Bacteria in circulation	–	+	+	+	–	–	+	–

Experiment 42.—Rabbit immunized against *Bacillus enteritidis Gaertner*. Titre 1 : 50,000. Inoculated intravenously with one slope of *Bacillus enteritidis Gaertner*. •Died in four hours.

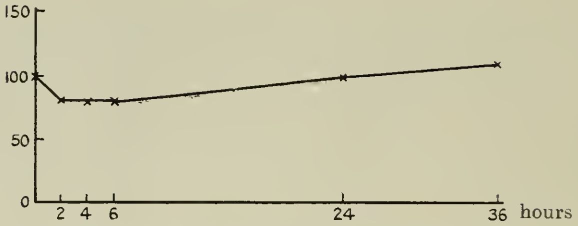
Antitryptic titre	Before inoculation	3	4 hours after inoculation
Unit in	60	80	80 c.mm. of serum



Experiment 42. Immune rabbit inoculated intravenously with one slope of *Bacillus enteritidis Gaertner*; death in four hours.

Experiment 43.—Similar experiment to above. Animal lived thirty-six hours.

Antitryptic titre	Before inoculation	2	4	6	24	30	36 hours after inoculation
Unit in .	40	50	50	50	40	35	35 c.mm. of serum



Experiment 43. Immune rabbit inoculated intravenously with *Bacillus enteritidis Gaertner*; death in thirty-six hours.

Experiment 44.—One slope of sensitized *Bacillus enteritidis Gaertner* was inoculated intravenously into a rabbit immunized against *Bacillus enteritidis Gaertner.* Died in fifty-two hours.

Antitryptic titre	Before inoculation	3	6	9	24	48	52 hours after inoculation
Unit in ...	80	80	90	100	75	50	50 c.mm. of serum

(III) RABBITS SENSITIZED AGAINST EGG ALBUMEN.

Experiment 45.—Rabbits each received 5 c.c. of 10 per cent. suspension of egg albumen intraperitoneally. Twenty-one days later they each received as follows:—

Rabbit	Antitryptic titre Before injection		After death
(1) 0·5 c.c. fresh egg albumen intravenously, death in two minutes	40	...	30 c.mm.
(2) 0·5 c.c. 20 per cent. egg albumen intravenously, death in ten minutes	50	...	40 ,,
(3) 0·5 c.c. 10 per cent. egg albumen ; very ill, recovered.			

Antitryptic titre	Before inoculation	½	1	4	7	24 hours after inoculation
Unit in ...	40	45	50	40	30	30 c.mm. of serum

Experiment 46.—A rabbit was inoculated with 10 c.c. of pure egg albumen intraperitoneally. Fourteen days later 0·25 c.c. of pure egg albumen was inoculated intravenously. The animal became very ill, temperature fell to below 95° F., rapid breathing, &c.

Antitryptic titre	Before inoculation	½	1	2	4	8	24	48 hours after inoculation
Unit in ...	50	50	60	70	70	80	55	55 c.mm. of serum

Experiment 47.—A series of guinea-pigs was inoculated each with 0·5 c.c. of pure egg white. Three weeks later they were injected as follows: sufficient blood was obtained before the operation for the control antitryptic titre.

	Antitryptic titre Before inoculation	 After inoculation
(1) Control; 0·5 c.c. of 10 per cent. egg albumen intravenously; death in two minutes	—	—
(2) Received 0·5 c.c. of 10 per cent. egg albumen intravenously; death in two minutes	50	50
(3) Received 0·5 c.c. of 10 per cent. egg albumen intravenously; death in ten minutes	50	50
(4) Received 0·25 c.c. of 10 per cent. egg albumen intravenously; killed after thirty minutes	55	85
(5) Received 0·25 c.c. of 10 per cent. egg albumen intravenously; killed one hour after	50	70
(6) Received 0·25 c.c. of 10 per cent. egg albumen intraperitoneally: killed one hour after	40	60
(7) Normal pig; 0·5 c.c. egg albumen intravenously; killed one hour after	50	70

Experiment 48.—Rabbit immunized against sheep's serum. Titre of rabbit's serum 1 : 10,000; 2 c.c. of sheep serum inoculated intravenously.

Titres	Before inoculation	Minutes 5	 15	 30	Hours after inoculation 3	 6	 12	 24	 48
Unit in	80	80	90	100	100	100	80	60	50 c.mm. of serum

Experiment 49.—Rabbit immunized against sheep's red cells. Titre 1 : 3,000; 2 c.c. of sheep's washed red cells inoculated intravenously. Animal had fall of temperature, rapid breathing, &c.

Titres		Before inoculation	3	6	12	24	36	48	72 hours after inoculation
Unit in	...	60	60	60	50	40	40	40	50 c.mm. of serum

Experiment 50.—Another rabbit immunized against sheep's red cells. Titre of serum 1 : 5,000; 5 c.c. of sheep's red cells inoculated intraperitoneally. The animal became very ill, but recovered.

Titre		Before inoculation	3	6	9	24	48	72 hours after inoculation
Unit in	...	60	70	80	60	40	30	40 c.mm. of serum

(IV) Influence of Food, &c.

(*a*) During infections animals do not take food, hence it was necessary to study the effect of starvation and food on the antitryptic titre.

Experiment 51.—Rabbit, 2,860 grm. had no food, only water in cage for forty-eight hours after a full meal, then food was given. The animal took water freely.

Titre		Before inoculation	12	24	36	48	Food 48	50	Food 58	72	96 hours after inoculation
Unit in	...	60	55	55	55	60	—	80	100	100	70 c.mm. of serum

Experiment 52.—Rabbit, 2,720 grm.; no solid food for sixty-eight hours, only water, food continuously after.

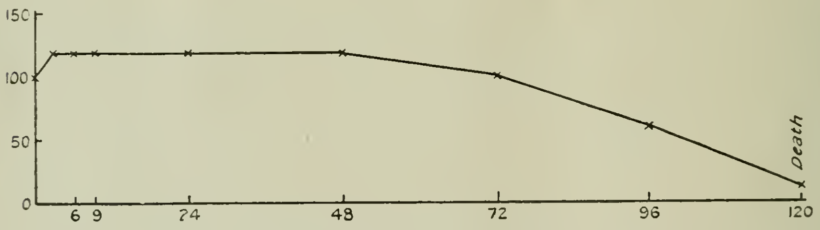
Titre		Before inoculation	24	45	53	Food 68	72	96 hours after inoculation
Unit in	...	60	55	65	65	85	85	75 c.mm. of serum

These experiments appear to show that a period of forty-eight hours' abstinence from food has no marked effect on the antitryptic titre of the blood, and that therefore the abstinence from food during toxæmia is not responsible for the alteration in the antitryptic titre.

(*b*) In phosphorus poisoning there appears to be evidence of autolytic degradation of the tissues, hence it seemed important to ascertain the changes in the antitryptic titre following the injection of oleum phosphoratum B.P.

Experiment 53.—A rabbit was injected subcutaneously with 1 c.c. of oleum phosphoratum B.P. The animal suffered no pain and ate well up to the evening before death.

Antitryptic titre		Before inoculation	3	6	9	24	48	72	96	120 hours after inoculation
Unit in	...	60	50	50	50	50	50	60	100	not in 500 c.mm. of serum



Experiment 53. Rabbit inoculated subcutaneously with 1 c.c. Ol. phosphoratum (B.P.).

Experiment 54.—Similar experiment. Rabbit inoculated subcutaneously with 1 c.c. of oleum phosphoratum B.P. Death in eighty hours.

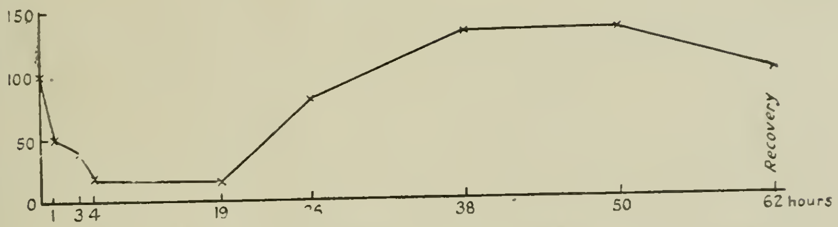
Antitryptic titre		Before inoculation	3	9	24	36	48	72	80 hours after inoculation
Unit in	...	60	60	60	50	50	60	100	300 c.mm. of serum

(*c*) *Effect of Reagents destroying the Antitrypsin in vitro, on the Titre in vivo.*

(1) Lactic acid.

Experiment 55.—A rabbit of 2,000 grm. was inoculated intravenously with 5 c.c. of n/1 lactic acid. This was followed by great rapidity of breathing and air-hunger for ten minutes; the animal then rapidly recovered and there was no subsequent ill effect.

Non-coagulable nitrogen in the blood: Rise in four hours, 57 per cent.; in five hours, 77 per cent.



Experiment 55. Rabbit inoculated intravenously with 5 c.c. N/1 lactic acid.

Titre of blood	Before inoculation	Minutes		Hours after inoculation								
		15	30	1	3	4	19	26	38	50	62	
Unit in ...	80	120	140	200	200	400	400	100	60	60	80	c.mm. of serum

Experiment 56.—A rabbit was kept continuously under the influence of ether for one hour, continually inhaling some.

Antitryptic titre	Before inoculation	1	3	6	24	48 hours after inoculation
Unit in ...	80	80	80	80	60	60 c.mm. of serum

Experiment 57.—A rabbit of 2,180 grm. received a solution containing 0·1 grm. of iodine, and 0·5 of potassium iodide in 10 c.c. of normal sterile saline intraperitoneally.

Antitryptic titre	Before inoculation	4	8	12	24	30	48 hours after inoculation
Unit in ..	60	70	70	70	60	50	40 c.mm. of serum

Another rabbit received the same quantity of iodine and potassium iodide subcutaneously. The change in the antitryptic titre was the same.

Experiments 58 and 59.—Injection of hypertonic saline and normal saline intravenously or intraperitoneally: No effect on the antitryptic titre.

(*d*) *Influence of Temperature-raising Drugs: β-naphthylamine.*

Experiment 60.—A rabbit of 2,420 grm. received 3·5 c.c. of a 3 per cent. solution of β-naphthylamine. The temperature rose from 102·4° to 106·2° F. Pupils became dilated, breathing disturbances, &c.

Antitryptic titre	Before inoculation	30 mins.	2	5	24	48	72 hours after inoculation
Unit in ...	50	50	60	75	55	45	45 c.mm. of serum

Experiment 61.—A rabbit was inoculated with 10 c.c. of a 5 per cent. solution of calcium chloride intraperitoneally. Temperature fell from 102·8 to 96° F.

Antitryptic titre	Before inoculation	30 mins.	1	3	6	24	48	72 hours after inoculation
Unit ...	60	60	60	60	60	55	50	50 c.mm. of serum

(*e*) *Influence of Drugs damaging the White Corpuscle-forming Tissue.*

Experiment 62.—A rabbit received 1 c.c. of benzol subcutaneously every day for four days.

Antitryptic titre	Before inoculation of the first dose	6 hours	24 hours' inoculation second dose	30 hours	48 hours' inoculation third dose	72	96	99	102	105	120 hours after inoculation
Unit in ...	60	60	60	60	60	60	60	60	65	60	60 c.mm. of serum

Experiment 63.—A variation in antitryptic titre similar to that occurring with albumen occurs when a fat such as pure cod liver oil is inoculated intraperitoneally into a rabbit. A rabbit was inoculated with 10 c.c. of sterile pure oleum morrhuæ intraperitoneally.

Antitryptic titre	Before inoculation	3	6	24	48 hours after inoculation
Unit in ...	50	60	60	50	35 c.mm. of serum

(V) ANAPHYLOTOXIN. IN VITRO PRODUCTION.

In another paper [4] it has already been shown that the substances which are commonly used for the production of anaphylotoxin *in vitro* such as agar, starch, bacteria, do not cause adsorption of antitrypsin but cause adsorption of the proteolytic ferment that may be present.

Experiment 64.—The following examples of experiments show that a serum may become intensely toxic without any alteration in antitryptic titre or increase in non-coagulable nitrogen. In the agar experiments 0·5 per cent. of agar sol. was made in normal saline; it was kept at 56° C. till it was mixed with the serum which was at 37° C., the mixture was thoroughly shaken and then gelled on the ice before incubation.

Serum	Treatment	Antitryptic titre		Non-coagulable nitrogen		Fatal dose to guinea-pig
		Before	After	Before	After	
(*a*) 10 c.c. rabbit ...	+ 1·5 c.c. agar	60	60	3·54	3·54	3 c.c. 280 grm. pig
	Four hours 37° C.; sixteen hours room temperature.					
(*b*) 10 c.c. guinea-pig	+ 1 c.c. agar	20	20	4·5	4·5	2·5 c.c. 285 grm.
	Four hours 37° C.; sixteen hours room temperature.					
(*c*) 10 c.c. guinea-pig	+ 100 mgr. bacillus 80	25	25	3·8	3·85	5 c.c.; death in five hours; 190 grm.
	Four hours 37° C.; twelve hours room temperature.					
(*d*) 7 c.c. guinea-pig	+ 3 c.c. starch	30	30	3·6	3·6	5 c.c.; *nil*
7 c.c. guinea-pig	+ 1 c.c. 1 per cent. silicic acid	30	26	3·6	3·6	3 c.c.; convulsions, paresis, recovery
7 c.c. guinea-pig	+ 1 c.c. 0·5 per cent. agar	30	26	3·6	3·6	3 c.c.; acute death
	Incubated four hours at 37° C., then inoculated, &c.					
(*c*) 10 c.c. guinea-pig	+ 2 slopes *Bacillus typhosus*	24	24	3·42	3·4	3 c.c.; acute death
	Incubated four hours at 37° C.					

Other experiments gave similar results.

From these experiments we can conclude—

(1) That in practically every case of bacterial infection there is a variation in the antitryptic index of the blood plasma—a negative phase varying in amount and duration, but usually representing a drop of 20 per cent. lasting on the average about eight hours, and a positive phase lasting from forty-eight to ninety-six hours.

(2) That the variation in the antitryptic index of the blood bears no relationship to the intensity of the infection or the toxicity of the protein inoculated: a first inoculation of egg albumen produces the same variations as a pathogenic bacterium, with equal weights of the various forms of protein. (Experiment 11.)

(3) That previous immunization appears to have practically no influence on the antitryptic curve produced by inoculation of the homologous antigen; similarly sensitization of the antigen produces no difference.

(4) That an infected animal may die with its antitryptic index in any phase: the index may be low if the death is rapid, high if slower; in this stage the blood and tissues may be swarming with bacteria and yet the index is high. On the other hand, an animal may recover with the index lower than its normal. (Experiment 20.) Again animals may die acutely and show no alteration in the titre. (Experiments 15 and 16.) And again an animal may die from an injection which has produced no initial negative phase in the titre (but only a rise). (Experiments 4, 23, 25.)

(5) That in anaphylactic shock the variations in the antitryptic index depend on the acuteness of the shock; in the fulminating shock there is no alteration, in the delayed there is a fall; and this is not on the average appreciably greater than with a first inoculation of a similar amount of antigen. With recovery from anaphylactic shock the index follows the same course as after a first injection.

(6) That anaphylotoxin can be prepared *in vitro* by substances or bacteria which cause no adsorption of antitrypsin, no auto-digestion such as can be demonstrated by an increase in the amount of non-coagulable nitrogen appears to take place. In fact, it has been shown that agar, &c., far from causing adsorption of antitrypsin and consequent auto-digestion, really hinder proteolytic degradation by adsorbing the ferment and not the anti-ferment.

(7) That food or a short period of abstinence from food has little influence on the antitryptic titre.

(8) That temperature-raising drugs like β-naphthylamine produce

a marked variation in the titre, but calcium salts, which also cause temperature variations, do not produce any change.

(9) That lactic acid produces a rapid and marked fall in the blood antitrypsin. Even when the fall equals 60 per cent. loss and lasts twenty-four hours, the animal appears to suffer no apparent ill-effect beyond the transitory marked dyspnœa directly after the inoculation. The animal, however, shows a great increase of non-coagulable nitrogen in the blood amounting to an increase of 50 per cent. in the first four hours. In infections there is no appreciable increase in non-coagulable nitrogen in the first four hours, in fact, in acute toxæmia the animal may die in four to six hours with no appreciable increase in the non-coagulable nitrogen. Then again the comparison between lactic acid and phosphorus poisoning is interesting. With lactic acid the antitryptic fall is rapid and prolonged and yet there are no signs of proteose poisoning, and the tissues show no gross change when the animal is killed some time after the inoculation; with phosphorus poisoning, the fall in antitrypsin only occurs shortly before death, but the blood has a great increase in non-coagulable nitrogen long before there is any fall in antitryptic titre [4].

(10) That although the antitryptic index varies especially after the inoculation of bacteria and protein substances, yet the inoculation of pure fatty substances produces the same type of variation.

The variations in the antitryptic index of the blood are important in view of Jobling's [5] theory of the causation of bacterial toxæmia, and anaphylotoxin production both *in vivo* and *in vitro*. Jobling like Vaughan [6] and Teale and Embleton [7] was impressed by the uniformity in the important symptoms of bacterial toxæmia in animals of the same species fatally infected with widely different types of bacteria. His observations led him to conclude that there was a common cause in all, that the symptoms of poisoning were all through caused by the formation and accumulation of toxic proteoses due, both in infection and anaphylactic shock, to auto-digestion of the plasma in the former, and with anaphylotoxin produced *in vitro* to the auto-digestion of the serum. His theory is that all these different conditions cause the adsorption of the antitrypsin and consequent auto-digestion of the serum or plasma.

(i) Dealing with anaphylotoxin produced *in vitro*. Our experiments show that an intensely toxic serum can be prepared and yet there is no evidence of any alteration in the antitryptic content or non-coagulable nitrogen of the serum used. Further they show, as seen in another paper by us [4], that far from the agar, &c., causing an adsorption of the antitrypsin and allowing unhindered activity of any proteolytic ferment, the agar hinders the activity of the ferment by adsorbing it. Hence, although the methods at our disposal for demonstrating the toxic higher albumoses in small quantity are very inadequate and their possible presence thus missed—the evidence on the whole does not appear to favour the idea that "anaphylotoxin" produced *in vitro* is due to proteolytic auto-digestion of the serum.

(ii) In acute anaphylactic shock there is no diminution of antitrypsin and in delayed shock the fall is not appreciably greater than with the primary inoculation. Hence if anaphylactic shock were solely due to proteose intoxication following adsorption of antitrypsin it ought to occur equally with the primary as with the second inoculation. Then again, if loss of antitrypsin is to account for the production of toxic proteoses, there must be local areas in which the antitrypsin must be completely adsorbed to allow of the action of the proteolytic enzyme —this might conceivably occur in the capillaries of the lungs and liver especially, where the bacteria and by analogy foreign protein may accumulate after injection. This would then mean that the adsorption of antitrypsin in these local areas is insufficient to make an appreciable difference in the antitrypsin of the blood as a whole. Jobling [8] further concludes that the rapid death following the intravascular inoculation of agar suspension or kaolin, &c., is due to the same cause— viz., the agar adsorbing the antitrypsin and permitting auto-proteose intoxication. These acute deaths however, as will be shown later, are due to intravascular clotting, &c., and are not accompanied by alteration in the antitryptic titre, and can be prevented by simultaneous or preceding injection of leech extract.

(iii) The question of the relation between antitrypsin and bacterial toxæmia next requires discussion. It has already been shown that the variations in the antitryptic curve show no relationship to the pathogenicity, toxicity or virulence of the bacteria or foreign protein inoculated. Hence it would appear that the toxin-provoking powers of a bacterium during infection are in no way parellel to the antitryptic fall, and this should be expected according to Jobling's theory if the toxicity is due to auto-proteose intoxication following antitrypsin adsorption by the inoculated bacteria.

Thus again for any auto-digestion of the plasma to occur we must suppose that there must be local areas of practically complete adsorption of the antitrypsin. This can conceivably only occur in the central organ capillaries where the bacteria rapidly become accumulated by becoming enmeshed in incipient clot prior to phagocytosis [9]. Hence if auto-degradation following removal of the antitrypsin inhibition is to occur at all it could only occur there, and the initial fall in the peripheral blood is due to this local removal of antitrypsin. Against this, in addition to the points mentioned above, are: (1) that a rapid fatal result can occur without any fall in the antitryptic titre; (2) that animals may die twenty-four to seventy-two hours after infection with a high antitryptic titre and in a septicæmic condition. The only possible explanation for this would be that the new production of antitrypsin provoked by the initial loss leads to over production, and thus the initial negative phase rapidly gives way to a marked positive phase.

These experiments show that an animal may die from septicæmic infection with the antitryptic titre 50 per cent. or more above normal, so that if auto-digestion is still to be invoked as the cause of the toxæmia it would still be necessary to postulate, forty-eight and even seventy-two hours after the primary infection, local areas of absent antitryptic inhibition unaffected by the general increase, which does not seem very probable. In the septicæmic condition such as in the secondary bacteriæmia of anthrax the phenomenon of the retention of the bacteria in the capillaries of the central organs and the consequent rapid clearing of the peripherally circulating blood does not occur—in the secondary bacteriæmia of this septicæmic state the bacteria are fairly evenly distributed so that, if the bacteria cause adsorption of the antitrypsin at all, there ought to be a rapid fall in titre with its occurrence. This bacteriæmia occurs rapidly and one would hardly expect a still further new rapid production to occur in this state to make up for the loss. There thus seems to be no relation between antitryptic titre and intensity of toxæmia. There thus seems to be no support for the theory of auto-proteose intoxication brought about in this way, especially as bacteria do not adsorb antitrypsin *in vitro*.

(iv) What is the reason of the variation in the antitryptic curve?

(1) Some experiments by us to be published soon, show that an animal may die in the first four hours after inoculation of a culture of bacteria without any evidence of proteoses in the blood or any increase of N.C.N.,[1] later the increase occurs rapidly and in marked degree, and is almost wholly due to urea accumulating in the blood. This increase is all the more marked since badly infected animals take no

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[1] N.C.N. = Non-coagulable nitrogen.

food and in normal animals abstinence from food greatly diminishes the amount of N.C.N. in the blood. The evidence, therefore, points to greatly increased tissue katabolism, so that the inference is that the serum antitrypsin may have an inhibiting effect on tissue autolysis; but our experiments [4] on this subject show that tissue autolysis is in no way influenced by serum antitrypsin except in so far as the serum alters the amount of free H ions on which the autolysis depends.

(2) The possibility of the antitrypsin having to do with the inhibition of leucocytic ferments which might possibly be liberated during phagolysis. *In vitro* experiments show that serum antitrypsin influences leucocyte autolysis. In infection, however, the antitrypsin variation occurs early—in fact too early to be explained by a neutralization of liberated leucocytic ferments due to phagolysis, and further the same occurs when apparently absolutely innocuous proteins are inoculated primarily.

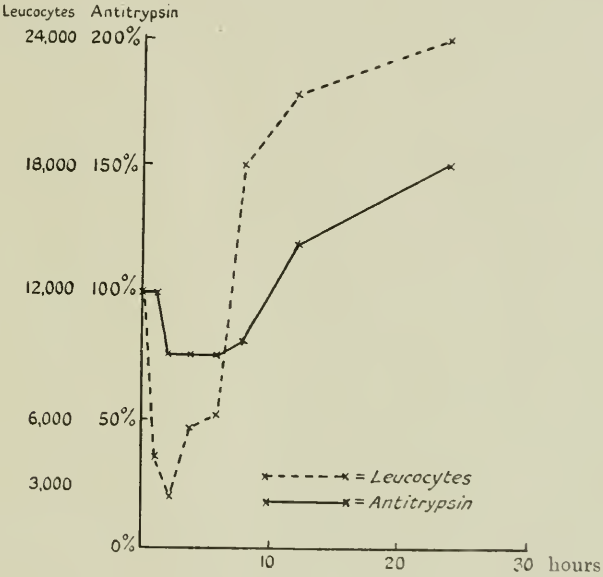

CHART 1.

Chart showing relationship between antitryptic titre and the leucocyte count in a rabbit inoculated with half a twenty-four hour agar slope of *Bacillus typhosus*. This relationship is by no means universal, since as has been shown above, the antitryptic titre may not fall or even go up but the leukopænia apparently always occurs.

The only fact is that the antitryptic curve varies to some extent with the leucocyte count, with the initial leucopenia it falls, with the subsequent leucocytosis it goes up, but there is no marked degree of relationship of the curves.

The antitrypsin is apparently not liberated from the leucocytes during clotting of the blood—it is present in the same amount in the plasma as the serum, and the amount is not diminished by the disintegration of the leucocytes during clotting.

(VI) INFLUENCE OF ANTITRYPSIN ON THE GROWTH OF BACTERIA.

(6) We finally have to consider the point raised by Wright. Wright states that certain bacteria can only grow in the animal body and produce pathogenic effects if the split products of protein digestion are ready at hand for them. He believes from his experiments that this occurs when the serum antitrypsin is destroyed, and the inhibition is therefore removed from the autolytic ferments. Bearing on this we have to note the following facts: (*a*) That serum antitrypsin is only inhibited when the reaction is N/20 acid (lactic acid) and the serum proteolytic ferment is destroyed by a much lower H ion concentration [4]. (*b*) That serum antitrypsin in no way affects tissue autolysis except as mentioned above in virtue of the serum neutralizing and diminishing the H ion concentration [4]. (*c*) That the plasma or serum autolytic ferment is a very weak one. (*d*) That the autolytic ferments of the tissues are very sensitive to increased H ion concentration [4].

Thus we would conclude that the split products necessary for the vitality of this special class of bacteria in the tissues are not derived from the serum or tissues because of the inhibition of serum antitrypsin, but are derived from the autolytic degradation of the tissues when the tissues locally are so much damaged that the tissue fluids there are no longer capable of neutralizing the H ion present, and in proportion to the concentration of these H ions autolytic degradation occurs. The viability of the organisms in no way depends on the removal of the inhibitory power of serum antitrypsin. These organisms can derive all the food they require from the tissues, which can autolyse even in the presence of functionally active antitrypsin.

Further, Wright's [10] experiments *re* the growth of *Bacillus Welchi* in blood serum do not show the necessary relationships postulated by him—viz., sufficient acidity to destroy the antitrypsin, and thus activate the proteolytic ferment. In his *in vitro* experiments it can be seen that the vigorous growth occurs with a degree of acidity insufficient to inhibit antitrypsin and yet capable of destroying the proteolytic ferment. Then again, Wright's method of demonstrating increased activity of growth of certain bacteria when the antitrypsin is "neutralized" by the addition of definite amounts of trypsin is inaccurate, and quite different results can be obtained if the serum antitrypsin is inhibited by other methods. Now neutralization of antitrypsin by trypsin is not a true one; it is only temporary, is incomplete, and lasts until the antitrypsin has been digested by the trypsin. Antitrypsin is purely protein in nature. Thus split products are produced during the course of the experiment from this source alone even if the amount of trypsin added is insufficient to act upon the rest of the substrate during the course of the whole experiment. Wright's method of estimating the amount of trypsin necessary to inhibit the antitrypsin is determined by a preliminary experiment of shorter duration than that in which the bacterial activity is determined. Hence the amount of trypsin added is such that the antitrypsin is capable of inhibiting the action of the trypsin on the rest of the substrate for only a short time, and for the remainder of the duration of the experiment the trypsin, having destroyed the antitrypsin, acts upon the substrate, producing an abundance of split products on which the bacteria grow readily.

Experiment 66.—Some rabbit serum was taken and divided into two parts: (*a*) untreated, (*b*) treated with chloroform at room temperature for three hours to destroy the antitrypsin, evaporated to dryness *in vacuo* to remove the chloroform and made up in sterile saline to original volume; 10 c.c. of each was tubed and inoculated with one drop of an emulsion of *Bacillus Welchi* from an agar slope growth. Each was incubated anaërobically and then tested by plating out in duplicate one drop of the culture. Each test was made in duplicate, and the figures given represent an average of the counts.

	(*a*) Untreated serum	(*b*) Treated serum
Without incubation	82 colonies	77 colonies
After twenty-four hours	54 ,,	94 ,,
After forty-eight hours	120 ,,	201 ,,

Other experiments gave similar results.

Hence the removal of serum antitrypsin has very little influence on the rate of growth of *Bacillus Welchi* in the serum. In the tissues the primary factor is the reaction, and this with the consequent autolysis favours the growth of this class of organism. The serum antitrypsin plays no inhibitory part, the only inhibitory action exercised is in proportion to its alkalinity.

CONCLUSIONS.

In this paper the variations in antitrypsin in infection and anaphylactic shock are recorded.

There appears to be no relationship between the antitryptic curve and the intensity of the infection, pathogenicity or virulence of the infecting organism.

There is also no relation between the curve and the toxicity of a bacterium to the infected animal.

These experiments negative the view taken by Jobling that the toxæmia resulting from bacterial infection is due to the formation of toxic proteoses subsequent to the removal of the antitrypsin inhibition on the plasma autolytic ferments.

Further, Wright's hypothesis of the viability and consequent infectivity of certain organisms being due to the removal of the antitrypsin inhibition with the consequent formation of protein degradation bodies for their growth is negatived; the degradation bodies are apparently formed from autolysis of the tissues as the result of the increase of the H ion concentration at the site of infection.

REFERENCES.

[1] TEALE and BACH. "Nature of Serum Antitrypsin," *Proc. Roy. Soc. Med.*, 1920, xiii (Sect. Path.), p. 1 *et seq.*

[2] TEALE and BACH. *Ibid.*

[3] TEALE and EMBLETON. *Lancet*, 1913, i, p. 236.

[4] TEALE and BACH. *Proc. Roy. Soc. Med.*, 1920, xiii (Sect. Path.), p. 33.

[5] JOBLING, &c. *Journ. Exp. Med.*, 1914, xix, p. 480, xx, p. 37.

[6] VAUGHAN. *Journ. Amer. Med. Assoc.*, 1914, lxii, p. 583.

[7] TEALE and EMBLETON. *Lancet*, 1913, i, p. 237.

[8] JOBLING. *Journ. Exp. Med.*, 1915, xxii, p. 590.

[9] TEALE and BACH. "Mechanism of Removal of Bacteria from the Circulation" (in the Press).

[10] WRIGHT. *Lancet*, 1917, i, p. 6; *Proc. Roy. Soc. Med.*, 1917, x, p. 1.

Section of Pathology.

President—Dr. W. BULLOCH, F.R.S.

Sphenoidal Empyema and Epidemic Cerebro-spinal Fever.[1]

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THERE were thirty-four post-mortems made in the Netley district between 1915 and 1919 on cases of cerebro-spinal fever. These are all grouped in the ensuing table under three types:—

(A) Those dying during the acute illness, twenty-one cases.

(B) Those dying during a recrudescence, six cases.

(C) Those dying from hydrocephalus during convalescence, seven cases.

One case only (22 Mn.) died as a result of a relapse occurring months after the preliminary attack, but as he suffered from an apparently typical attack of the disease with a recrudescence he is grouped under heading (B) for convenience.

Of these thirty-four deaths, only eleven occurred within the first week of illness. In this observation we differ from Rolleston [1] who states that "more than half the fatal cases occur during the first week of the disease." A possible explanation of this difference is that at Netley we were fortunately able to insist that every case in which the least suspicion of cerebro-spinal fever was entertained should be immediately admitted to the isolation hospital. On admission lumbar puncture was performed as a routine within a few minutes of arrival, antimeningococcic serum being administered there and then to all cases showing turbid cerebro-spinal fluid. A bacteriological diagnosis was

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[1] At a meeting of the Section, held November 4, 1919.

(A) ACUTE CASES.

Name	Number of days from onset to death	Cerebro-spinal fluid type	Nasal type	Cribriform ethmoid	SPHENOIDAL SINUS		SPHENOIDAL BONE		Pituitary fossa	Middle ear
					Empyema	Meningococci	Inflammation	Meningococci		
(1) Sh.	1	N. O.	N. O.	*Nil*	Serous effusion	Not found	*Nil*	—	Inflammation	*Nil*
(2) Wh.	1	II	N. O.	,,	Yes	Not found	Yes	Not found	,,	,,
(3) Cl.	2	II	N. O.	,,	,,	II	*Nil*	—	,,	,,
(4) La.	2	II	II	,,	,,	II	,,	—	,,	,,
(5) Ha.	3	Pos.	Pos.	,,	,,	Pos.	Yes	Pos.	,,	,,
(6) Me.	3	Pos.	Pos.	,,	,,	Not found	*Nil*	—	,,	,,
(7) Wa.	3	II	N. O.	,,	Serous effusion	II	,,	—	,,	,,
(8) Es.	3	I	I	,,	Yes	Meningococci	Yes	Not found	,,	Congestion of bone
(9) Va.	4	I	N. O.	,,	,,	Meningococci	*Nil*	—	,,	Pus in right ear
(10) Hr.	5	Pos.	N. O.	,,	,,	Not found	,,	—	,,	*Nil*
(11) Ch.	7	Pos.	N. O.	,,	,,	Not found	,,	—	,,	,,
(12) An.	9	I	I	,,	,,	I	,,	—	,,	,,
(13) Ho.	10	Pos.	Pos.	,,	,,	Not found	Yes	Not found	,,	,,
(14) Po.	11	I	N. O.	,,	,,	Not found	*Nil*	—	,,	,,
(15) Cr.	16	II	II	,,	,,	Meningococci	Yes	II	,,	,,
(16) He.	19	Pos.	N. O.	,,	,,	Meningococci	*Nil*	—	,,	,,
(17) Su.	27	III	N. O.	,,	*Nil*	—	,,	—	,,	,,
(18) Hu.	34	Pos.	Pos.	,,	Yes	Pos.	Yes	Pos.	,,	,,
(19) Ti.	40	Pos.	N. O.	,,	,,	Not found	*Nil*	—	,,	,,
(20) Th.	47	Pos.	Pos.	,,	,,	Not found	,,	—	,,	,,
(21) Wt.	68	Pos.	N. O.	,,	,,	Meningococci	Yes	Not found	,,	,,

(B) CASES WITH KNOWN RECRUDESCENCES.

Name	Number of days from onset to		Cerebro-spinal fluid type	Nasal type	Cribriform ethmoid	SPHENOIDAL SINUS		SPHENOIDAL BONE		Pituitary fossa	Middle ear
	Recru-descence	Death				Empyema	Meningococci	Inflammation	Meningococci		
(22) Mu.	8	8	II	II	*Nil*	Yes	Meningo-cocci	*Nil*	—	Inflammation	*Nil*
(23) Ws.	5	9	I	I	,,	,,	I	,,	—	,,	,,
(24) Pu.	9	14	I	I	,,	,,	I	,,	—	,,	,,
(25) Co.	12	16	III	I or III	,,	,,	Meningo-cocci	,,	—	,,	,,
(26) Bo.	42	48	II	N. O.	,,	Normal	—	,,	—	,,	,,
(27) Hp.	Repeated recru-descences	99	Pos.	Pos.	,,	Yes	Meningo-cocci	,,	—	,,	,,

(C) CONVALESCENT CASES DYING FROM HYDROCEPHALUS.

Name	Number of days from onset to death	Cerebro-spinal fluid type	Nasal type	Cribriform ethmoid	SPHENOIDAL SINUS		SPHENOIDAL BONE		Pituitary fossa	Middle ear
					Empyema	Meningococci	Inflammation	Meningococci		
(28) Sp.	87	Pos.	II	*Nil*	Yes	Not found	*Nil*	—	Inflammation	*Nil*
(29) Ck.	90	Pos.	Pos.	,,	,,	Meningococci	,,	—	,,	,,
(30) Fl.	93	II	II	,,	,,	II	,,	—	,,	,,
(31) Tr.	109	Pos.	N. O.	,,	,,	Meningococci	,,	—	,,	,,
(32) Mu.	133	Pos.	I	,,	,,	I	,,	—	,,	,,
(33) Co.	147	Pos.	II	,,	,,	Not found	,,	—	,,	,,
(34) Br.	199	I	I	,,	,,	Not found	,,	—	,,	,,

N. O. = culture not obtained.
Roman numerals denote Gordon's four types of meningococcus as proved by serological tests.
Pos. = meningococcus grown, but serological tests not performed.
Meningococci = organisms having the morphological and staining characteristics of the meningococcus seen, but not grown.

only waited for when there was middle-ear disease or a head wound. This very early administration of serum, often within an hour or two only of the suspicion of cerebro-spinal fever being present, probably saved many cases who would otherwise have died during the first week.

In a previous paper [2] it was shown that of thirty-six cases in which it was possible to obtain cultures from both the nose and the cerebro-spinal fluid, in thirty-five the types of the meningococci in the two sites were identical, as proved by both agglutination and saturation tests. The irregularity shown by the remaining case has been discussed.

In no case was any inflammatory process discoverable about the cribriform plate of the ethmoid by naked-eye examination. The contents of the pituitary fossa showed inflammation in every case. The middle ear was seen to contain pus in one case, and there was congestion of the bone, but no pus, in another case. There was an empyema of the sphenoidal sinus in :—

(A) Acute case deaths: twenty out of twenty-one cases.
(B) Recrudescence deaths: five out of six cases.
(C) Hydrocephalus deaths: seven out of seven cases.

An empyema of the sphenoidal sinus is taken to mean, a cavity filled with pus, muco-pus containing fresh and autolysed pus cells or glairy fluid showing pus cells, and the mucous membrane of the sinus showing congestion. Thus in thirty-four post mortems, an empyema of the sphenoidal sinus was present in thirty-two cases. In the pus from the sphenoidal sinus: meningococci were proved serologically in eight cases; meningococci were cultured but were not tested serologically in two cases; meningococci were seen but not grown in ten cases; no meningococci were seen or grown in twelve cases.

Inflammation of the sphenoid bone over the empyematous sphenoidal sinus was not found at all in recrudescence and hydrocephalus deaths, but was found in seven cases of deaths from the acute illness. In these seven cases the meningococcus was only found three times, and the type of the meningococcus was only able to be proved once, but was in this case the same as that found in the nose and cerebro-spinal fluid.

Thus in thirty-four deaths from cerebro-spinal fever, empyemata of the sphenoidal sinus were found thirty-two times, meningococci were found twenty times in the sinus and three times in the sphenoid bone. In no other diseases were empyemata of the sphenoidal sinus observed, except during the epidemic of "Spanish influenza," when empyemata of the sphenoidal sinus were frequently found.

The relationship between an empyema of the sphenoidal sinus and cerebro-spinal fever may now be considered. The primary site of a meningococcus infection in the human body is undoubtedly the nasopharynx. The meningococcus has been demonstrated in this position not only early in the acute attack, but also in the incubation period [4], in carriers who never develop meningeal symptoms, in meningococcal pneumonia, &c. The infection is propagated from person to person as is a common cold, from nose to nose. The frequency of the carrier condition and the relative infrequency of the disease is noteworthy. It is probable that the whole population of England has been a carrier at some time or other during the last five years, and yet the total number of cases is relatively small.

It appears that the meningococcus can give rise to a nasal catarrh, and it seems probable that this is the natural disease produced by this organism, and it is only when some other factor comes into play that meningitis or septicæmia results. This other factor does not seem always to be a rise in virulence of the meningococcus, for of the camp epidemics about Southampton and Winchester during the war, it was only in one epidemic that all the cases of meningitis were due to one serological type of meningococcus. Usually two or three serological types of meningococcus were recognized in the cases of one epidemic, the carriers found in the camp corresponding with the serological types of meningococcus in the cases. Thus in the Winchester epidemic of the spring of 1917 there were eight cases, three due to Gordon's type 1 coccus, two due to Gordon's type 2, two due to type 4, and one in which the type was not proved. The carriers found were all infected with one of these three types of meningococcus; no type 3 carriers were found. On another occasion two cases occurred in one ward in a hospital within four days of one another. If serological tests had not been performed, it would have been felt certain that, either one case infected the other, or that both were infected from the same source. This was not so, as the first case was due to Gordon's type 2, and the second due to Gordon's type 1 or 3 coccus.

Some epidemics in other parts may have been produced by a particularly virulent meningococcus of one serological type, but it is evident that this is by no means a necessary condition for the outbreak of an epidemic.

The frequency of the appearance of a sphenoidal sinus empyema suggests that this might be the determining factor in the onset of the meningitic form of the disease. The empyema may be the result of an inflammatory reaction to the meningococcus, on the part of the mucous membrane lining the ostia. The fact that the meningococcus was several times isolated in pure culture from the sinus suggests that, for the inflammatory closure of the ostia, a mixed infection is not necessary, although streptococci and staphylococci were also found on occasions to be present. Thus a sphenoidal empyema might be produced by a meningococcal infection of the nasal mucosa of an individual whose resistance to the meningococcus was such that the infection resulted in a vigorous inflammatory reaction. Anatomical or pathological peculiarities in this region might also favour the production of an empyema.

In cases dying from cerebro-spinal fever, an empyema of the sphenoidal sinus was common; in only two cases in this series (17 Su. and 26 Bo.) out of thirty-four, was this condition not found. Both these cases died at a considerable number of days from the onset, twenty-seven and forty-eight respectively. Forty-seven completely recovered cases on the other hand were examined by rhinological experts, Dr. Peters and Dr. Bryant, who found that no sphenoidal empyemata were present. Chronic carriers [5] were also carefully examined bacteriologically, and no meningococcic infection of the sinus was found. Thus in dying cases an empyema was frequently found; in completely recovered cases no empyemata were discovered. A series of five cases showing symptoms of hydrocephalus were operated on for drainage of the sphenoidal sinus [6]. Of these cases all showed the presence of a sphenoidal empyema. The meningococcus was only cultivated and proved serologically in one of these cases from the sphenoidal pus obtained at the operation; a typical Gram-negative diplococcus was seen only but not grown in another case. But in two other cases, subsequent to the operation, the meningococcus reappeared in the throat in pure culture, after it had entirely disappeared for some weeks previously. Subsequent to the operations all cases appeared temporarily worse, three made complete recoveries, two died with typical hydrocephalus post mortem. Of the recovered cases one went through a typical relapse after the operation, with reappearance of the meningococcus and pus in the theca, subsequently completely recovering. From this it would seem that if early and vigorous serum treatment be given, and if the sphenoidal empyema disappear, recovery will probably occur; if, however, the empyema persist the case will, if it recover from the acute illness, either relapse or go on to hydrocephalus.

The next point under consideration is, how do the meningococci get from the nasal mucosa to the meninges? There are many possible routes:—

(1) The arachnoid prolongations round the olfactory nerves.
(2) The perineural lymphatics.
(3) Direct inflammatory extension.
(4) The pituitary route.
(5) The systemic lymphatics.
(6) The blood stream.
(7) The middle ear.

(1) *The Arachnoid Prolongations round the Olfactory Nerves.*—This route is favoured by Netter and Debré and Flexner. Netter and Debré show that dyes, Indian ink and meningococci can get from the cerebro-spinal fluid into the nose by this route, but they do not show that the reverse can take place. Their experiments show that the natural direction of the current is from the cerebro-spinal fluid to the lymphatics; it is thus difficult to see how the meningococci could progress against the stream, to gain access to the meninges, except by inflammatory extension. In none of this series of post-mortem examinations was there any naked-eye appearance of inflammation about the cribriform plate of the ethmoid.

(2) *The Perineural Lymphatics.*—In papers by Orr and Rows [7], Teale and Embleton [8], it has been shown that not only dyes and toxins, if injected into a nerve, will travel up the perineural lymphatics to the cord, but also particulate material, such as washed spores. These spores can be traced up the perineural lymphatics into the substance of the cord itself. In this way it might be possible for the meningococci to travel up the perineural lymphatics into the brain substance. Cerebrospinal fever does not, however, appear to start as an encephalitis, so, although this route would be a satisfactory one for polio-encephalitis, it does not seem suitable for cerebro-spinal meningitis, especially as it has been shown that neither toxins, nor dyes nor particulate material when passing up a perineural lymphatic, get into the cerebro-spinal fluid.

(3) *Inflammatory Extension.*—It has been suggested that meningococci could gain access to the cerebro-spinal fluid by a process of inflammatory extension through the bone separating the nose and accessory sinuses from the meninges. In this series of necropsies there is evidence of direct spread from the sphenoid sinus to the sphenoid bone, and so to the overlying meninges. In seven cases there was inflammation, and the meningococcus was found three times in the bone. In the other cases no evidence, as viewed by the naked eye, of inflammatory extension could be discovered.

(4) *The Pituitary Route.*—An infection from Luschka's pharyngeal tonsil along a possibly patent connexion between this and the pituitary gland has been suggested. Luschka's tonsil is one of the sites in the nose most richly infected with the meningococcus. In this series of necropsies the pituitary body always showed infection, but it is obvious that this would certainly occur anyhow from its intimate relationship with the cerebro-spinal fluid.

(5) *The Systemic Lymphatics.*—The ordinary flow of lymph from the nasal passages is away from the brain, and in connexions between the subarachnoid space and the lymphatic system the current is away from the meninges. If bacteria [9] are injected into the arachnoid space they rapidly appear in the lymphatics of the head and neck. If, however, a process occurs, such as an empyema of the sphenoidal sinus, the ordinary direction of lymph flow might be reversed, and bacteria be thus forced from the lymphatics of this neighbourhood towards the meninges, without showing any sign of inflammatory extension. In the case of a sphenoidal empyema large numbers of meningococci would get into the lymphatics, and by this means would also get into the blood stream. It would only be necessary for a sphenoidal empyema to exist for a very short time to produce these effects, possibly only for half an hour. If the sphenoidal empyema discharged at the end of a short time into the nose, and if the meningococci that had reached the meninges were destroyed, a recovery would ensue. If, however, the sphenoidal sinus empyema persisted, and kept on pouring fresh infection into the meninges, the patient would probably die.

(6) *The Blood Stream.*—Many workers have considered that the infection of the meninges occurs by way of the blood stream, the meningococci gaining access to the blood from the nasopharynx. This suggestion has been based, partly upon the early appearance of the meningococci in the blood, and partly upon those cases of meningococcal infection in which no meningitis occurs. The blood stream, however, would receive meningococci very early in the infection of the nasal mucosa, at any rate as soon as an empyema of the sphenoidal sinus occurred. The meningococci would reach the blood stream from this region via the deep cervical lymphatic trunks, which join the thoracic duct just before it enters the venous system in the neck. Also, as soon as the meningococci gained access to the cerebro-spinal fluid, they would appear [9] in the blood stream, both direct, via the Pacchionian bodies, and also by way of the lymphatics,

through their junctions with the sub-arachnoid space. The blood then would receive, from the very earliest stages of the disease, repeated and heavy showers of meningococci from the sphenoid region and the cerebro-spinal fluid. Thus it can be said that the early finding of the meningococcus in the blood stream is no indication that the meninges are primarily infected by this route. In the recent outbreaks of "Spanish influenza," empyema of the sphenoidal sinus was a common finding in the fatal cases, but meningitis was rarely found. It is not necessary then that an empyema should be followed by meningitis. It is thus possible that the meningococci can gain access to the blood stream from a sphenoidal empyema, and so be carried round to the meninges and set up inflammation.

(7) *The Middle Ear.*—Inflammation of this region was only found in two necropsies in this series. It is unlikely that this is the portal of entry of the meningococcus to the meninges.

Thus it would appear that it is improbable that the meningococci gain access to the meninges through the arachnoid prolongations round the olfactory nerves, or by way of the perineural lymphatics. It is probable that the meningococci pass by way of the lymphatics from the sphenoidal sinus direct to the meninges, with or without naked-eye signs of inflammation. It is possible also that they can be conveyed to the meninges by the blood stream.

The relationship between a persistent sphenoidal empyema and hydrocephalus may now be considered. In the seven deaths from this condition an empyema was found in each case. Five cases were operated on, and pus was found in the sphenoidal sinus each time; two of these cases died and are grouped in with the post-mortem cases. There was thus a total of ten cases of hydrocephalus, and in each case a sphenoidal empyema was found. The cerebro-spinal fluid in the ventricles of each of the cases which died contained pus cells. The cerebro-spinal fluid taken from the lumbar region was usually quite clear. This suggests that there is a close relationship between empyema of the sphenoidal sinus and the hydrocephalus that develops after an attack of cerebro-spinal fever. The hydrocephalus develops as a result of chronic infection about the foramina of Luschka and Magendie. This chronic infection also appears to exist in the ventricles. The exact path by which the meningococci in the sphenoidal sinus empyema reach the interior of the ventricles is not clear, unless it be by the blood stream. But there would appear to be some relationship between the two positions, inasmuch as the only three patients who recovered from the hydrocephalous condition did so after an operation for drainage of the sphenoidal sinus.

The course of events in a case after infection of the nasal mucous membrane with the meningococcus might be as follows :—

(1) A simple catarrh, followed either by recovery or a chronic infection.

(2) A vigorous local reaction in the nasal mucosa followed by sphenoidal empyema. This may produce a general blood infection, with or without infection of the meninges.

If the sphenoidal empyema disappears, the body, with or without the aid of antiserum, may be able to deal with the meningococci that have gained access to it, or death may result. If the sphenoidal empyema persists, and very heavy discharges of meningococci are poured into the body, death will probably result. If, however, the empyema remains quiescent, improvement may occur, but at any time a recrudescence or a relapse may supervene. A quiescent empyema may also keep up a smouldering infection which will lead to hydrocephalus.

REFERENCES.

[1] ROLLESTON, Sir H. D. "Lumleian Lectures on Cerebro-Spinal Fever," *Lancet*, 1919, i, p. 647.

[2] EMBLETON and STEVEN. "Persistence of Cerebro-spinal Fever Cases as Carriers of the Meningococcus during Convalescence," *Lancet*, 1919, i, p. 789.

[3] WESTENHOEFFER. *Berl. klin. Wochenschr.*, 1905, lxii, p. 737.

[4] FILDES. Report to Medical Research Committee.

[5] EMBLETON and BRYANT. "Site of Carrying in Chronic Meningococcus Carriers," *Lancet*, 1919, ii, p. 679.

[6] PETERS. *Proc. Roy. Soc. Med.*, 1919, xii (Sect. Laryng.), p. 250.

[7] ORR and ROWS. *Edin. Med. Journ.*, 1916, xvii, pp. 78, 89.

[8] TEALE and EMBLETON. *Journ. Path. and Bact.*, 1919, xxiii, pp. 50-68.

[9] TEALE and EMBLETON. "Infection. Paths of Spread in Bacterial Infection." *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Path.), pp. 69-96.

Section of Pathology.

President—Dr. W. BULLOCH, F.R.S.

The Factors leading to the Removal of Bacteria from the Peripheral Circulation and Phagocytosis.[1]

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IN a previous paper by Teale and Embleton [1] it was noted that bacteria inoculated into the circulation rapidly disappeared from the peripheral circulation and could then be demonstrated only in the internal organs such as the lung, liver, spleen and marrow. Wyssokowitsch [2] noted that the bacteria rapidly disappeared from the blood, and expressed the view that it was simply due to a process of filtration in the capillaries. Werigo [3] showed that virulent anthrax bacilli are rapidly phagocytosed by the leucocytes and fixed phagocytes in the circulation of the rabbit. Bull [4] in a series of papers draws the conclusion that the process is one of agglutination and only occurs if the animal inoculated has an agglutinin against the bacterium inoculated. Metchnikoff [5] believed that agglutination played no part in active or passive immunity processes. Salimbeni [6] came to the same conclusion. Sawtschenko and Melkick [7] believed that agglutination of the spirilla of relapsing fever occurred in the plasma, but incompletely because of the rapid movement of the blood.

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[1] At a meeting of the Section, held February 3, 1920.

[2] The expenses of the research were defrayed by a grant from the Medical Research Committee.

The subject of the removal of bacteria and the factors causing it were investigated in the following series of experiments:—

(A) Experiments dealing solely with the disappearance of different types of bacteria.

- (1) Those against which the animal normally has some type of antibody.
- (2) Those against which the animal has no antibody.
- (3) Those which kill the animal relatively slowly.
- (4) The differences with inoculation into normal and immune animals.
- (5) Virulent bacteria.

(B) The mechanism of removal.

- (*a*) Phagocytosis by free and fixed cells.
- (*b*) Factors preliminary to phagocytosis.

(C) The mechanism of removal by sera, &c., during secondary bacteriæmia.

Rabbits were used for these experiments. The ears were shaved, sterilized by rubbing with lysol, the lysol was removed with alcohol, the ear was then dried and warmed. The marginal vein on one side was cut, lightly clipped, and covered with sterile wool. The injection was made into the opposite marginal vein, blood was allowed to flow from the cut marginal vein at intervals into sterile watch glasses and accurately measured. Amounts of 100 c.mm. of this blood were taken and blown into 6-in. Petri dishes containing a small quantity of broth. The pipette was then washed out with nutrient broth, the washings being added to the contents of the Petri dish; after careful mixing, liquefied agar, rebipelagar or other medium was added and the whole intimately mixed. The number of colonies developing was counted after twenty-four hours or longer, depending on the rate of the growth of the organisms experimented with. All experiments were made in duplicate and the average of the results taken.

(A) (1) ORGANISMS AGAINST WHICH THE RABBIT HAS AN AGGLUTININ OR BACTERIAL ANTIBODY.

Experiment 1: Bacillus typhosus.—A quarter of an agar streak slope of a laboratory strain was inoculated intravenously into a rabbit of 2,090 grm. Agglutinating titre of the serum against *Bacillus typhosus* 1 : 10.

Number of Colonies per cubic centimetre in the Blood.

1 minute after inoculation	50,000
5 minutes ,, ,,	32,000
15 ,, ,, ,,	4,000
30 ,, ,, ,,	1,500
60 ,, ,, ,,	300
2 hours ,, ,,	200
3 ,, ,, ,,	320
4 ,, ,, ,,	40
8 ,, ,, ,,	*nil*
12 ,, ,, ,,	20
24 ,, ,, ,,	160
36 ,, ,, ,,	*nil*
48 ,, ,, ,,	*nil*

Rabbit recovered.

Experiment 2.—One slope of *Bacillus typhosus* was inoculated intravenously into a rabbit of 2,560 grm. Agglutination titre *nil.*

Colonies in the Blood per cubic centimetre.

1 minute after inoculation	800,000
5 minutes ,, ,,	50,000
15 ,, ,, ,,	3,000
30 ,, ,, ,,	1,000
60 ,, ,, ,,	1,000
4 hours ,, ,,	600
8 ,, ,, ,,	250
12 ,, ,, ,,	10
24 ,, ,, ,,	180
48 ,, ,, ,,	*nil*

Animal recovered.

Experiment 3: Bacillus typhosus.—Rabbit weighing 1,850 grm. inoculated intravenously with half a twenty-four hours' growth agar slope. Agglutinating titre 1 : 30. Animal died shortly after two hours after the injection.

Colonies per cubic centimetre after 1 minute	500,000
,, ,, ,, ,, 5 minutes	80,000
,, ,, ,, ,, 15 ,,	8,000
,, ,, ,, ,, 30 ,,	5,000
,, ,, ,, ,, 60 ,,	1,000
,, ,, ,, ,, 2 hours	200

Experiment 4: Bacillus coli.—Half a slope of a twenty-four hours old agar streak culture was inoculated intravenously into a rabbit of 2,520 grm. Agglutination titre 1 : 20. Animal died in two hours.

Colonies per cubic centimetre of Blood.

1 minute after inoculation	200,000
5 minutes ,, ,,	80,000
10 ,, ,, ,,	25,000
15 ,, ,, ,,	10,000
30 ,, ,, ,,	6,250
60 ,, ,, ,,	3,000
90 ,, ,, ,,	2,500
2 hours ,, ,, (Just after death)	240

Experiment 5: Bacillus mycoides.—A non-virulent culture growing at 37° C. [7] was used, a quarter of a twenty-four hours' agar slope culture being inoculated intravenously into a rabbit of 1,890 grm.

Colonies per cubic centimetre of Blood.

1 minute after inoculation	400,000
5 minutes ,, ,,	20,000
10 ,, ,, ,,	5,000
20 ,, ,, ,,	750
30 ,, ,, ,,	550
60 ,, ,, ,,	180
2 hours ,, ,,	100
6 ,, ,, ,,	*nil*
12 ,, ,, ,,	120
24 ,, ,, ,,	*nil*

Experiment 6: Bacillus anthracis.—An ordinary slightly virulent laboratory strain was used. A quarter of a twenty-four hours' agar slope culture was inoculated intravenously into a rabbit of 2,100 grm.

Colonies per cubic centimetre of Blood.

1 minute after inoculation	480,000
5 minutes ,, ,,	36,000
15 ,, ,, ,,	7,500
30 ,, ,, ,,	1,000
60 ,, ,, ,,	850
2 hours ,, ,,	500
4 ,, ,, ,,	150
6 ,, ,, ,,	500
24 ,, ,, ,,	*nil*

Died in 48 hours.

In the experiments recorded so far organisms were used against which the serum of the rabbit is naturally usually agglutinative (*Bacillus typhosus*) or bactericidal.

(A) (2) THE NEXT EXPERIMENTS WERE PERFORMED WITH ORGANISMS AGAINST WHICH THE RABBIT HAS NO APPARENT AGGLUTININ, AND WHICH ARE VERY PATHOGENIC TO THE RABBIT.

Experiment 7 : Bacillus dysenteriæ (Shiga).—Half an agar slope of a twenty-four hours' culture was inoculated intravenously into a rabbit of 1,960 grm.

Number of Colonies in 1 cubic centimetre of the Blood.

1 minute after inoculation	250,000
15 minutes ,, ,,	40,000
30 ,, ,, ,,	2,400
1 hour ,, ,,	500
3 hours ,, ,,	300
12 ,, ,, ,,	2.400
24 ,, ,, ,,	150

The animal died in 36 hours.

Experiment 8.—Half an agar slope of a twenty-four hours' culture of *Bacillus dysenteriæ* (Shiga) was inoculated intravenously into a rabbit of 2,120 grm.

Number of Colonies per cubic centimetre of Blood.

1 minute after inoculation	200,000
15 minutes ,, ,,	24.000
30 ,, ,, ,,	3,200
60 ,, ,, ,,	800
3 hours ,, ,,	320
8 ,, ,, ,,	50
12 ,, ,, ,,	400
24 ,, ,, ,,	250

Died in 30 hours.

In view of the results of Bull [8] with this organism other experiments were made with the same organism and with similar results, thus differing from those of Bull.

Experiment 9 : Bacillus of Chicken Cholera.—One-third of a forty-eight hours' culture on an agar slope was inoculated intravenously into a rabbit of 1,800 grm.

Number of Colonies per cubic centimetre of Blood.

1 minute after inoculation	84,000
5 minutes ,, ,,	25,000
15 ,, ,, ,,	4,000
30 ,, ,, ,,	1,500
60 ,, ,, ,,	450
2 hours ,, ,,	100
6 ,, ,, ,,	350
10 ,, ,, ,,	150
24 ,, ,, ,,	8,000

Died in 30 hours.

Experiment 10: Bacillus suicholeræ (Bacillus of Hog Cholera).—One-third of a twenty-four hours' culture on an agar slope was inoculated into a rabbit weighing 2,000 grm.

Number of Colonies developing per cubic centimetre of Blood.

1 minute after injection	200,000
5 minutes ,, ,,	15,000
15 ,, ,, ,,	4,000
30 ,, ,, ,,	1,000
60 ,, ,, ,,	200
2 hours ,, ,,	200
3 ,, ,, ,,	*nil*
6 ,, ,, ,,	420

Died in 12 hours.

(A) (3) ANIMALS DYING LATER THAN TWENTY-FOUR HOURS AFTER INOCULATION, TO NOTE PERIODIC RECURRENCE OF BACTERIA IN THE CIRCULATION AND THE FINAL BACTERIÆMIA.

Experiment 11: Virulent Bacillus mycoides.—Rabbit, weighing 1,760 grm., was given half a slope of a twenty-four hours' culture intravenously.

Number of Colonies developing from 1 cubic centimetre of Blood.

1 minute after inoculation	800,000
15 minutes ,, ,,	20,000
30 ,, ,, ,,	8,000
1 hour ,, ,,	500
2 hours ,, ,,	150
6 ,, ,, ,,	*nil*
24 ,, ,, ,,	3,500
30 ,, ,, ,,	24,000

Died in 32 hours.

Experiment 12: Virulent Bacillus mycoides.—A rabbit, weighing 2,680 grm., was given half a slope intravenously. It lived about sixty hours.

Number of Colonies developing from 1 cubic centimetre of Blood.

1 minute after inoculation	560,000	per c.c.
30 minutes ,, ,,	2,500	,,
1 hour ,, ,,	240	,,
3 hours ,, ,,	100	,,
6 ,, ,, ,,	*nil*	
8 ,, ,, ,,	*nil*	
24 ,, ,, ,,	500	,,
30 ,, ,, ,,	100	,,
48 ,, ,, ,,	320	,,
56 ,, ,, ,,	20,000	,,

Animal very ill.

Experiment 13: Bacillus dysenteriæ (Shiga).—A rabbit, weighing 2,500 grm., inoculated intravenously with a quarter of a twenty-four hours' culture on agar slope.

Number of Colonies developing from 1 cubic centimetre of Blood.

1 minute after inoculation	120,000	per c.c.
30 minutes ,, ,,	1,600	,,
1 hour ,, ,,	500	,,
2 hours ,, ,,	100	,,
4 ,, ,, ,,	260	,,
8 ,, ,, ,,	1,400	,,
24 ,, ,, ,,	*nil*	,,
32 ,, ,, ,,	200	,,
48 ,, ,, ,,	*nil*	,,
56 ,, ,, ,,	860	,,

Died overnight.

Experiment 14: Bacillus of Chicken Cholera.—A rabbit, weighing 2,045 grm., was inoculated with one-third of an agar slope intravenously. Animal died in thirty-six hours.

1 minute after inoculation	32,000
10 minutes ,, ,,	1,200
1 hour ,, ,,	180
2 hours ,, ,,	40
4 ,, ,, ,,	*nil*
6 ,, ,, ,,	40
8 ,, ,, ,,	40
12 ,, ,, ,,	*nil*
24 ,, ,, ,,	200
32 ,, ,, ,,	3,800

Experiment 15: Washed Spores.—"Washed" spores of hot growing *Bacillus mycoides*. A well-spored culture was washed up in saline, heated to 80° C. for three hours to "wash" the spores, and 0·5 c.c. of the thick emulsion was inoculated into a rabbit, weighing 2,020 grm., intravenously.

Number of Colonies developing per cubic centimetre of Blood.

1 minute after inoculation	50,000
5 minutes ,, ,,	10,000
10 ,, ,, ,,	2,000
15 ,, ,, ,,	800
30 ,, ,, ,,	800
60 ,, ,, ,,	400
120 ,, ,, ,,	100
180 ,, ,, ,,	*nil*
5 hours ,, ,,	*nil*
12 ,, ,, ,,	*nil*
24 ,, ,, ,,	*nil*
48 ,, ,, ,,	*nil*

This experiment shows that spores are removed in the same way from the peripheral circulation as vegetative forms.

(A) (4) IMMUNE *v.* NORMAL ANIMALS.

Experiments to note the effect of active immunity on removal of bacteria from the peripheral blood.

Experiment 16: Bacillus typhosus.—Two rabbits, one normal the other immunized, were inoculated intravenously with half a slope each of a twenty-four hours' agar slant culture.

Colonies developing per cubic centimetre	Normal rabbit, 2,095 grm. Agg. titre 1 : 30	Immune rabbit, 1,950 grm. Agg. titre 1 : 10,000
1 minute after inoculation	160,000	200,000
5 minutes ,, ,,	48,000	54,000
15 ,, ,, ,,	4,000	5,000
30 ,, ,, ,,	1,500	2,500
60 ,, ,, ,,	300	500
2 hours ,, ,,	200	250
3 ,, ,, ,,	2,400	1,600
24 ,, ,, ,,	40	500

Experiment 17: Bacillus typhosus.—Half a slope of a twenty-four hours' agar slant culture inoculated intravenously into a rabbit as follows:—

Number of colonies developing per cubic centimetre	Normal, 1,860 grm., titre *nil*	Immunized, 2,126 grm., titre 1 : 20,000
1 minute after inoculation	400,000	400,000
5 minutes ,, ,,	25,000	21,000
15 ,, ,, ,,	1,500	2,500
30 ,, ,, ,,	500	300
60 ,, ,, ,,	500	500
4 hours ,, ,,	300	100
6 ,, ,, ,,	960	500
24 ,, ,, ,,	120	*nil*
	Recovered	Recovered

Experiment 18: Pneumobacillus.—Into each rabbit, normal and immune, was inoculated intravenously half of a mixture of two agar slopes of a twenty-four hours' culture of the pneumobacillus. The immune rabbit had received six injections of dead and three of living bacilli, the last more than fourteen days before the experiment.

Number of colonies developing from 1 c.c. of blood	Normal rabbit 2,100 grm.	Immune rabbit 2,400 grm.
1 minute after inoculation	4,000,000	5,000,000
5 minutes ,, ,,	680,000	—
15 ,, ,, ,,	50,000	3,000,000
30 ,, ,, ,,	8,000	12,000
60 ,, ,, ,,	8,000	8,000
2 hours ,, ,,	4,000	1,000
4 ,, ,, ,,	1,000	2,500
24 ,, ,, ,,	200	120
	Recovery	Recovery

Experiment 19: Bacillus dysenteriæ (Shiga).—Experiments were next carried out with this organism since the normal animal has no agglutinin normally present against it. Hence the contrast between the normal and immune animal would be more complete than in the case of *Bacillus typhosus*. The rabbits were immunized according to the method published by Miss Chick [10]. Into each rabbit was injected intravenously half an agar slope of a twenty-four hours' culture of *Bacillus dysenteriæ* (Shiga).

Number of colonies developing per cubic centimetre		Normal rabbit, 2,860 grm., agg. titre = 0		Immune rabbit, 3,020 grm., titre 1/250 against *B. dysenteriæ* (Shiga)
1 minute after inoculation	...	200,000	...	220,000
5 minutes ,, ,,	...	15,000	...	20,000
15 ,, ,, ,,	...	4,000	...	6,500
30 ,, ,, ,,	...	1,000	...	2,000
60 ,, ,, ,,	...	*nil*	...	1,000
2 hours ,, ,,	...	*nil*	...	1,000
4 ,, ,, ,,	...	20	...	*nil*

Experiment 20: Bacillus suicholeræ.—A normal and an immune rabbit were inoculated intravenously with one half of a twenty-four hours' agar culture. The immune animal had received four inoculations of dead bacilli, the last three weeks before the experiment.

Colonies per cubic centimetre of blood developing		Normal animal		Immune animal agg. titre 1 : 1,000
1 minute after inoculation	...	400,000	...	480,000
5 minutes ,, ,,	...	55,000	...	70,000
15 ,, ,, ,,	...	10,000	...	15,000
30 ,, ,, ,,	...	1,500	...	3,000
60 ,, ,, ,,	...	500	...	1,000
2 hours ,, ,,	...	100	...	500
4 ,, ,, ,,	...	800	...	200
		Died overnight		Died in 6 hours

(A) (5) VIRULENCE.

Experiment 21.—A very virulent culture of *Bacillus anthracis* was obtained and half of a twenty-four hours' agar slope culture was inoculated intravenously into a rabbit of 2,120 grm. The rabbit died in less than twenty-four hours.

Number of Colonies per cubic centimetre of blood.

1 minute after inoculation	...	..	...	..	...	420,000
5 minutes ,, ,,	...	...	...	...	...	50,000
15 ,, ,, ,,		...	...	...	...	12,000
30 ,, ,, ,,	...	...	...	...	...	2,400
60 ,, ,, ,,	..	...	...	...	..	500
2 hours ,, ,,	...	...	...	...	...	300
4 ,, ,, ,,	...	...	...	...	...	1,800
8 ,, ,, ,,	...	...	...	...	...	150
10 ,, ,, ,,	...	...	...	...	. .	*nil*
12 ,, ,, ,,	...	...	...	...	...	*nil*

Died during the night.

Experiment 22: *Bacillus coli.*—Half of a slope of a very virulent twenty-four hours' agar culture was inoculated intravenously into a rabbit of 1,860 grm. The rabbit died in four hours.

Number of Colonies per cubic centimetre of Blood.

1 minute after inoculation	340,000
5 minutes ,, ,,	60,000
15 ,, ,, ,,	14,000
30 ,, ,, ,,	2,800
60 ,, ,, ,,	800
2 hours ,, ,,	150
4 ,, ,, ,,	250

Experiment 23: *Virulent Streptococcus.*—1 c.c. of a serum broth culture (virulent to rabbits) was inoculated intravenously into a rabbit of 1,620 grm.

Number of Colonies per cubic centimetre.

1 minute after inoculation	68,000
5 minutes ,, ,,	5,000
15 ,, ,, ,,	820
30 ,, ,, ,,	600
60 ,, ,, ,,	100
2 hours ,, ,,	32
4 ,, ,, ,,	860

Animal died overnight.

Experiment 24: *Pneumococcus.*—Two animals of nearly same size and weight were inoculated intravenously with non-virulent and virulent pneumococci. Each received 2 c.c. of a broth culture.

	Rabbit, 1,940 grm. Virulent organism	Rabbit, 2,005 grm. Non-virulent organism
1 minute after inoculation	220,000	280,000
15 minutes ,, ,,	18,000	10,000
30 ,, ,, ,,	5,000	3,500
60 ,, ,, ,,	800	1,000
2 hours ,, ,,	200	50
4 ,, ,, ,,	10	*nil*
8 ,, ,, ,,	*nil*	*nil*
24 ,, ,, ,,	2,000	100
30 ,, ,, ,,	Died in 30 hours	*nil*
36 ,, ,, ,,		*nil*
72 ,, ,, ,,		180
96 ,, ,, ,,		32,000
		Died in about 120 hours

From these experiments it is obvious that bacteria rapidly disappear from the peripheral blood stream and as shown by the previously published experiments [1] from the heart's blood.

(1) The disappearance apparently does not depend on the presence of an agglutinating antibody in the blood of the infected animal, nor upon the rapid bactericidal action of the blood as judged by the known action of the serum *in vitro*.

(2) The pathogenicity of the organism has little influence on the rate of disappearance, since bacteria which are invariably fatal to rabbits, the *Bacillus dysenteriæ* (Shiga), *Bacillus suicholeræ* and the chicken cholera bacillus disappear as readily as others which are not pathogenic or less constantly so. This is in agreement with the result obtained by C. Ten Broeck [11] with *Bacillus shiga* and *Bacillus suicholeræ*.

(3) Increasing the virulence of the bacterium only slightly affects the disappearance from the circulation in the initial stages of the infection. This agrees with the results of Hopkins and Parker [14] and is contrary to those of Bull.

(4) With a fatal issue the bacteria may reappear in the circulation in large numbers or the animal may die before such secondary bacteriæmia can occur.

(5) The rate of removal is exactly the same even if the animal dies very rapidly (in two hours) from the inoculation (cf. Experiments 3 and 4).

In addition there are small secondary waves of reappearance of bacteria in the peripheral circulation.

(B) EXPERIMENTS BEARING ON THE MECHANISM OF THE REMOVAL OF BACTERIA FROM THE PERIPHERAL CIRCULATION.

(*a*) *Phagocytosis by fixed Cells and Leucocytes.*

The experiments under section (A) clearly prove that the removal is not due to the agglutinative properties of the blood plasma.

In the experiments previously published by Embleton and one of us [1] it was seen that the bacteria, whilst absent from the heart's blood, were present in large numbers in the liver, spleen, lungs and bone-marrow, and could be demonstrated there for long periods. Hence the rapid disappearance from the peripheral circulation and heart's blood cannot be due to a rapid germicidal action of the blood plasma. C. T. Bull [4] as the results of his experiments came to the conclusion that the removal of bacteria from the circulation was due to the action of agglutinins in the plasma, thus facilitating very active phagocytosis in the capillaries, especially of the lungs and liver.

From our experiments we note that phagocytosis occurs in the capillaries of the liver, lungs and spleen especially. The phagocytosis is effected not only by the leucocytes of the blood but by the fixed cells in the liver, spleen and lungs. In the animals experimented upon both wandering and fixed phagocytes bore a part in the removal of the bacteria. No difference was noted in this respect with any of the bacteria experimented with, whether pathogenic or not. It was also found that no bacterium was specially phagocytosed by one type of cell and not by the other. This is contrary to the results obtained by Preston Kyes [12] in the case of pneumococcal infection in the pigeon. This bird is very immune to the pneumococcus, and Kyes states that the phagocytosis of this bacterium is wholly carried out by the fixed phagocytes of the liver and spleen, the wandering phagocytes apparently playing no part at all. Our experiments further show that there is no difference in the degree of rapidity of phagocytosis in an immune animal as compared with a normal one. There appears to be no evidence that the antibodies, normal or acquired, act as opsonins. In smears made from the capillary blood it appears that the bacteria are taken up very irregularly, about 10 per cent. of the polymorphonuclear neutrophiles contained bacteria, and these had engulfed large numbers so that the distribution was just like that in the phagocytosis *in vitro* in the absence of opsonin.

Finally, our experiments show that the mechanism underlying the removal of the bacteria in the capillaries of the liver, lungs, &c., is the entanglement of the bacteria into loose clumps by a mass of blood platelets and gelatinous material (? incipient fibrin). The leucocytes then get into contact with the periphery of these masses of entangled bacteria, and are thus enabled to take up the bacteria readily in large numbers from these clumps. Later the platelets and gelatinous material disappear, and only the bacteria-laden phagocytes remain. This can be readily seen from the drawings made from photographs (figs. 7-13, pp. 101-104).

All the films made for these observations were spread thinly and as evenly as possible and rapidly dried. The emulsions of bacteria used for the inoculations were mechanically shaken for separation as in vaccine preparation. In all the films it was noted that the bacteria did not remain separate long, even quite early after the inoculation—three minutes—the majority of the bacteria were collected into loose irregular groups, lying in a gelatinous-looking mass, which apparently consisted of blood platelets and incipient fibrin.

In the films made from experiments shortly after the inoculation—one to three minutes—these collections of bacteria, more or less closely aggregated, lying in the mass of blood platelets, were not phagocytosed to any appreciable degree: the leucocytes in the majority of cases not being in very close contact. Later, five minutes or more, usually several phagocytes were noted in the neighbourhood of and, apparently, inside the mass of blood platelets, actively taking up the bacteria. Finally the process of phagocytosis was completed, and the blood platelet masses disappeared.

It might be argued that these masses with their included bacteria were artefacts, and were not present *in vivo*, but were formed during the process of drying and preparing the film. If this were so it is difficult to explain—

(1) How such relatively large masses of blood platelets, &c., should occur so regularly in these blood films made thinly and dried quickly, as it is well known that ordinary blood films made from blood just about to coagulate show scattered platelets or only very small groups.

(2) How these masses of platelets should be able to entangle the bacteria and draw them away from a wide surrounding area and leave the other cells undisturbed.

(3) How later the leucocytes become involved in these masses of platelets and bacteria.

It is difficult to believe that the relatively large leucocytes could be drawn in from a wide surrounding area by a simple process of entanglement and shrinkage in early clot formation even if it were so with bacteria. Again, it is practically only polymorphonuclear leucocytes which are seen in these clumps.

These clumps of leucocytes, bacteria and blood platelets occur with such unfailing regularity in all specimens made, and are practically the only places where there is evidence of bacteria or of phagocytosis, that it seems to us to be absolutely indicated that the removal of bacteria from the blood is due to the entanglement of the bacteria in the masses of blood platelets and gelatinous fibrinous material (the bacteria acting as foreign particles) and subsequent phagocytosis. Ten Broeck [11] in his experiments with *Bacillus suicholeræ* notes the same gelatinous masses entangling these bacteria.

The following experiments were performed to observe and compare the rate of phagocytosis of different bacteria in the various tissues after intravenous inoculation. Films were made from the various tissues and stained with Leishman's stain. A large number of areas of the film were examined, the number of free and phagocytosed bacteria and percentage of leucocytes with bacteria were enumerated.

Experiment 25.—Experiment with a non-pathogenic non-multiplying bacterium, the ordinary cold growing *Bacillus mycoides.* A series of guinea-pigs was inoculated intravenously each with half a slope and the animals killed after—

		Heart's blood Per cent.	Lung Per cent.	Liver Per cent.	Spleen Per cent.	Marrow Per cent.
5 minutes	(*a*)	—	4·0	5·0	34·0	5·0
	(*b*)	—	1·0	1·0	5·0	0·5
15 ,,	(*a*)	—	50·0	50·0	34·0	5·0
	(*b*)	—	5·0	5·0	5·0	0·5
30 ,,	(*a*)	—	60·0	40·0	70·0	100·0
	(*b*)	—	5·0	10·0	15·0	5·0
60 ,,	(*a*)	—	50·0	40·0	90·0	100·0
	(*b*)	—	15·0	10·0	10·0	5·0
90 ,,	(*a*)	—	50·0	60·0	90·0	100·0
	(*b*)	—	10·0	10·0	10·0	5·0
6 hours	(*a*)	—	90·0	95·0	90·0	100·0
	(*b*)	—	10·0	10·0	5·0	2·0

(*a*) = Percentage of bacteria phagocytosed.
(*b*) = Percentage of phagocytes with bacteria.

Experiment 26.—A similar experiment with virulent *Bacillus mycoides* (37° C.). A quarter of a twenty-four hours' agar slope culture inoculated intravenously into each of a series of guinea-pigs.

		Heart's blood Per cent.	Lung Per cent.	Liver Per cent.	Spleen Per cent.	Marrow Per cent.
5 minutes	(*a*)	—	11·0	1·0	70·0	70·0
	(*b*)	—	15·0	1·0	12·0	1·0
10 ,,	(*a*)	—	35·0	1·0	80·0	70·0
	(*b*)	—	16·0	1·0	45·0	1·0
15 ,,	(*a*)	—	25·0	20·0	80·0	70·0
	(*b*)	—	15·0	5·0	45·0	1·0
30 ,,	(*a*)	—	60·0	50·0	80·0	90·0
	(*b*)	—	10·0	10·0	40·0	1·0
90 ,,	(*a*)	—	50·0	50·0	90·0	90·0
	(*b*)	—	20·0	5·0	10·0	1·0
3 hours	(*a*)	—	70·0	80·0	95·0	90·0
	(*b*)	—	15·0	10·0	10·0	1·0

Experiment 27: with Bacillus dysenteriæ (Shiga).—A quarter of a twenty-four hours' agar streak was used for each experiment; methods as before; inoculated intravenously into guinea-pigs.

		Lung Per cent.	Liver Per cent.	Spleen Per cent.	Marrow Per cent.
5 minutes	(*a*)	5·0	5·0	20·0	10·0
	(*b*)	4·0	2·0	5·0	1·0
15 ,,	(*a*)	40·0	50·0	60·0	80·0
	(*b*)	10·0	15·0	10·0	1·0
30 ,,	(*a*)	60·0	40·0	80·0	90·0
	(*b*)	10·0	10·0	15·0	5·0
60 ,,	(*a*)	40·0	50·0	40·0	90·0
	(*b*)	5·0	15·0	10·0	1·0

Similar results were noted with *Bacillus dysenteriæ* (Flexner).

Experiment 28: with Bacillus typhosus.—Comparison between normal and immune animals. The immune guinea-pigs had a titre of 1 : 2,000.

		Lung		Liver		Spleen		Marrow	
		Normal	Immune	Normal	Immune	Normal	Immune	Normal	Immune
		Per cent.		Per cent.		Per cent.		Per cent.	
5 minutes	(*a*) ...	5·0	3·0	5·0	2·0	30·0	40·0	10·0	10·0
	(*b*) ...	5·0	1·0	2·0	2·0	5·0	7·0	1·0	0·5
15 ,,	(*a*) ...	56·0	30·0	40·0	40·0	60·0	60·0	40·0	40·0
	(*b*) ...	15·0	10·0	10·0	10·0	15·0	10·0	1·0	1·0
30 ,,	(*a*) ...	60·0	70·0	40·0	50·0	60·0	80·0	80·0	60·0
	(*b*) ...	15·0	15·0	10·0	15·0	10·0	15·0	5·0	3·0
60 ,,	(*a*) ...	80·0	70·0	60·0	50·0	70·0	70·0	80·0	80·0
	(*b*) ...	10·0	10·0	10·0	10·0	10·0	10·0	5·0	5·0

From these experiments it will be seen that—

(I) (*a*) Non-pathogenic organisms, (*b*) virulent organisms, (*c*) pathogenic organisms, are phagocytosed at about the same rate and about the same percentage of leucocytes acts as of phagocytes.

(II) The phagocytosis in the immune animal is the same as in the non-immune and the circulatory antibodies appear to take no part in the preliminary removal of bacteria from the circulation.

(B) (*a*) For the experiments relating to phagocytosis by the fixed cells, three bacteria only were used, viz., a virulent *Bacillus mycoides* as type of highly virulent pathogenic organism, *Bacillus Hoffmann* as an example of a bacterium which was practically non-pathogenic, and *Bacillus coli* as a Gram-negative organism. The cultures were inoculated intravenously, the animals killed after definite intervals, and the organs removed so as to keep the vessels distended. They were fixed in formalin and Müller's solution, and stained by the eosin-Gram-Weigert method, or by Loeffler's method.

Experiment 29: Bacillus mycoides.—One twenty-four hour agar slope was inoculated. *Five minutes:* No fixed tissue cell phagocytosis could be seen either in the spleen or the lung. *Fifteen minutes: Liver*—The Kupffer's cells showed the presence of *Bacillus mycoides* inside them. *Spleen*—*Bacillus mycoides* present in leucocytes and fixed tissue cells at the periphery of the Malpighian corpuscle; none in centre of the corpuscle; sinuses contained leucocytes with bacteria, also showed phagocytosis by the endothelial cells of the sinuses. *Lung*—Showed no fixed tissue cell phagocytosis. *One hour:* Spleen and liver the same as above only more marked; lung also marked phagocytosis by the endothelial cells.

Experiment 30 : Bacillus Hoffmann.—Three slopes were used for each inoculation. *Five minutes* : No fixed tissue cell phagocytosis seen, but the leucocytes showed phagocytosis. *Fifteen minutes : Liver*—The Kupffer's cells had taken up numerous bacteria. *Spleen*—In addition to the phagocytosis by leucocytes in the periphery of the Malpighian corpuscle, the fixed splenic cells had taken up large numbers, as well as endothelial cells of sinuses. *Lung*—The endothelial cells of the capillaries also contained large numbers of bacteria. *One hour* : Same as fifteen minutes. *Six hours :* Same as fifteen minutes, only more marked (vide plates).

Experiment 31 : Bacillus coli.—One slope of *Bacillus coli* was inoculated intravenously into each of a series of guinea-pigs. *Five minutes :* No apparent phagocytosis by the fixed tissue cells : the leucocytes show some. *Fifteen minutes : Liver*—The Kupffer's cells contain numerous *Bacillus coli. Spleen :* At the periphery of the Malpighian corpuscle the fixed, oval, elongated and round tissue cells contain bacteria ; the leucocytes in the sinuses and the endothelial cells of the sinuses contain bacteria. *Lung*—Endothelial cells of capillaries contain bacteria.

(*b*) *Consideration of the Factors leading to Acceleration of the Phagocytosis of Bacteria in the Circulation.*

At first we were unable to obtain leech extract to render the blood incoagulable or greatly delay its coagulability, so our preliminary experiments were made on cats, the coagulability of whose blood is greatly delayed by peptone.

Experiment 32.—Two cats were anæsthetized with ether : one slope of *Bacillus coli* was inoculated into the jugular vein of each, and one was given 4 grm. of peptone per kilo five minutes before the bacterial inoculation. Specimens of blood were taken by puncturing the opposite jugular vein in a different place each time.

Number of Colonies developing from 1 cubic centimetre of Blood.

	Control	Peptone animal
1 minute after inoculation ...	200,000 ...	250,000
5 minutes ,, ,, ...	80,000 ...	200,000
15 ,, ,, ,, ...	3,000 ...	220,000
30 ,, ,, ,, ...	4,000 ...	160 000
60 ,, ,, ,, ...	600 ...	90,000

Thus we see that ether narcosis does not affect removal of the bacteria, and apparently interference with clotting affects the removal of the bacteria in the cat. In order to prove that peptone is not inhibitory to phagocytosis, two guinea-pigs were inoculated intraperitoneally with equal amounts of *Bacillus coli*, and one had 1 c.c. of 10 per cent. peptone as well; after five minutes both were killed and smears from the peritoneal cavity and omentum showed equal degree of phagocytosis in both.

Subsequently we were able to obtain leeches and prepare leech extract. The extract was very effectual; 1 c.c. completely prevented the clotting of 10 c.c. of rabbit's blood *in vitro* for twenty-four hours.

Experiment 33.—Three rabbits were taken of approximately the same weight: (1) normal, (2) normal, (3) immune, agglutinating titre against *Bacillus typhosus* 1 : 10,000. Into each was inoculated intravenously half a slope of *Bacillus typhosus*, and into (2) and (3) were inoculated 5 c.c. of above leech extract with the culture.

Number of Colonies developing from 1 cubic centimetre of Blood after—

	Rabbit 1	Rabbit 2 + leech extract	Rabbit 3 (immune) + leech extract
1 minute	400,000	400,000	500,000
5 minutes	25,000	370,000	500,000
15 ,,	1,500	200,000	420,000
30 ,,	500	50,000	120,000
60 ,,	500	20,000	75,000
4 hours	300	5.000	15,000

These results were not always, however, obtained. Sometimes the leech extract had no apparent effect in spite of the delay of coagulation, and the same was noted for peptone in cats (see below).

Experiment 34.—A cat was anæsthetized and given 10 c.c. of leech extract, and then half a slope of *Bacillus coli* intravenously. Samples taken as before.

Number of Colonies in 1 cubic centimetre of Blood.

1 minute after inoculation	120,000
5 minutes ,, ,,	16,000
15 ,, ,, ,,	3,000
30 ,, ,, ,,	3,000

Experiment 35 : Cat.—Anæsthetized; given 8 c.c. of 10 per cent. peptone intravenously. Then a half slope of *Bacillus coli.*

Colonies developing from 1 cubic centimetre of Blood.

1 minute after inoculation	200,000
5 minutes ,, ,,	80,000
15 ,, ,, ,,	3,000
30 ,, ,, ,,	4,000
60 ,, ,, ,,	6,000

Blood coagulated when withdrawn in two hours

Experiment 36.—Typhoid immune rabbit (*a*), 1,500 grm., was given 5 c.c. leech and half a slope of *Bacillus typhosus*, a normal rabbit (*b*), 1,850 grm., was given the same.

Colonies developing from 1 cubic centimetre of Blood.

		(*a*)		(*b*)
1 minute after inoculation	...	120,000	...	102,000
5 minutes ,, ,,	...	150,000	...	40,000
15 ,, ,, ,,	...	80,000	...	6,000
30 ,, ,, ,,	...	20,000	...	800
60 ,, ,, ,,	...	20,000	...	3,000

Experiments with Bacillus dysenteriæ (Shiga).

Experiment 37.—Two normal rabbits: (*a*) 1,920 grm., was given one third of a slope of *Bacillus dysenteriæ* (Shiga) intravenously in 5 c.c. normal saline; (*b*) 1,800 grm., was given one-third of a slope of *Bacillus dysenteriæ* (Shiga) intravenously in 5 c.c. of leech extract.

Number of Colonies developing per cubic centimetre of Blood taken.

		Without leech extract		With leech extract
1 minute after inoculation	...	180,000	...	210,000
5 minutes ,, ,,	...	30,000	...	52,000
15 ,, ,, ,,	...	16,000	...	40,000
30 ,, ,, ,,	...	2,400	...	5,000
60 ,, ,, ,,	...	400	...	600

These experiments show that although leech extract and peptone sometimes prevent the initial rapid disappearance of the bacteria from the circulation, they do not always do so. Generally, however, they cause delay, the disappearances not being so rapid as when the peptone and extract are not used. Further experiments showed that it is possible to kill guinea-pigs by intravascular clotting if quantities of 5 c.c. of a very thick bacterial emulsion (*Bacillus mycoides*) are used, but if the emulsion is made up in leech extract this does not occur.

Further evidence of the purely physical coagulative factor shown in these experiments *in vivo* on the blood is given by the observations made by us that when bacteria are mixed with leech whole blood or leech plasma more or less rapid coagulation of these takes place after the addition of the bacterial emulsions. Thus leech blood was obtained from a rabbit (2 c.c. of leech extract to 8 c.c. of rabbit's blood); this mixture did not coagulate when left alone at the room temperature for twenty-four hours. It was divided into two parts: one was centrifugalized at a high speed to sediment the corpuscles to obtain the plasma, the other was left untouched. Emulsions of bacteria, *Bacillus coli*, staphylococci, &c., were added to these and it was found that coagulation of the blood occurred in thirty to sixty minutes, according to the amount of the bacterial emulsion used; the plasma coagulated after several hours.

The conclusion to which we have been forced to come is that the preliminary removal and phagocytosis of bacteria in the circulation are not due either to agglutination or opsonization, but to the bacteria acting as foreign particles and producing small areas of clotting around them. These masses of bacteria and early clot accumulate in the periaxial stream of the large capillaries of the liver, lung, spleen, &c., and then phagocytosis occurs.

The previous section as well as this appear to give conclusive evidence that agglutinins take no part in the removal of bacteria injected into the circulation, since in the highly immune animals the rate of removal is the same as in the normal, and bacteria which are highly virulent and against which the animal has no agglutinin—e.g., Shiga's bacillus, and the chicken cholera bacillus, are removed in the same way, and as rapidly as others.

The relatively small proportion of leucocytes taking up bacteria and the dense packing of these with bacteria is just like what is seen in the spontaneous phagocytosis in the absence of opsonin *in vitro*.

(C) The question of removal of the bacteria from the peripheral blood in the stage of bacteriæmia following the temporary primary removal was next investigated. C. J. Bull [13] showed that anti-sera caused the disappearance of the organisms from the peripheral circulation at that stage, and believed that this occurred in virtue of the specific agglutinating properties of the antisera used.

(*a*) *Experiments showing that an Antiserum causes the Disappearance of Bacteria from the Circulation in the Secondary Bacteriæmia.*

Experiment 38.—A rabbit weighing 1,620 grm. was given half a slope of fairly virulent *Bacillus typhosus* intravenously; twenty-four hours later a culture was made from its blood and then 4 c.c. of rabbit typhoid agglutinating serum (1 : 10,000) was inoculated intravenously. Cultures made as before.

	Per cubic centimetre of blood
Before the inoculation of the serum there were	2,500 colonies
5 minutes after ,, .. ,, ,,	180 ,,
15 ,, ,, .. ,, ..	50 ,,
30 ,, ,, .. ,, ..	200 ,,
60 ,, ,, ,, .. ,,	3,500 ,,

Experiment 39.—Same experiment as above, only one slope of the same strain of *Bacillus typhosus* as Experiment 38, inoculated intravenously; twenty-four hours afterwards 4 c.c. of same agglutinating serum was inoculated intravenously.

						Per cubic centimetre of blood	
Before the inoculation of the serum there were						8,000	colonies
5	minutes after	,,	,,	,,	,,	1,200	,,
15	,,	,,	,,	,,	,,	200	,,
30	,,	,,	,,	,,	,,	160	,,
60	,,	,,	,,	,,	,,	2,400	,,
2	hours	,,	,,	,,	,,	6,000	,,

The animal died four hours after the inoculation of the serum. These experiments agree with those of Bull.

(*b*) *Inoculation of Normal Non-agglutinative Serum.*

Experiment 40.—Rabbit of 2,020 grm. was inoculated intravenously with half a slope of same *Bacillus typhosus* as in Experiment 38. Twenty-four hours later 4 c.c. of normal horse serum were inoculated intravenously.

						Per cubic centimetre of blood	
Before the inoculation of the serum there were						5,600	colonies
5	minutes after	,,	,,	,,	,,	1,000	,,
15	,,	,,	,,	,,	,,	400	,,
30	,,	,,	,,	,,	,,	500	,,
60	,,	,,	,,	,,	,,	2,000	,,

Animal died in two hours.

Experiment 41.—Rabbit of 1,800 grm. was given intravenously half a slope of *Bacillus coli*, twenty-six hours afterwards 4 c.c. normal horse serum.

Colonies per cubic centimetre of blood				before		injection of the serum,		6,000
,,	,,	,,	,,	5	minutes after	,,	,,	400
,,	,,	,,	,,	15	,,	,,	,,	*nil*
,,	,,	,,	,,	30	,,	,,	,,	200
,,	,,	,,	,,	60	,,	,,	,,	3,000

Animal died in 30 hours.

Experiment 42.—Same as above (Experiment 41). 4 c.c. normal horse serum inoculated intravenously twenty-four hours after the *Bacillus coli* had been injected.

Colonies per cubic centimetre of blood				before		inoculation of the serum,		5,000
,,	,,	,,	,,	5	minutes after	,,	,,	1,000
,,	,,	,,	,,	15	,,	,,	,,	*nil*
,,	,,	,,	,,	30	,,	,,	,,	*nil*
,,	,,	,,	,,	60	,,	,,	,,	400

(*c*) *Influence of Leech Extract on the Action of the Antisera, &c.*

Experiment 43.—Several rabbits were each given half a slope of *Bacillus typhosus* intravenously; three which had survived, nineteen hours afterwards, were given 2 c.c. typhoid agglutinating serum (rabbit) (1 : 50,000) and 5 c.c. leech extract.

Colonies developing from 1 cubic centimetre of Blood.

	Rabbit 1	Rabbit 2	Rabbit 3
Before inoculation of the serum ...	510	170	360
5 minutes after ,, ,, ...	400	130	350
15 ,, ,, ,, ...	290	170	330
30 ,, ,, ,, ...	460	110	770
60 ,, ,, ,, ...	1,760	300	390

(*d*) *Effect of Inoculating Bacteria in Cases in which the Secondary Bacteriæmia has occurred.*

Experiment 44.—Rabbit of 2,100 grm. was given half a slope of *Bacillus anthracis* intravenously, twenty-four hours after one third of a slope of *Bacillus coli* was inoculated intravenously. Plates were made with agar and rebipelagar to allow of differentiation.

	Bacillus anthracis Colonies per cubic centimetre of blood	*Bacillus coli*
Before inoculation of *Bacillus coli* ...	500	—
1 minute after inoculation	500	80,000
5 minutes ,, ,, ...	100	25,000
15 ,, ,, ,, ...	100	4,000
30 ,, ,, ,, ...	80	800
60 ,, ,, ,, ...	75	240

Experiment 45.—Similar experiment only with dead *Bacillus coli* for the second inoculation.

Control : Before inoculation	200	colonies	*Bacillus anthracis*	per cubic	centimetre
5 minutes after ,,	50	,,	,,	,,	,,
15 ,, ,,	20	,,	,,	,,	,,
30 ,, ,,	*nil*	,,	,,	,,	,,
60 ,, ,,	5	,,	,,	,,	,,
2 hours ,, ,,	5	,,	,,	,,	,,
4 ,, ,, ,,	300	,,	,,	,,	,,

Experiment 46.—A rabbit of 1,920 grm. was inoculated intravenously with one slope of *Bacillus coli*. Nineteen hours later one third of an agar slope of *Bacillus anthracis* (eighteen hours' culture) inoculated intravenously.

	Colonies *Bacillus coli*	Colonies *Bacillus anthracis*	
Before inoculation of *Bacillus anthracis*	63	—	per c.c.
1 minute after inoculation	27	140,000	,,
5 minutes ,, ,,	6	80,000	,,
15 ,, ,, ,,	0	8,000	,,
30 ,, ,, ,,	45	1,000	,,
60 ,, ,, ,,	6	250	,,

Experiment 47.—A rabbit was inoculated with a slope of *Bacillus coli* intravenously, same as last experiment; twenty-two hours later was inoculated with a quarter of a slope of *Bacillus anthracis* in 5 c.c. of leech extract.

	Colonies *Bacillus coli*	Colonies *Bacillus anthracis*
Before inoculation of *Bacillus anthracis*	120	--
1 minute after inoculation	140	160,000
5 minutes ,, ,,	100	160,000
15 ,, ,, ,,	120	120,000
30 ,, ,, ,,	80	100,000
60 ,, ,, ,,	160	80,000

Here again the leech extract prevents the rapid elimination from the peripheral circulation.

CONCLUSIONS.

These experiments bring out the following facts :—

(1) All forms of bacteria get rapidly removed from the circulating blood and accumulate in the capillaries of the liver, spleen, lung, &c.

(2) This removal occurs with pathogenic, virulent, or non-pathogenic bacteria.

(3) This removal is not due to the action of antibodies acting as agglutinins or opsonins. The process is purely a physical one and is due to the bacteria being foreign particles, causing the formation of incipient clot and entanglement of the bacteria.

(4) This aggregation and phagocytosis can be greatly delayed by anticoagulants.

(5) There is no difference in the amount of phagocytosis by the fixed tissue cells and leucocytes if the bacterium is pathogenic or non-pathogenic.

(6) Antisera do not cause removal of bacteria in secondary bacteriæmia in virtue of agglutinins, &c., but merely because they are foreign sera. The same result can be obtained by the inoculation of other bacteria. The free bacteria in bacteriæmia get entangled in the masses of clot formed and get phagocytosed with the other bacteria inoculated.

REFERENCES.

[1] TEALE and EMBLETON. *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Path.), p. 69
[2] WYSSOKOWITSCH. *Zeitschr. f. Hyg. u. Infect. Krank.*, 1886, i, p. 3.
[3] WERIGO. *Ann. de l'Inst. Pasteur*, 1892, vi, pp. 478-511.
[4] BULL. *Journ. of Exper. Med.*, 1915, xxii. pp. 475, 484.
[5] METCHNIKOFF. "Immunity in Infectious Diseases," 1903, p. 263.
[6] SALIMBENI. *Ann. de l'Inst. Pasteur*, 1897, xi, p. 277.
[7] SAWTSCHENKO and MELKICK. *Ann. de l'Inst. Pasteur*, 1901. xv, p. 497.
[8] TEALE and EMBLETON. "Pathogenicity and Virulence of Bacteria," *Lancet*, 1913, i, p. 234.
[9] BULL. *Journ. of Exper. Med.*, 1915, xxii, p. 488.
[10] CHICK, Miss H. *Lancet*, 1916, i, p. 857.
[11] TEN BROECK. *Journ. of Exper. Med.*, 1917, xxvi, p. 441.
[12] KYES, PRESTON. *Journ. Infect. Diseases*, 1916, xviii, pp. 277-292.
[13] BULL. *Journ. of Exper. Med.*, 1915, xxii, p. 457.
[14] HOPKINS and PARKER. *Journ. of Exper. Med.*, 1918, xxvii, pp. 1-26.

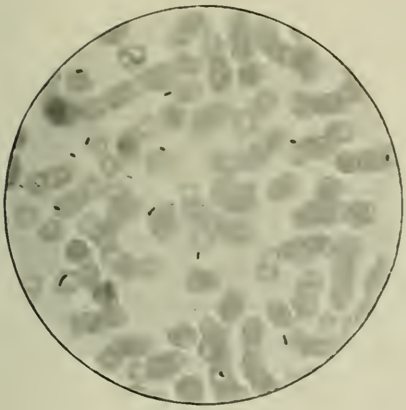

FIG. 1.

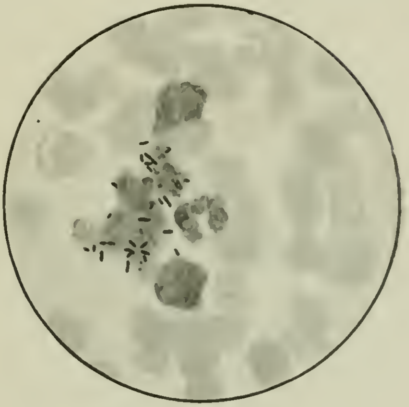

FIG. 2.

Fig. 1.—Film from the liver of a guinea-pig inoculated thirty seconds previously with an emulsion of *Bacillus dysenteriæ* (Shiga). Note the uniform distribution of the bacteria.

Fig. 2.—Film made from the liver of a guinea-pig inoculated with the same emulsion as fig. 1 three minutes previously. Note the bacteria in a mass of blood platelets and the leucocyte in the immediate neighbourhood.

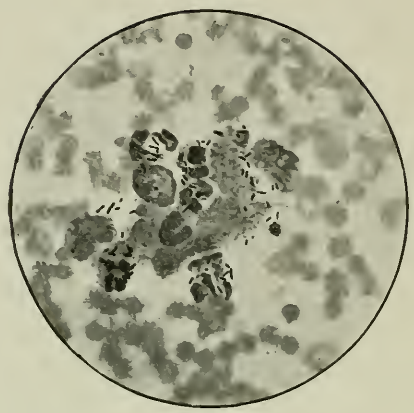

FIG. 3.

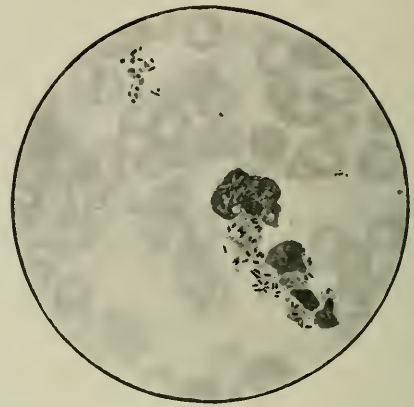

FIG. 4.

Fig. 3.—Similar experiment to fig. 2. *Bacillus dysenteriæ* (Flexner) inoculated three minutes previously. Note here again the bacteria in the mass of platelets, &c., and the phagocytosis by the leucocytes.

Fig. 4.—Similar experiment using an emulsion of *Bacillus enteritidis* of Gaertner; three minutes after the inoculation.

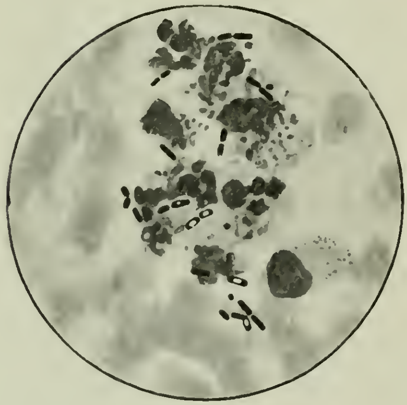

FIG. 5.

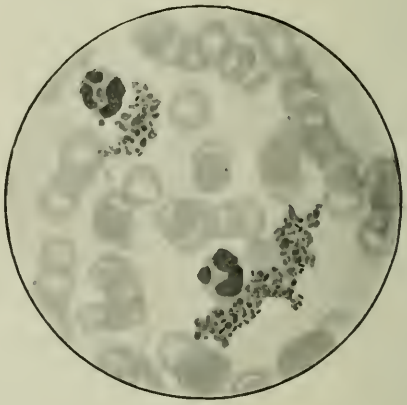

FIG. 6.

Fig. 5.—Experiment with an emulsion of *Bacillus mycoides* three minutes after inoculation. Film from the liver.

Fig. 6.—Similar experiment with *Bacillus coli;* film from liver of the guinea-pig three minutes after inoculation.

Note bacteria in masses of blood platelets

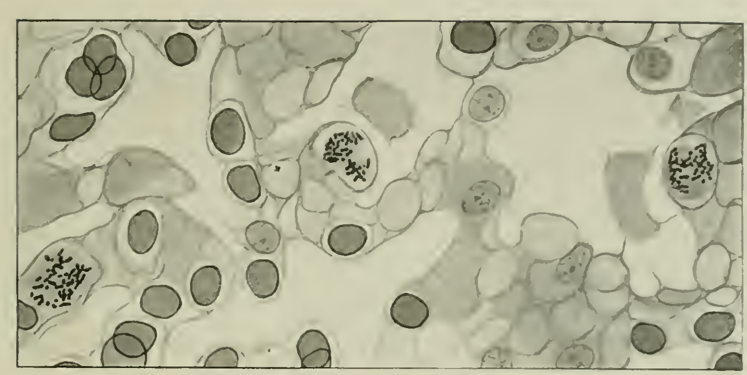

FIG. 7.

Section of the lung of a guinea-pig inoculated fifteen minutes previously with an emulsion of *Bacillus Hoffmann*. Note numerous bacteria in swollen endothelial cells.

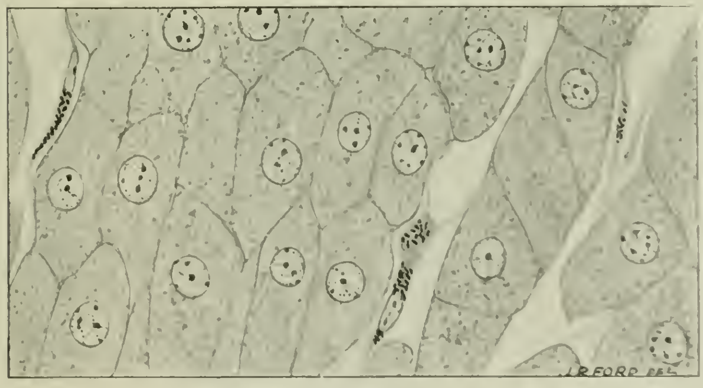

FIG. 8.

Section of the liver of a guinea-pig inoculated fifteen minutes previously with an emulsion of *Bacillus Hoffmann*. Note bacteria in Kupffer's cells.

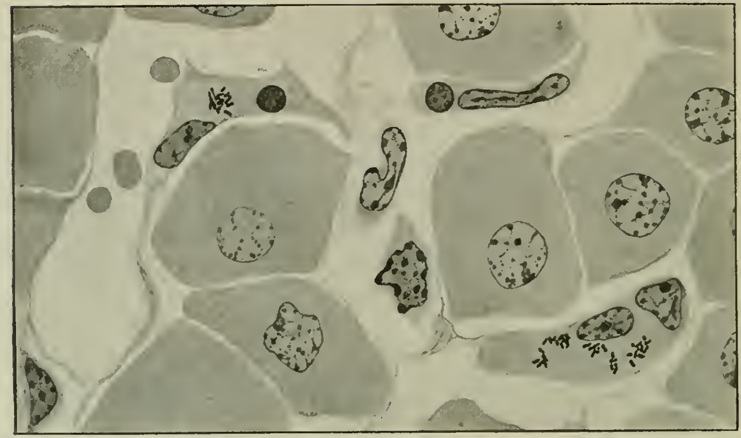

FIG. 9.

Section of the liver of a guinea-pig inoculated, sixty minutes previously, with an emulsion of *Bacillus Hoffmann*.

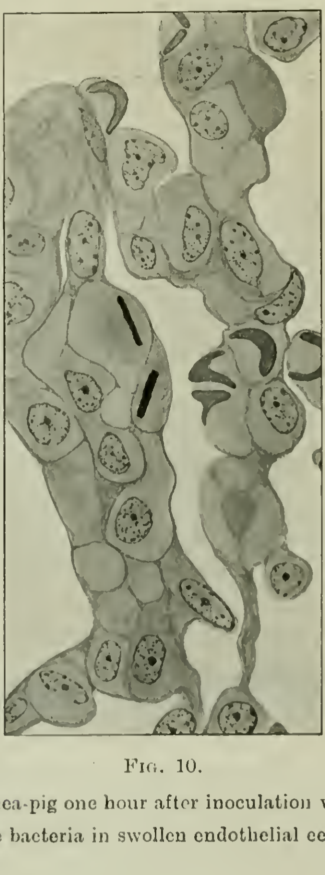

FIG. 10.

Section of lung of guinea-pig one hour after inoculation with *Bacillus mycoides*. Note bacteria in swollen endothelial cells.

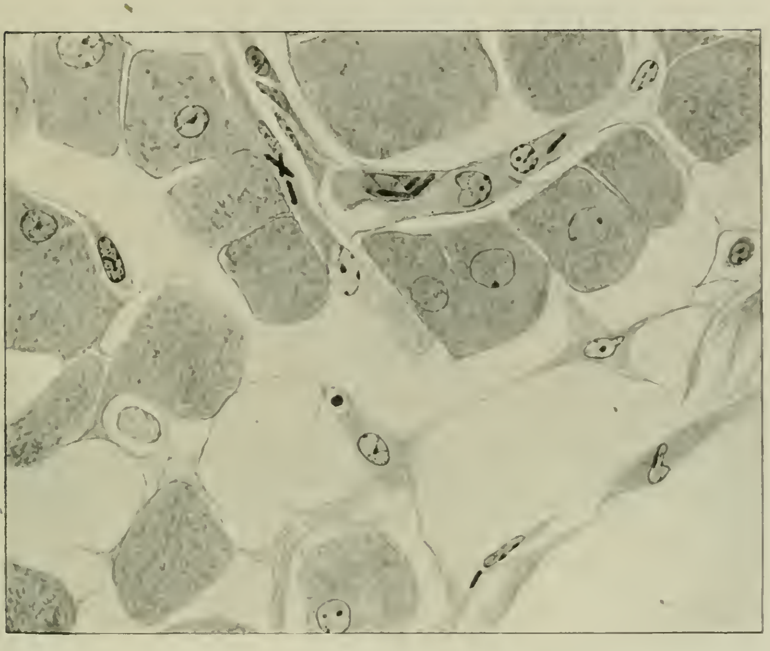

Fig. 11.

Section of the liver of a guinea-pig inoculated, fifteen minutes previously, with an emulsion of a very virulent culture of *Bacillus mycoides*. Note bacteria in Kupffer's cells.

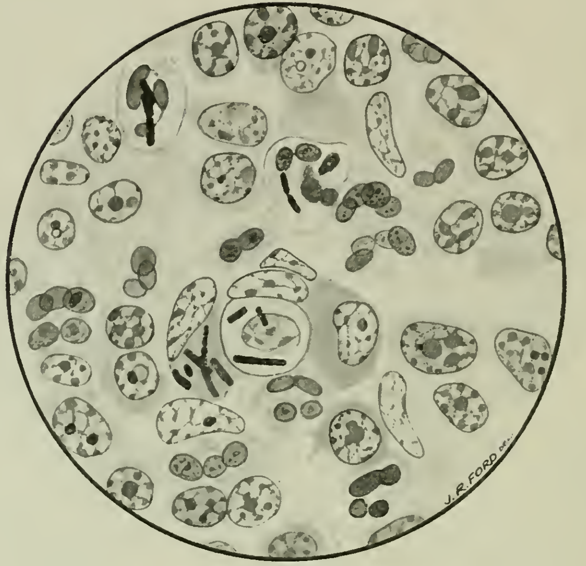

Fig. 12.

Section of the spleen of a guinea-pig inoculated, fifteen minutes previously with an emulsion of a very virulent culture of *Bacillus mycoides*. Periphery of Malpighian corpuscle. Note bacteria in polymorphonuclear cells and in the splenic cells.

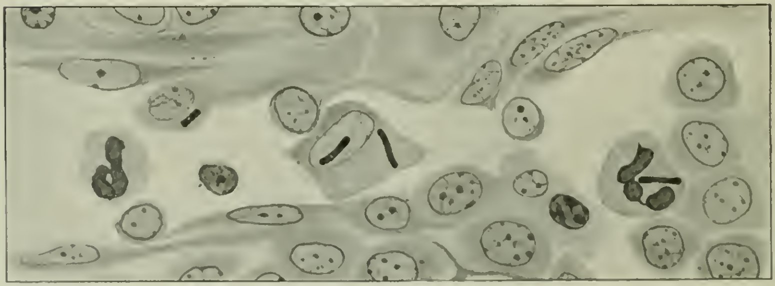

Fig. 13.

Section of the spleen of a guinea-pig inoculated sixty minutes previously with an emulsion of a very virulent culture of *Bacillus mycoides*. Showing a sinus. Note bacteria in polymorphonuclear cells and in endothelial cells.

Section of Pathology.

President—Dr. W. Bulloch, F.R.S.

The Traumatic Causation of Appendicitis: and Appendicular Concretions.[1]

By S. G. Shattock, F.R.S.

(Continued from Vol. IX.)

Contra originem appendicitis traumaticam.

(Sequentia tractus in tomâ IX, 1916.)

De concretionibus appendicularibus.

Summarium.

Exemplis paucissimis exclusis, ut determinatur concretionibus sub aquâ delaminandis, nuclei absunt; e.g., fructuum semina, plantarum aut animalium pili, corpora metallica, et cetera.

Scrutatio demonstrat microscopica has concretiones (quinque vigintis examinatis) ex herbarum residuis disintegratis solum constare, cum calcii carbonate et phosphate commixtis, bacteriis naturaliter quoque inclusis.

Cellulosae reactionis nihil dant residua.

Concretionem nullam inveni de avenae pilis constructam ut illae quae in colo repertae sunt, etsi pili plurimi ab avenae aut tritici pericarpo aufracti nonnunquam adsunt, quae structurae quum e cutino constent non destruuntur in colo sicut cellulosa et ligninum, bacteriorum actione.

Fibrae musculares striatae, amyli quoque granulae raro, conidia hyphomycetium saepius reperiuntur, atque animalium pilorum fragmenta.

---

[1] At a meeting of the Section, held April 20, 1920.

In exemplo uno solum adfuerunt particulae metallicae quarum scrutatio chemica demonstravit alias esse ferri alias stanni.

Non probatur ergo hypothesis illa quae concretionum harum originem ascribit in appendicem introitui particularum metallicarum ab cylindris illis ferreis abrasarum qui nunc in tritico terendo usui sunt.

Ferri inorganici tamen scrutatio chemica (ammonii sulphocyanido) vestigium minutissimum demonstrat in farinâ quae hoc modo praeparata est.

Notari autem debet vestigium hoc ita repertum non illud excedere quod aut in tritico ipso adest intacto, aut in farinâ inter molas tritâ.

De ferro inorganico quod e tritico intacto extrahi potest nulla dubitatio est quin agrorum pulveri ascribi debeat origo. In hoc pulvere (2 mg.) ferrum facile demonstravi.

Inter se vix delimitantur appendicis scybala et concretiones laminatae. In mediâ concretione laminatâ quidem, nonnunquam adest nucleus scybalis minutis aggregatis constructus.

Concretionum appendicularium picturae skiagraphicae sic inter se differunt ut quantitas calcii carbonatis et phosphatis in eis deposita. Zonulae aut puncta opaca in picturis skiagraphicis discerni possunt quae partes calcificatas repraesentant.

Sales ipsi inorganici calcii carbonas et phosphas sunt; cum his nonnunquam commiscetur ammonio-magnesii phosphas. Nucleis absentibus concretionum origo vitio cuidam appendicis neuromusculari ascribi solum potest.

The first part of this communication (vol. ix, 1916) closed with an enumeration of the various foreign bodies which had been found in the appendix, either as nuclei of concretions, or in a free condition. And before proceeding to concretions without nuclei, I may notice a specimen in which the appendicular contents are almost, though not absolutely, unique in their character. The appendix was excised by Lieutenant-Colonel C. G. Shaw, from a man, aged 23, in October, 1918. He had been seized three days previously with pain in the lower part of the abdomen on the right side, and had vomited the following morning. On admission there was well-defined tenderness over McBurney's point; and some rigidity over the right iliac fossa: temperature, 99° F. Previous attacks of appendicitis had occurred in 1916 and 1917. For three years he had felt constant dragging pain in the right iliac region. Four or five years ago he had on several occasions passed round worms, 6 or 7 in. long, and had suffered from worms as a child. The appendix

was removed on October 22, 1918. Recovery was uneventful. The appendix, which is uniformly though not greatly enlarged, was found on longitudinal section tightly packed with semitransparent spherical bodies like fish roe: some of these were observed to escape when it was cut through in its removal. There are membranous patches of inflammatory adhesion on the peritoneum.

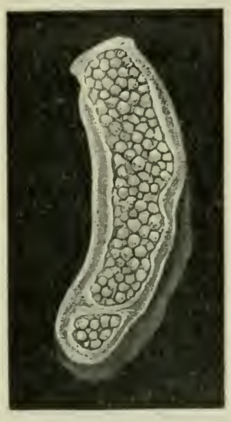

FIG. 1.

A vermiform appendix longitudinally bisected. It is uniformly filled with spherical bodies like fish roe, which consist of pellets of mucin containing shed epithelium. (Natural size.)

Figurae explicatio.

Appendix vermiformis in longitudinem bisecta. Sphaerulis distenditur compacte aggregatis sicut piscium ova quae e mucino cum epithelii cellulis constant. (Magnitudinis naturalis.)

One is tempted, at first, to connect the bodies with the ascarides, and to imagine that the female worm had discharged its ova directly through the mouth of the appendix. But not only is the ovum of the round worm almost microscopic, but the bodies in question have no enveloping striated membrane, and their nature admits of being otherwise explained. When crushed, and stained with iodine, they exhibit groups of oval nuclei distributed in a soft homogeneous matrix; the use of scharlach demonstrates the presence of fat droplets in the cytoplasm of some of the cells. A more detailed study carried out by means of serial sections including the wall of the appendix and the adjacent contents yielded the following results: The lining epithelium is proliferating; between the cells there are irregular spaces containing mucin:

here and there a multinucleated giant cell lies immediately beneath the epithelium. The tissue between the epithelium and the proper muscular coat is fibrotic and contains groups of lymphocytes; the glandular crypts are quite wanting. On its inner aspect the epithelium is succeeded by mucin in which are included desquamated and necrotic epithelial cells, polymorphous and elongated: this is followed by the spheres, the deepest of which are confluent, the rest discrete. The spheres, like the mucin elsewhere, enclose desquamated epithelium.

The morbid histology is thus one of catarrhal inflammation accompanied with hypersecretion of mucin, and desquamation of the proliferating epithelium, the mucin affecting the form of spherules as though extruded from and accompanied with the destruction of the crypts. The only things with which they bear comparing are the spherules of fat now and then found in teratomatous cysts of the ovary and in dermoid cysts elsewhere. These consist of the sebum discharged from the cutaneous glands into the cavity. In the Museum of St. Thomas's Hospital there are the following examples:—

No. 2367.—A collection of spherical masses of sebaceous substance and hairs, of about the size of marbles, from a teratomatous (so-called dermoid) cyst of the ovary which held 7 pints of similar bodies.

No. 2557.—A large dermoid cyst which was removed from the top of the head. It contained fluid in which were a large number of spherical bodies about the size of small marbles which were found to consist almost entirely of fat. (Cooper and Travers's "Surgical Essays," Part 2, p. 214.)

But, so far as size goes, the roe-like bodies are best matched by the contents of the following dermoid cyst in the Museum of the Royal College of Surgeons:—

No. 256 H.—A portion of the contents of a submaxillary dermoid cyst removed by operation. The material consists of epithelial scales and fatty matter, the latter having assumed, for the most part, the form of minute globules, the largest of which are about the size of pins' heads. They are readily soluble in chloroform. From a man, aged 45, who was admitted with a soft fluctuating swelling in the right submaxillary region, which extended from the angle of the jaw to the chin. The swelling was first noticed ten years previously. At the operation it was found that the cyst was situated upon the middle constrictor of the pharynx and was partly covered by the hyoglossus muscle. The patient made a good recovery.

To this may be added an example of a hair-concretion in the appendix of a chimpanzee lately received at the College of Surgeons from the Zoological Society through Dr. J. A. Murray. The middle of the appendix is filled with a cylindrical, slightly curved, closely matted collection of coarse black hairs. The concretion is 4 cm. in length. Microscopically the hairs correspond with those from the skin of the animal, which was 17 years old. The upper and lower jaws with the frontal region of the calvaria were much thickened from some form of spreading osteitis arising apparently in connexion with the teeth. This is the nearest approach to a "hair-ball" of the appendix that has yet been met with, and it may be classed with those so well known and not uncommon in the human stomach.

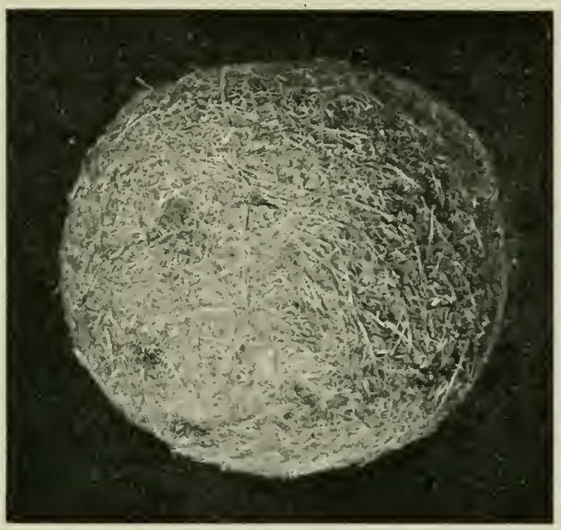

FIG. 2.

A concretion consisting of small fish bones (ribs) ejected from the stomach or intestine of some cetacean. (Natural size.)

Figurae explicatio.

Concretio e ventriculo aut intestino animalis cetacei cujusdam ejecta. Ex ossibus tenuibus, piscium costis, constat. (Magnitudinis naturalis.)

In the first part of this communication, lastly, reference was made in a footnote (p. 87) to hair-balls of the human stomach, and similarly felted concretions of cocoa-nut fibre, and other materials. The list may be here completed by adding to these the concretions of small fish-bones ejected from the stomach or intestinal canal of cetacea. These curious objects are known from a group now in the College of Surgeons, which came from the Collection of Sir Hans Sloane—the nucleus of our present British Museum. The group comprises five examples, of which the largest is barrel-shaped, and measures 2½ by 2 in. The others are spherical and range from 1 to 2 in. in diameter. They all consist of fine elongated fish-bones (ribs and fins) closely felted as in the case of analogous concretions of vegetable or animal hairs. In the box in which they originally were there were two labels. On one label was written "Sloanian Catalogue, Pisces No. 613: A very large ball from the sea, cast up in Cornwall, after whales had been on the coast pursuing spratts, whose bones it is thought to be. They were thrown up one tide and left the next. This was given me by Lady Coply (*see* No. 787)." On the other label is "Sloanian Catalogue, Pisces No. 1075: A round ball of redish small bones thrown on shore on the coast of Cornwall—*vide antea* (No. 613), Dr. Woodward." Amongst the bones of the last there are some small vertebræ.

APPENDICULAR CONCRETIONS WITHOUT NUCLEI.

Their Microscopic Structure.

The cases in which appendicular concretions are formed upon nuclei are exceptional. As a rule there is nothing discoverable centrally which is differentiated from the rest of the concretion. The method adopted for this study has already been referred to (*Proceedings*, vol. ix, p. 85). The result of a minute examination of the concretions from twenty-five cases demonstrates that their composition is remarkably uniform: they consist almost exclusively of the residues of disintegrating vegetable tissue. And this is true not only of such as have no nuclei but equally so of those which have. Nor has the amount of calcareous deposition any relationship whatever with the presence or absence of a nucleus. To describe these twenty-five specimens in detail would be superfluous, and I may, therefore, select one which is typical of the series, and afterwards notice what is to be gleaned from the study of the rest. For the examination of the vegetable tissue alone the best preparations are made by freely treating a sample of the material on a slide with 25 per cent HCl, which clears it of any calcareous deposition and renders it more translucent; washing it with distilled water, staining with iodine solution, and finally mounting it in glycerine, or in Farrant's medium. As a type, then, the following may be taken :—

Specimen No. 11.—An ovoidal concretion of pale buff colour, 1·2 cm. in chief diameter, taken from an excised appendix which was gangrenous and perforated. Embedded in its surface there were a few reddish-brown or darker particles, which proved on microscopic examination to be fragments of deeply coloured vegetable tissue. These objects are not uncommon in the surface and deeper substance of such concretions. On being gently scraped and delaminated under water the concretion was found to be pale in colour and of soft consistence, somewhat like pipe-clay, and it was finely laminar or flaky. Towards the centre it became firmer, and of a deeper brown colour, the lamination becoming at the same time coarser. When reduced to the size of a hemp seed the central remnant was transferred *in toto* to a slide and washed in glycerine for microscopic study with the object of ascertaining whether anything of the nature of a nucleus was present. Microscopic examination of portions of the flakes detached from the main mass at different depths, lightly broken on slides and studied in Farrant's medium : The material is composed almost solely of the residues of vegetable tissue—cell walls and fibro-vascular structures in various stages of digestive disintegration. That bacteria are included goes without saying, but the bacteriology of appendicitis is a subject beyond the purpose of the present communication. The bacteria are recognizable in unstained perforations, but they can be readily dyed with carbol thionine in dried films prepared after the usual method; many, as might be anticipated, stain imperfectly like dead or degenerate forms.

Microscopically, the plant residues in this particular concretion are resolvable into two chief forms : (1) flattened, sometimes slightly curved, coherent groups of small elongated elements, and (2) a finer dèbris the source of which is traceable to the further disintegration of the latter. Certain cylindrical fragments represent the less disintegrated remnants of tracheides or of elongated cells. The organic nature of these different elements is proved by their undergoing no solution or other changes when freely treated, on the uncovered slide, with 25 per cent. HCl, or with the undilute acid. No starch is demonstrable by the iodine test, and when the iodine-stained material is tested with 75 per cent. H_2SO_4, it gives no blue colour, showing that the cellulose constituent has been removed (by bacterial digestion) and that the residues represent the disintegrating secondary thickenings of cell walls, woody fibre, and vessels. In addition to this, the main material, there are a certain number of thick-walled pointed vegetable hairs, with narrow lumina, and oblique slightly expanded bases traversed by short canals, characters which identify them with the hairs from the pericarp of wheat. The last-named structures consisting as they do of cutine and not of lignine, are insusceptible of the disintegration which even the latter undergoes. Furthermore, there are a few thick-walled sclerenchymatous cells of reddish-brown colour, with vacuolated interior, entire or in fragments; spirals freed mechanically, or after disintegration of their containing vessels; and lamellæ of flat elongated epidermal cells. In one of the preparations there is a fragment of striated muscle fibre, the occurrence of which in concretions is uncommon. The calcareous depositions in preparations of lamellæ untreated with HCl appear as translucent colourless or pale brown (stercobilin-stained) plaques, often irregularly fissured by the technique, the resulting elements being then of the most diverse form and size, like a very badly constructed mosaic.

The Central Portion of the Concretion.—The microscopic study of this, made when the concretion had been reduced by delamination to the size of a hemp seed, showed it to consist of disintegrating plant tissue like the rest, although more deeply stained. It contained in addition, a group of intact, colourless, sclerenchymatous cells, like those familiar in the fruit of the pear, together with a certain number of the thick-walled hairs from the pericarp of the wheat or oat, or of both. None of the central objects are macroscopic, and they are too minute and not sufficiently numerous to be viewed as constituting a proper, i.e., a mechanically acting nucleus. And this is equally true of the whole of the twenty-five specimens grouped under the heading of "Concretions without Nuclei."

Summary of the Microscopic Examination of Twenty-five Appendicular Concretions without Nuclei.

This will be most simply made by enumerating the different objects met with and adding remarks upon each as may be necessary. Of *macroscopic* objects the only ones that require mention are the yellow, brown, or blackish particles which are not infrequently embedded in the surface, and sparsely distributed through the substance of the concretions. These, on examination, prove to be, sometimes, minute fruit seeds or their fragments, but usually they consist of groups of plant cells with deep brown contents; some are fragments of bran, as told by the form and arrangement of the cells and their pale brown homogeneous contents. In only one instance did any of such dark particles prove to be metallic. The scattered opaque white points now and then met with are undigested fat, and consist microscopically of spherular groups of acicular crystals, resistent to HCl, and colourable with scharlach.

Wood Parenchyma, Tracheides.—Although a certain number of unaltered plant cells and fibro-vascular structures are to be met with, their proportion to the disintegrating tissue is small. As already stated, the whole of the material composing concretions consists practically of disintegrating plant cells and vessels, together with wood prosenchyma and bast fibre. The prosenchyma is recognizable in the earlier stages of its disintegration by the fineness and length of the processes into which the fibres become resolved. Excluding this and bast fibre, the material takes the form of flattened, it may be slightly curved, coherent groups of bright, elongated particles, more or less parallel, and sometimes oval or bacilloid in form. Their origin can be traced to the walls of cells, by the disintegration of which they arise. Their organic nature

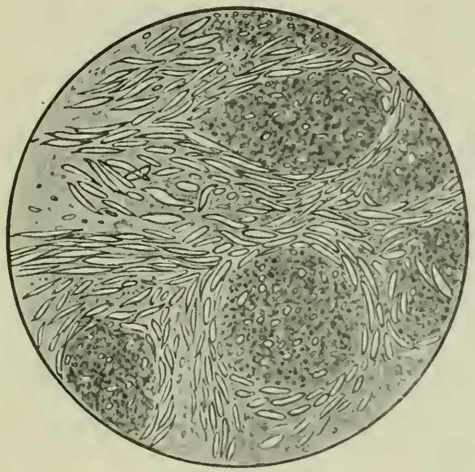

Fig. 3.

Showing walls of vessels disintegrating into bacilloid elements; from an appendicular concretion. ($\frac{1}{6}$ obj.)

Figurae explicatio.

Monstrantur vasorum plantarum parietes disintegrantes; ex appendicis concretione. ($\frac{1}{6}$ obj.)

is proved by the fact that they are unaffected by 25 per cent. or by pure HCl. They stain with iodine solution, but give no cellulose reaction on the subsequent use of 75 per cent. H_2SO_4. These forms, in the next stage, break up into finer and shorter particles, the ultimate products being so amorphous that nothing remains to indicate their origin.

Wood Prosenchyma.—This is highly characteristic in concretions, although it varies much in amount in different cases. The long, thick-walled wood fibre breaks up longitudinally into a tress of extremely fine fibrils. At times a certain amount of crossing of the fibrils is observable, corresponding with the normal disposition of the cell-thickening. The fibrils, when displaced, produce a tangled mass or felt work, at first suggestive of crystalline aggregations. In their further disintegration they break up into shorter lengths and bacilloid forms, which would eventually give no clue to their source, were it not that the stages of the transformation admit of being traced. The final product of the tapering wood fibres and of vessels is similar to that of cells.

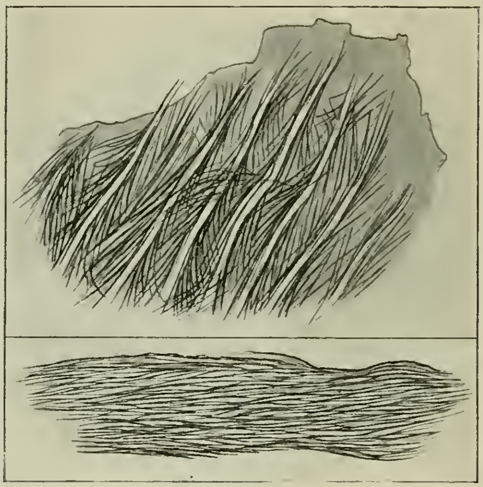

Fig. 4.

Showing the manner in which wood prosenchyma breaks up into tresses of fine fibrils; these ultimately disintegrate into molecular débris. ($\frac{1}{6}$ obj.)

Figurae explicatio.

Monstratur modus quo prosenchymatis fibrae disintegrantur; ex appendices concretione. ($\frac{1}{6}$ obj.)

Bast Fibre.—This is but seldom met with, doubtless because it does not commonly occur in any amount in the fibrovascular bundles of the vegetables or fruits which are commonly used as food.

Sclerenchyma. — Groups of sclerenchymatous cells, of the kind familiar in the mesocarp of the pear, are at times met with in an intact condition. More frequently the sclerenchymatous cells are from some other source, and are of reddish brown colour with a vacuolated interior, and occur either entire or in fragments.

Vegetable Hairs.—A certain number of thick-walled, pointed hairs, with oblique expanded bases, are almost invariably met with in appendicular concretions. These are derived from the pericarp of the oat or wheat, the base of the latter being furnished with short canals. As stated in the previous part of this communication such hairs from the oat at times form intestinal concretions, large and numerous enough to obstruct the colon. But I have not examined any appendicular concretion which could be said to be constructed, even centrally, of these hairs, although the number of them is now and then striking. Some of the hairs are intact, others fractured, as they may be found in any sample of flour or of oatmeal.

Conidia.—The conidia of moulds are not infrequent in appendicular concretions.

Starch.—This is rare. The undigested grains are readily discovered after treatment of a sample with iodine solution.

Cellulose.—When the usual débris is freely treated in the open with iodine solution and afterwards with 75 per cent. H_2SO_4, no blue reaction is obtained, showing that no cellulose persists, and that the disintegrating residues represent the secondary thickenings of cells, fibre, or vessels. Only rarely are small groups of parenchyma cells found which give a clear blue or violet colour, a proof that they have escaped bacterial digestion. Similar groups of colourable undigested cells may be found in the fæces of normal persons.

Animal Hairs.—Fragments of human and other hairs are not uncommon, either embedded in the surface or distributed through the substance of the concretion.

Muscle Fibre.—In two instances only have I met with striped muscle fibre. This rarity is, of course, explained by the fact that normally the whole of the protein should be digested before the intestinal contents are allowed a passage through the ileocæcal valve; and secondly, that no proteolytic ferments are present in the contents of the colon. The absence of striated muscle fibre, however, is by no means absolute in the fæces of normal persons.

Bone. – In one case I came across a minute fragment of bone. On the addition of HCl, CO_2 was disengaged, showing that it had escaped even the preliminary decalcification which precedes its digestion.

Coal Dust.—In each of two concretions there was encountered a small black flake, unaffected by HCl, with translucent sharply fractured edges, and corresponding in its microscopic characters with coal dust.

Silica.—In one, a pale brown concretion, the size of a pea, towards, but not actually constituting, the centre, there was a minute gritty particle which proved to be silica; with this there were two minute seeds.

Saucepan Enamel.—In no instance were any flakes of this substance (silicate) discovered.

Metallic Particles.—In only one concretion have I found any particles of metal. This was from the gangrenous appendix of a boy, aged 14, and it may be detailed, since it was especially in the search for such particles, that the examinations were made. The presence of the metal may be in some way connected with the occupation of the patient, who was a chemist's errand boy, and had not worked in any other capacity. The concretion was pale brown, oval, 1 cm. in chief diameter. On delamination under water two fine hairs appeared at one pole; a fair number of minute black or brown particles were embedded in its substance, and were encountered throughout it; these proved, on microscopic examination, to consist of disintegrating plant parenchyma, but a few showed microscopically, when examined by direct light, the bright points indicative of their being metallic. Two of these such particles were heated in HCl, an abundant evolution of H taking place; the fluid gave the usual iron reactions. A third particle, about the size of a small pin's head, attracted attention from its spherical form. On being boiled in a watch glass in HCl, H was evolved, the particle retaining its bright metallic lustre. A complete chemical examination proved that it was tin: it was dissolved by heat in pure HCl, and the fluid evaporated to dryness; the residue was dissolved in distilled water; the solution, when treated with ammonium molybdate gave the blue colour characteristic of tin. The microscopic examination of a flake from the peripheral part of the concretion showed the usual disintegrating vegetable material and a few unaltered wheat hairs. Not far below the surface there was a brown hair, curved in conformity with the concretion, and close to it a shorter black fragment of hair, the markings of which showed it to be from one of the lower animals. Three more hairs were encountered as the delamination progressed, lying in the longer axis of the concretion between its laminæ; and when nearing the centre there appeared three short hairs which projected from one pole. The fact that one of the metallic particles consisted of tin negatives the hypothesis that their source was roller milled flour; nor were the particles aggregated centrally so as to furnish a nucleus.

The "Centres" of Concretions without Nuclei.—In regard to these different concretions, as stated in the prefatory remarks, they contain no nuclei—i.e., nothing differentiated from the rest of the concretion, either to the naked eye or microscopically. A "centre" was arbitrarily made by prolonging the process of delamination until a portion about the size of a hemp-seed remained; this was then crushed in glycerine between a slide and cover glass and made into a microscopic preparation for study. Such "centres" consist of disintegrating vegetable tissue of the usual kind, including also free spirals, films of plant-epidermis, groups of sclerenchymatous cells and thick-walled vegetable hairs. In concretion No. 2, it contained one minute particle of silica, and one or two undigested fibres of striated muscle. In No. 6 there were amongst the vegetable débris two short pieces of human hair. In No. 7 the "centre" contained a few pieces of black hairs with cross markings, which latter exclude bacon rind as their source. The number of oat or wheat hairs, or of both, was conspicuous; this was likewise the case in Nos. 10 and 14.

From the foregoing details it will appear that the microscopic elements composing appendicular concretions are identical with those disclosed by an examination of fæcal material, which contains not only the finer débris of disintegrated plant structures, but intact fruit seeds of small size, flakes of pericarp (oat or wheat), of epidermis, or structureless folded films of cuticle detached from the last; and, it may be, striated muscle fibre, and groups of vegetable cells which still give a normal cellulose reaction; this reaction, it may be observed, is as well pronounced in boiled as in fresh tissue. In no instance were either metallic particles or flakes of the white enamel from the interior of cooking utensils encountered in the centre of the concretions.[1] The supposition that the ingestion and accumulation of such foreign material might serve as the starting point of a fæcal deposition may be thus taken as unfounded.

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[1] With respect to this enamel, which was referred to and figured in the first part of this communication (ix, p. 25), Mr. C. S. Tomes, F.R.S., has pointed out to me that its whiteness is not due solely to the inclusion of air (as is that of the silica dishes and flasks used in the chemical laboratory), but that it is increased by the addition of stannic oxide: the finely divided insoluble material is by no means uniformly distributed, as studied microscopically.

Appendicular Scybala.—In the contents of a fair proportion of the 125 normal appendices examined there were small pellets of fæcal material lying in the shed epithelium occupying the lumen. The pellets are at times so firm in consistence that they may be viewed as scybala, for they resist being broken down in water under the brush.

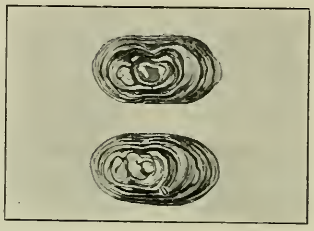

FIG. 5.

A typical appendicular stercolith, dried and bisected. The upper figure is that of a section carried almost through the centre, the surface being afterwards rubbed on a dry hone. The whiter incomplete concentric zones represent calcareous depositions about the vegetable débris which composes the concretion, and which is elsewhere of a deep brown colour and of less opacity. The lower shows the concretion at its exact centre, which consists of a small group of minute scybala. The concretion was removed by operation from the abdominal cavity of a youth, aged 17; the peritoneum contained fæculent pus. (Specimen No. 1102, St. Thomas's Hospital Museum.) (Natural size.)

Figurae explicatio.

Stercolithi typici sectiones duae. Monstrantur concretionis laminatio et zonulae albicantes incompletae, e calcii salibus constantes. Centrum sive nucleus ejus scybalis minutis aggregatis repraesentatur. E cavitate abdominali extracta est, appendice perforatâ. (Magnitudinis naturalis.)

Although their small size would render them *per se* negligible as factors of disease, yet they may serve as the starting points of laminar depositions or proper concretions. In their essential nature scybala and concretions are closely akin, the microscopic elements composing them are the same; the only distinction that can be drawn is, that in the concretion the accumulation is laminar. In appendices removed for disease it is rarely that proper scybala are encountered. As an example of scybala in a normal appendix, I may select the following (No. 23) from the group of twenty-five appendices taken from patients dying between the ages of 15 and 25 years inclusive. Male, aged 20. Death from chronic nephritis and uræmia. The appendix was examined in the recent condition without having been placed in any fixative. Its distal half was filled with a cylindrical process of soft, white friable material (shed epithelium) in which there were embedded pale yellow fæcal pellets: the proximal half contained a scanty amount of shed epithelium only. When the contents were washed out and irrigated, six oval, yellowish, firm pellets were isolated; five, the size of canary seed, and the sixth double this. The smaller were examined, each by crushing it on a slide in glycerine, and completing the preparation with a cover-glass. They consisted solely of disintegrating plant residua and were devoid of lamination and of any differentiated nucleus. The largest presented an imperfect lamination towards the centre; microscopically it resembled the others. The use of strong HCl on the uncovered slide liberated no CO_2.

In the same group occurs: No. 11; female, aged 18. Died September 30, 1915; mitral obstruction. A somewhat long appendix, filled with a narrow cylinder of soft pale brown fæcal material more voluminous in the distal 3 cm. In the washings there were a few small scybala—i.e., fæcal pellets, which resisted disintegration beneath the pressure of the brush. The largest, the size of a hemp seed, was crushed and rubbed down in glycerine to make a microscopic preparation. It consisted of groups of sclerenchymatous cells, vegetable hairs, free spirals, and disintegrating plant parenchyma: there was no differentiated nucleus.

A third example is contained in the same series: No. 7; female, aged 22. Died June 16, 1915: Diabetes mellitus. Contents: The proximal third contained a scanty white friable material (mucus and epithelium); the distal two-thirds, a friable cylinder of brown fæcal material coated with a small amount of the other. In washing the contents a certain number of small, dark brown spheroidal masses, the largest the size of a canary seed were isolated; these did not break down under the brush like the rest of the fæcal material. Their microscopic examination proved them to be scybala consisting of the usual disintegrating plant tissues, unaffected by HCl, and devoid of any differentiated centres.

The Skiagraphy of Appendicular Concretions.

The skiagraphic picture given by stercoliths varies in depth according to the amount of calcium salts with which the fæcal basis is infiltrated. The salts, as is well known, consist of calcium carbonate and calcium phosphate; ammonio-magnesium phosphate may be associated with these, and will aid in the production of the resulting shadow. The amount of calcification varies much. It is usually palpable by the greater firmness of the laminæ so involved; its distribution is equally erratic, for it may be pronounced in some zones and absent in others; and as shown in the divided surface after drying, it may be incomplete even in the same zone—i.e., may not involve its whole circuit. The shadow given by concretions in which there is but little calcification is naturally very faint and would escape recognition during life. A skiagram of such a one taken after its removal from the appendix is shown in the accompanying figure. The concretion was the smaller of two (1107C, St. Thomas's) which were present in the same appendix: neither had been at any time allowed to dry. All the skiagrams here produced, I may say, were from concretions which had been from the first kept in alcohol or formol; the shadow given by objects in the dried state being reduced in depth. The skiagram discloses no lamination, although as studied afterwards the concretion had the usual laminar construction. It was not palpably calcified. Nevertheless, an examination of twenty samples from its whole thickness carried out on the uncovered slide with undilute HCl showed a disengagement of CO_2 in six, the gas invariably coming off from small, pale brown, translucent scales amidst the usual débris.

In the same appendix there was a second, larger concretion of different gross characters, and giving a different skiagraphic picture. It was of dull white colour, 1·7 cm. in chief diameter, with small, deep brown elevations or tubercles projecting through; and one pole was of similar brown colour. One of the elevations proved to be a small fruit seed, the others were projections from a deeper calcified crust beneath. On delamination the white surface-layer showed the usual microscopical elements; no CO_2 was disengaged on the addition of pure HCl. For a depth of 2 mm. below this the substance was of a pale brown colour but soft, like clay; and immediately beneath this lay a hard, deeper brown, comparatively thin, calcareous crust, the whole of the interior of which was filled with a softer substance of paler colour. Microscopic examination, in water, of small flakes of this crust showed the usual homogeneous-looking, fissured material, common to the calcified areas of appendicular concretions; the use of HCl liberated CO_2 and disclosed the presence of some plant cells with entire walls, and the usual débris of disintegrating vegetable tissue, together with wheat or oat hairs and sclerenchymatous cells. The examination of one of the tubercles projecting from the exterior of the crust yielded similar results. The study of the skiagram shows quite clearly the presence of the calcareous zone and of its projecting tubercles; the uncalcified surface-layer being only just discernible.

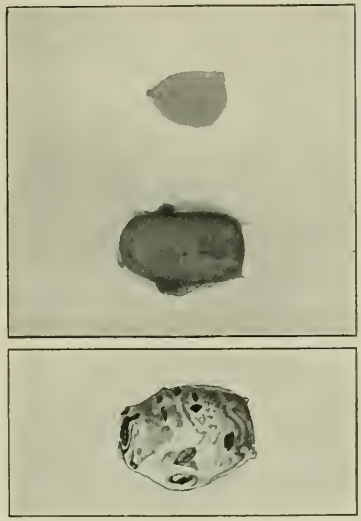

Fig. 6.

Two concretions from the appendix referred to in the text. The skiagram of the smaller shows no differentiated lamination. In that of the larger there is a calcareous zone a short way beneath the surface, furnished with two tubercles. The lowest of the three represents the larger concretion as it appeared to the naked eye. (Natural size.)

Figurae explicatio.

Duarum concretionum ex eadem appendice picturae skiagraphicae. In harum minore pictura nullam dispositionem in stratis demonstrat; in majore, zonam calcificatam superficialem. Concretio ipsa infra repraesentatur. (Magnitudinis naturalis.)

A skiagram of a similarly constructed concretion is shown with the two first described. This, the largest but one of the concretions I have yet examined, came from a gangrenous appendix removed by Dr. C. E. Shattock, to whom I am indebted for many of the twenty-five concretions systematically studied. Its exterior which was hard and of deep brown colour, the pole being corrugated or granular, consisted of a dense calcareous crust, 1·5 mm. thick, within this was buff-coloured fæcal material, moderately firm and laminated, but here and there with darker patches like the calcareous crust. The most central portion of this was examined microscopically *in toto;* it showed the usual elements; a few more resistent particles proved to be calcareous. The skiagram yields a deep shadow which includes the tubercles at one pole, and results from the presence of the calcareous shell referred to; the lesser density of the central substance is likewise evident. Of this crust or shell a full chemical examination was carried out. The following inorganic constituents were demonstrable:—

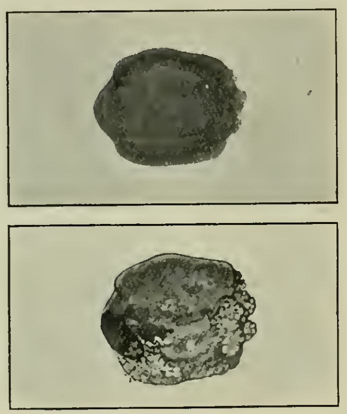

Fig. 7.

A skiagram; and, beneath it, a drawing of a large appendicular concretion. The skiagram shows a thick, calcified crust, the presence of which was confirmed by delamination. (Natural size.)

Figurae explicatio.

Concretionis appendicularis pictura skiagraphica crustam monstrans calcificatam. Concretio ipsa infra repraesentatur. (Magnitudinis naturalis.)

Calcium Carbonate.—The addition of HCl produced an abundant evolution of CO_2.

Calcium Phosphate.—The solution in HCl yielded an abundant yellow precipitate with ammonium molybdate on heating. The presence of calcium was determined by the more recent method of first removing the phosphoric acid from the solution in HCl, by the addition of stannic chloride; on heating, stannic phosphate was precipitated; the fluid was filtered; the excess of tin removed by passing H_2S through the solution, which yielded a yellow precipitate of stannic sulphide; after filtration and the addition of ammonium hydrate to neutralize the HCl, ammonium chloride gave an abundant white precipitate of calcium chloride.

Ammonio-magnesium Phosphate.—The presence of magnesium was shown by concentrating (boiling) some of the phosphoric-acid-free solution and adding sodium phosphate; this produced a crystalline precipitate of ammonio-magnesium phosphate by reason of the presence of free ammonia.

As noticed in the preceding part of this communication (*Proceedings*, vol. ix, p. 59) the intestinal calculi of the horse, consist almost, or in some instances entirely, of ammonio-magnesium phosphate, and give a moderately good skiagraphic picture. Of human enteroliths of this composition there is but one in the Museum of the Royal College of Surgeons (2350·1); it was formed upon part of a tooth which had been accidentally swallowed many years previously.

As an example in which the lamination is only faintly discernible by skiagraphy, I may adduce that of a flattened oval, brown concretion, 1·3 cm. in longer diameter, from a gangrenous appendix removed from a boy, aged 16. On delamination, the surface proved fairly soft; the substance then became firmer from calcareous impregnation, the deposition being limited to parts of the circuit. The lamination was well pronounced throughout. The centre was ill-defined and represented by a group of minute scybala; in the midst of the latter there was a small oval fruit seed, the finely corrugated surface of which showed it to be from the raspberry or blackberry. In the skiagram there is an indication of the lamination in the periphery as well as of the irregularity about the centre. Twenty small fragments were tested with undilute HCl on slides; from all but five CO_2 was disengaged, usually from one or more spots in them.

The skiagram of the last stercolith shows a somewhat voluminous centre, considerably darker than the rest, the peripheral part of which is traversed by one or more lines of similar depth. The concretion was afterwards dried and studied in hemisection. Its centre, which was dark in the skiagram, consisted of opaque buff-coloured material; beyond, it was of deep blackish-brown colour, with white lines corresponding with those brought out in the skiagram. Microscopic examination of the central material: On the addition of HCl the fragments, in distilled water on a slide, cleared up and became translucent, CO_2 being disengaged. The central material, when tested in the usual way, gave a well-marked yellow precipitate, proving the presence of phosphoric acid (calcium phosphate); the flame test carried out upon the concentrated solution in HCl gave the red, calcium colour. This concretion was taken from the excised appendix of a girl aged 9, suffering from peri-appendicular abscess.

Lastly, I may add skiagrams of concretions taken whilst in their excised and unopened appendices. The first appendix was distended with inflammatory exudate, the end having been tied up at the operation, before its division; it came from a boy 7 years of age.

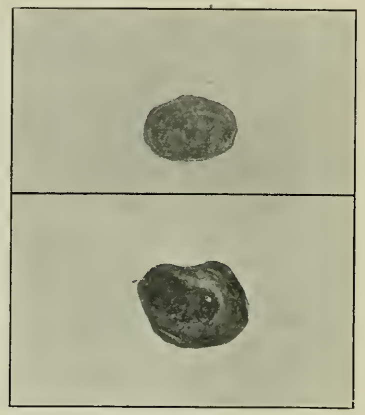

Fig. 8.

Skiagrams of two concretions from different cases; the first showing lamination, but faintly; the lower, well pronounced, with a dark centre. (Natural size.)

Figurae explicatio.

Concretionum picturae skiagraphicae e casibus duobus. In superiore vix apparet dispositio in stratis; in inferiore bene monstratur. (Magnitudinis naturalis.)

The skiagram shows a series of unconnected darker points and incomplete concentric lines in a clear periphery, and a darker, somewhat ill-defined more central portion. The concretion was afterwards removed from the appendix and cut through with a scalpel. It proved to be laminated, soft like clay, and of pale brown colour; in the laminæ were ill-defined patches and lines of white calcareous deposit. An inspection of this divided surface, after drying, shows that the dark spots and lines, and the central area in the skiagram are precisely represented by white calcareous depositions, whilst the rest of the concretion, which is transparent in the skiagram, had become in the process of drying of a deep blackish colour. Fragments of the whiter centre cleared up beneath the microscope on the addition of HCl, and yielded CO_2.

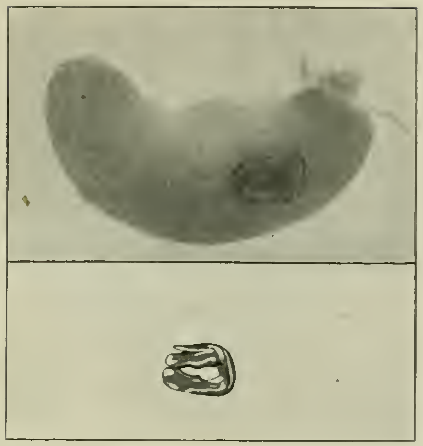

FIG. 9.

A skiagram of an excised, unopened appendix with a contained concretion. Below it is a drawing of the same concretion, dried and bisected: the white areas of calcification correspond with the darker in the skiagram. (Natural size.)

Figurae explicatio.

Pictura skiagraphica appendicis excisae in quâ concretio includitur. Concretio ipsa bisecta infra repraesentatur. Zonulae nigrantes in alterâ ut albicantes in alterâ apparent. (Magnitudinis naturalis.)

In the following case, the skiagram was likewise made with the concretion *in situ*, after the removal of the appendix. The lamination is particularly well shown. A vermiform appendix, the more proximal portion of which is much distended by the formation of an oval concretion of unusual size, measuring 3·5 cm. ($1\frac{3}{8}$ in.) in its longer diameter. This is the largest that has fallen under my notice. The appendix was successfully removed by Mr. Gifford Nash from a man aged 30, who three days previously had been seized with a sudden pain in the abdomen, in spite of which he continued his work until admission. At the operation general suppurative peritonitis was found. After its excision two drainage tubes were inserted into the right iliac fossa and pelvis. There had been no previous attacks, or symptoms of appendicitis.

The skiagram of a stercolith within the excised appendix, in a third case, gave a shadow located to a calcareous centre. On delamination under water the concretion proved to be moderately soft, becoming firmer more deeply; a few fragments of bone were encountered during the process. The lamination was mainly polar, as it is in a ureteral calculus; this is recognizable in the skiagram. The

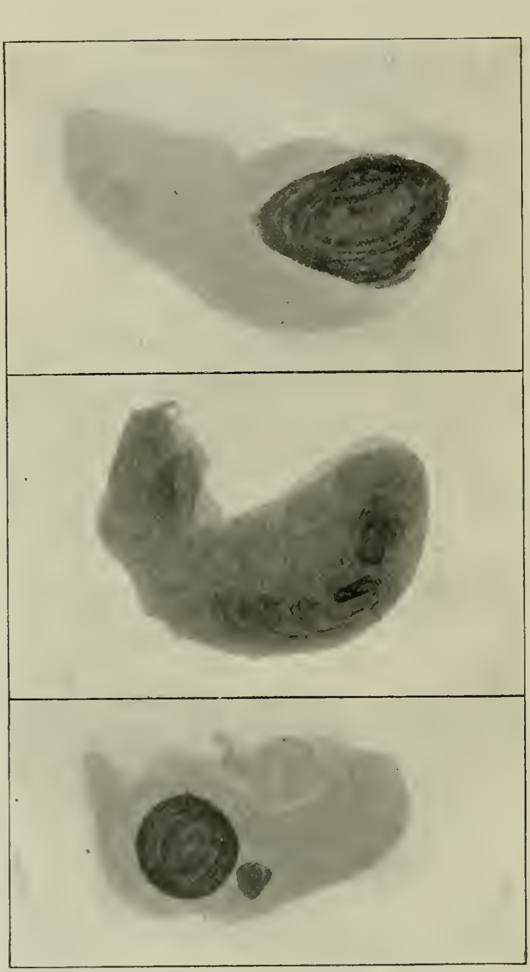

Fig. 10.

A skiagram of three appendices after their excision, with included concretions, referred to in the text. They all exhibit lamination, the highest in a particularly clear degree. In the middle, the centre of the concretion has undergone fracture during life. (Natural size.)

Figurae explicatio.

Picturae skiagraphicae appendicium trium excisarum in quibus concretiones includuntur. In singulis picturis dispositionem in stratis monstrat concretio. Concretionis mediae centrum fractum erat antequam appendix excisa est. (Magnitudinis naturalis.)

double nucleus was of some firmness, but was readily divided with a scalpel; it became white on drying, and crushings yielded CO_2 on the addition of HCl; the decalcified material consisted of the usual vegetable residua. The duplication of the centre is clearly due to fracture, as is proved by the discontinuity of the lamination, and irregularity of the corresponding ends of the fragments.

To conclude with a fourth example, where the concretion was skiagraphed within the appendix after its excision. The lamination is particularly regular and well pronounced. The concretion was afterwards removed, slowly dried, and divided; its macroscopic construction corresponded with that shown in the picture. There is a small, second concretion near the other. The appendix was gangrenous.

The clinical diagnosis of appendicular concretions by skiagraphy, although it might be made at times with certainty, would be unreliable on the side of exclusion; a negative picture would be valueless, so variable is the amount of calcareous deposition. In certain cases, moreover, the appendix lies behind the cæcum, and thus obscured by the contents of the latter.

The Causation of the Stercolith.

The fact that in the great majority of cases no nucleus is present, leaves only one view open as to the initial cause in the production of appendicular concretions, viz., some form of appendicular stasis. This is borne out by the occasional discovery of small scybala in the appendix, and the fact that the centre is at times resolvable into a group of such.

Not only are nuclei of a gross kind almost invariably absent, but there is no central collection of inorganic material, either flakes of enamel from cooking utensils, or particles of steel from roller milled flour, which might serve as a starting point for the concretion.

In any statistical study of appendicitis, those cases associated with the formation of concretions should be grouped apart from the rest; one form may have increased in frequency without the other.

Whether the rise of vegetarianism may be causally related to an increase in concretional appendicitis amongst Europeans may be here suggested as a question for consideration. An exclusively vegetable diet involves a greatly increased intake, and a proportional increase of indigestible residue, with a consequent overloading of the whole intestinal canal, not excluding the appendix. In idiopathic dilatation or megacolon, when the condition in its backward extension from the pelvic colon, reaches the cæcum, the appendix may participate in the enlargement. One of the earlier examples of this disease, and a typical one both clinically and pathologically, is contained in the museum of St. Thomas's Hospital. It occurred in the practice of Dr. T. B. Peacock, by whom it was recorded in the *Transactions of the Pathological Society*, xxiii, 1870. The patient, a man, aged 28, had always suffered from constipation and flatulence, which had much increased during the last six months of life. He had kept his bowels regular for the last ten years by the use of enemata two or three times a week, The large intestine was throughout the greater part of its length 6 in. in diameter; the descending colon 8 in. Over fifteen quarts of moderately firm fæces were removed from the large intestine after death. The appendix in this specimen measures 15·3 cm. (6 in.) in length, and is 1 cm. ($\frac{3}{8}$ in.) in diameter; no statement as to its contents is recorded.

Without an accompanying stasis, nevertheless, a persistent overloading of the appendix with normal fæcal material would not suffice for the production of scybala or of concretions.[1]

The local stasis might arise from a neuromuscular fault or a mechanical one. And whatever general conditions (want of exercise, insufficient intake of fluid, the habitual use of food deficient in residues) lead to stasis of the large intestine, will naturally affect the appendix also. The neuromuscular fault might be a sluggish reflex, achalasia of the orifice, or even spasm of the latter.

It might be argued that whatever interfered with the exit of the appendicular contents would equally interfere with their entry. But it may be pointed out that ingress is easier than egress, since the contraction of the cæcum upon its contents aids in the one, whilst the other depends upon the appendix alone.

In his work on the operative treatment of "Chronic Intestinal

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[1] That vegetarianism *per se* is not a cause of concretional or other forms of appendicitis is shown by the infrequency of the disease amongst natives of India. The amount of exercise taken by the native cannot be put as a counterpoise to the overloading of the intestinal canal with rice and pulse, for it is less than amongst Europeans. Of the Bengalis and Biharis, Major E. O. Thurston, I.M.S., writes to me that, speaking generally, no native takes exercise for the sake of it. The younger students play cricket and football, but cease as soon as they leave college; the children and young men of the wealthy overeat habitually, seldom take exercise, and are grossly obese. The agriculturist, on the other hand, works for many hours daily, although without the expenditure of much effort. (This class forms about 80 per cent. of the entire population.) Defæcation is carried out amongst all classes, in the squatting position, and so is micturition.

Stasis" (3rd ed. 1915), Sir Arbuthnot Lane gives two modes in which the appendix may become involved in ptosis of the colon. It may become fixed in acquired adhesions, along the outer or inferior surfaces of the cæcum, and made to share in supporting the latter; it may be secured by its extremity or at any point in its length. In the former case the drag exerted upon it at its junction with the cæcum tends to interfere with the passage of material from it into the cæcum, whilst in the latter case the appendix is also kinked at the point in its length where it is fixed by these acquired bands.

The presence of kinks as a cause in the formation of concretions, however, must be exceptional. It is against surgical experience to find the axis of the acutely inflamed appendix containing a concretion, otherwise than normal; mere curvatures are not to be confused, of course, with proper kinks; nor are the kinks producible in the delivery of an adherent appendix to be mistaken for those present *in situ*.

Besides kinks, mechanical obstructions may result from the effects of inflammation and ulceration. Out of 100 appendices taken from persons dying from other causes than disease of the appendix, and over 40 years inclusive, I found the lumen of the organ obliterated throughout in nine; obliterated at the distal end in twelve; the orifice closed, without complete obliteration of the lumen, in three.

In the group of twenty-five appendices taken after death, from subjects dying from other causes than disease of the appendix, between the ages of 15 and 25 years inclusive, there were four in which the mucosa presented a varying number of small ulcers. It is somewhat remarkable that all these patients were suffering from pulmonary tuberculosis.

No. 4.—Male, aged 18. About 1 cm. from the distal end, there were two small ulcers, side by side, with raised edges and necrotic surfaces; and about 1·5 cm. on the proximal side of these, were two whitish hemispherical elevations, probably infected lymph follicles. Post mortem: Pulmonary tuberculosis, associated with tuberculosed mediastinal glands. Microscopic examination showed the presence of foci of necrosing tissue in the edges and bases of the ulcers, accompanied with marked chromatolysis; the muscularis was involved. No giant cells, however, were encountered. The lesion, therefore, cannot be relegated to a tubercular infection.

No. 12.—Male, aged 19. In the distal end there was a small oval ulcer, 4 mm. in chief diameter, and some lesser punctate ulcers apparently arising in the lymph follicles. Pulmonary tuberculosis, accompanied with tubercular meningitis, and miliary tuberculosis of the liver, spleen and kidneys.

No. 17.—Female, aged 16. Towards the distal end there was a small group of ulcers. Phthisis.

No. 21.—Male, aged 25. In the mucosa there were one or two small ulcers. Pulmonary tuberculosis accompanied with meningitis.

That the formation of concretions may proceed without the occurrence of inflammatory attacks is a clinical commonplace. Their formation is nevertheless to be reckoned as a predisposing cause of acute inflammation, since the presence of a stercolith involves a local distension of the part around it, and a consequent local diminution in resistance to bacterial infection. It is well known, indeed, that the fulminating forms of appendicitis are invariably associated with the presence of a concretion. The proportion of concretional appendicitis to other forms is a subject that would need statistical inquiry; one form as already remarked, may have increased in frequency, and not the other.

I may conclude this communication with the results of an examination of the contents of the appendix in 125 subjects dying from various causes unconnected with appendicular disease. The object of this investigation was to ascertain whether the ingestion of particles of steel, or flakes of hardware enamel could be put forward as a cause in the production of appendicitis.

An Examination of the Contents of 100 Appendices taken after Death from Subjects of 40 Years and upwards; and of Twenty-five Appendices, between the Ages of 15 and 25 years inclusive: from Cases unconnected with Appendicular Disease.

Method of Observation.

The first series of 100 observations were confined to subjects of 40 years of age and upwards, in order to allow of ample time having elapsed for the accumulation of any regularly ingested foreign inorganic material in the appendix. No cases were used in which appendicular disease was in known progress at the time of death. The whole of the material was obtained from the post-mortem room; none from dissection subjects. Immediately after its removal from the body, the organ was laid in diluted alcohol, on cotton wool, and was examined on the following day. It was fixed to a sheet of gutta-percha, slit up under water with fine scissors and pinned open; the whole procedure was conducted in a white porcelain dish furnished with a lip at one corner. A microscopic preparation in dilute glycerine was made from the contents *in situ;* the remainder of which was then lightly brushed out and broken up with a brush (hog-hair) in water; any sedimentary material which appeared was irrigated, and submitted to microscopic examination in dilute glycerine.

The findings in the first hundred cases may be grouped under the following headings:—

(1) The appendix completely empty.

(2) The contents consisting of shed appendicular epithelium, without fæcal coloration, and yielding no sedimentary residue on irrigation.

(3) Fæcal material, or shed appendicular epithelium intermixed with fæcal material as small oval pellets, or in a formless condition.

(4) Blood, in cases of melæna.

(5) Fat crystals in pancreatic disease.

(6) Thread worms and whip worms.

(7) Foreign bodies.

(8) Obliteration, in different degrees.

(9) Orifice closed.

(10) Neoplasms.

(1) *The Appendix Completely Empty.*

This was observed on three occasions only:—

No. 25.—Aged 56, male; a comparatively small appendix, lumen patent throughout. Death from acute obstruction, 4 ft. of the ileum having prolapsed through an omental slit.

No. 35.—Aged 54, male; appendix slender; lumen narrow. Death from alcoholic pneumonia.

No. 52.—Aged 53, female; death from acute obstruction; fæcal vomiting. After death the abdominal viscera were found matted together; pelvic adhesions; fibromyomata of uterus.

The empty condition of the appendix in two of the cases may be referred to the intestinal obstruction on the proximal side of the large bowel. In the third the appendix was slender and its lumen narrow, the organ being atrophic and functionless.

(2) *The Contents consisting of Shed Appendicular Epithelium, without Fæcal Coloration, and yielding no Residue on Irrigation.*

This was observed in twenty-three cases. Under these circumstances, on slitting up the appendix beneath water and carefully pinning it out, the contents present themselves as a cylinder of varying thickness in different cases; and in the same appendix, it may be interrupted, the cylinder being white and usually friable. When brushed in water no sedimentary residue is disclosed. Microscopic examination shows the material to consist solely of columnar epithelial cells, with, it may be, a small amount of mucin; the cells are at times in excellent condition; at others the majority are granular and degenerate.

No. 3.—Aged 40, male; death from cerebral tumour.
No. 6.—Aged 66, female; carcinoma of rectum.
No. 8.—Aged 41, female; chronic nephritis.
No. 9.—Aged 69, female; pernicious anæmia.
No. 10.—Aged 53, male; abscess of lung.
No. 15.—Aged 65, male; enlarged prostate.
No. 24.—Aged 69, male; carcinoma of prostate.
No. 26.—Aged 42, male; biliary calculus.
No. 30.—Aged 70, female; strangulated hernia.
No. 45.—Aged 44, male; cirrhosis of liver and endocarditis.
No. 47.—Aged 50, male; mediastinal tumour.
No. 48.—Aged 64, female; arterial atheroma.
No. 53.—Aged 54, male; ulceration of cæcum.
No. 64.—Aged 40, female; pancreatitis and chronic biliary obstruction.
No. 65.—Aged 46, female; pneumonia.
No. 68.—Aged 40, male; aortic and mitral disease.
No. 78.—Aged 57, female; perforating gastric ulcer.
No. 90.—Aged 42, male.
No. 91.—Aged 47, female.
No. 93.—Aged 57, female; suppurating pleurisy and peritonitis.
No. 94.—Aged 46, male; miliary tuberculosis of lungs.
No. 97.—Aged 73, female; obstructed femoral hernia.
No. 98.—Aged 40, female; rupture of uterus from obstructed labour.

The presence of shed epithelium in these cases, unmixed with fæcal material proves that the organ does not regularly participate in the intestinal peristalsis. For the amount of epithelium occupying its lumen in some of them indicates that the shedding and accumulation had been proceeding for some time. This is parallel with the accumulation of shed epithelium which takes place in an isolated loop of the living intestine.

(3) *Fæcal Material.*

In fifty cases the appendix contained simply fæcal material, or shed epithelium intermixed with fæcal material as small oval masses or in a formless condition. A microscopic examination was made of the residues left from the washings, but nothing of sufficient interest to record, was found.

(4) *Blood in Melæna.*

In nine cases blood was found in the appendix. These were: in four instances, gastric ulcer; in two, duodenal ulcer; in three, carcinoma of the stomach. In none was the mucosa of the appendix pigmented, after the manner described in the first part of this communication. This adds to the reasons for viewing the pigmentation as hæmatogenous or of the nature of an internal excretion.

(5) *Undigested Fat.*

This was found once, viz., in a case of pancreatic disease.

No. 81.—Aged 55, male. Death from carcinoma of pancreas. Contents: A somewhat voluminous cylinder consisting in part of the uncoloured, friable material characteristic of shed epithelium, but chiefly of a peculiarly opaque white substance mingled with it. A microscopic specimen made directly from the lumen shows the latter to consist of large numbers of acicular crystals of medium length; they are mostly without any order, but some occur as loosely arranged spheroidal collections, some of which lie in groups suggesting that they have been produced within fat cells; and unaltered fat is here and there present. The crystals become of a distinct red when treated with scharlach; the fat droplets, of a deep scarlet. The contents when lightly brushed with a little water assumed a cream-like appearance. From the washings was obtained a small collection of pale reddish brown, flattened, flexible particles; in all, about the volume of a hemp seed. These consisted of vegetable tissue. Besides such there were a few fragments of striated muscle fibre; some embedded in the spherular groups of acicular crystals. The presence of crystalline fat and of droplets of oil, is, of course, referable to a defective output of pancreatic secretion, resulting from the disease of the gland.

(6) *Animal Parasites.*

These were found in four instances :—

No. 1.—Aged 62, male; two oxyurides (thread worm).

No. 43.—Aged 60, female; one oxyuris.

No. 74.—Aged 51, male; one oxyuris.

No. 80.—Aged 68, male; one trichocephalus (whip worm).

The percentage of *Oxyuris vermicularis* (3 per cent.) closely corresponds with Brumpt's (1910), who examined 800 appendices from autopsies in Paris, and found the percentage from 3 to 5. In children, as determined by Still (1899), the percentage is much higher, and in healthy appendices reaches 19 per cent. The occurrence of *trichocephalus* and *Ascaris lumbricoides* is rare. My own data give 1 per cent. for the former.

(7) *Foreign Bodies.*

Foreign bodies, of small size, were found in three cases of the 100 :—

(1) A fragment of a slender fish bone.

(2) A fragment of brown bast fibre.

(3) A few animal hairs.

The last were found in a female, aged 50; they were five in number, and lay in white friable material (shed epithelium) in the distal end beyond a sharp bend at the junction of the middle and distal thirds. (Specimen No. 16.)

The hairs were fine, unpigmented, disposed lengthwise, and corresponded in microscopic characters with the human.

(8) *The Appendix Obliterated (A) Throughout.*

This was met with in nine cases of the 100.

No. 1A.—Aged 55, female; the appendix converted into a long slender solid cord.

No. 4A.—Aged 40, male; the appendix converted into a slender solid cord.

No. 9A.—Aged 47, male; a solid cord; obliterated throughout.

No. 19.—Aged 82, male; obliterated throughout, without being obviously abnormal externally.

No. 20.—Aged 55, male; a cord, of which the lumen is mostly obliterated; in spots a minute but quite empty lumen appears to be present.

No. 23.—Aged 64, male; a solid cord, somewhat contracted in thickness.

No. 27.—Aged 48, male; a solid cord except for the proximal 1 cm.

No. 39.—Aged 42, male; completely obliterated throughout.

No. 62.—Aged 54, female; a solid cord, not particularly contracted in diameter.

(*B*) *Obliteration of the Distal End.*

In the following twelve cases the distal portion only was obliterated: these are used a second time in the other groups, for their contents.

No. 9.—Aged 69, female; distal 2 cm. solid.

No. 64.—Aged 40, female; gradually narrowing for 3·5 cm., then solid.

No. 5.—Aged 49, male; distal 1·5 cm. solid.

No. 11.—Aged 63, male; distal 3 cm. solid.

No. 13.—Aged 47, female; distal 2·5 cm. solid.

No. 17.—Aged 47, male; distal 2 cm. solid.

No. 50.—Aged 55, female; distal 1 cm. solid.

No. 56.—Aged 42, male; distal 1 cm. solid.

No. 75.—Aged 50, male; distal 1 cm. solid.

No. 80.—Aged 68, male; distal 1 cm. solid.

No. 87.—Aged 65, female; distal 1 cm. solid.

No. 100.—Aged 51, male; distal 2 cm. solid.

In regard to these findings, in 9 per cent. the obliteration was complete; in 12 per cent. it was limited to the distal end. In four of the nine, the ages were under 50; and in six of the twelve, the same. It cannot be held, therefore, that the obliteration is merely a senile form of atrophy, as has been sometimes supposed. The probability is that both the complete and the incomplete occlusion is due to past inflammation, which has been followed by epithelial desquamation, and subsequent cohesion.

In twenty-five further appendices taken between the ages of 15 and 25 inclusive, there were two of incomplete occlusion; none of complete. In one of these two (No. 24), the appendix was from a male, aged 17: the terminal 2·5 cm. were solid. In the other (No. 5), a female, aged 15, the distal 1 cm. was solid; the whole organ was not more than 5 cm. (two inches) in length; the cæcal orifice was closed.

(9) *Closure of the Cæcal Orifice.*

In three cases the orifice was closed.

No. 31.—Aged 65, male; cæcal orifice closed; appendix of full size. Contents: somewhat abundant white granular material, which on microscopic examination was found to consist of shed columnar epithelial cells, and much finely granular débris, with a certain number of polymorphonuclear leucocytes. It presented four or five slender, incomplete diaphragms; and areas of sieve-like appearance, resulting from enlargement of the gland orifices.

No 79.—Aged 66, male; cæcal orifice closed. A firm, plump, appendix, unusually stiff. Lumen irregular; results of ulceration, recognizable; a little mucus lying here and there on the mucosa.

No. 85.—Aged 67, female; cæcal orifice closed; the distal end for 5·5 cm. converted into a solid cord without being markedly shrunken. Beyond the obliterated orifice the lumen for 3 cm. is slightly dilated, and contains a soft white material which proved to be shed epithelium.

(10) *Neoplasms.*

In two cases new growths were encountered, which had escaped diagnosis during life. As both forms of growth are uncommon in this particular position, I have ventured to introduce figures of them.

No. 2.—Aged 42, male; appendix unusually large. Contents: Pale yellow, granular, with some mucin and a little blood clot; no sedimentary residue on irrigation. This appendix was the seat of a typical carcinoma, resembling in miniature exactly such a one as might occur in the colon. I have thus described the specimen in the catalogue of the Royal College of Surgeons' Museum.

1,755 (1). A vermiform appendix which is somewhat dilated and elongated; the two or three circular apertures seen from the interior are the mouths of small sacculi. Occupying about 4·5 cm. ($1\frac{3}{4}$ in.) of its lumen, towards the proximal end, there is a new growth, the distal part of which is pedunculated and papillary; elsewhere the tumour is superficially ulcerated, and, as seen in the section, has invaded the muscular wall. Microscopic examination shows it to be a columnar-celled carcinoma. From a man, aged 42, who was admitted with a hard nodular mass in the epigastrium. After death this was found to lie between the liver and transverse colon—i.e., it was peritoneal; the gut was embedded in its lower border, but no perforation had taken place. The gall bladder, which contained calculi, was completely surrounded, and infiltrated with new growth. Sessile and pedunculated masses were freely scattered over the peritoneum. The stomach and duodenum were intact, although the lumen of the latter was narrowed by the carcinomatous mass external to it. The head of the pancreas was involved in the new formation, the primary site of which was possibly this gland. Histologically the growths in the various positions, other than the appendix, proved to be carcinomata of spheroidal-celled kind. It would seem therefore that the typically columnar-celled carcinomatous neoplasm in the appendix was of independent origin. That it arose by the direct implantation of a fragment detached from the growth in the pancreas or gall-bladder and conveyed by way of the common duct into the intestine, is improbable; the proteolytic action of the pancreatic ferment renders such a supposition unlikely. Carcinoma of the stomach, although so very common, is never followed by implantation-metastasis in the intestine.

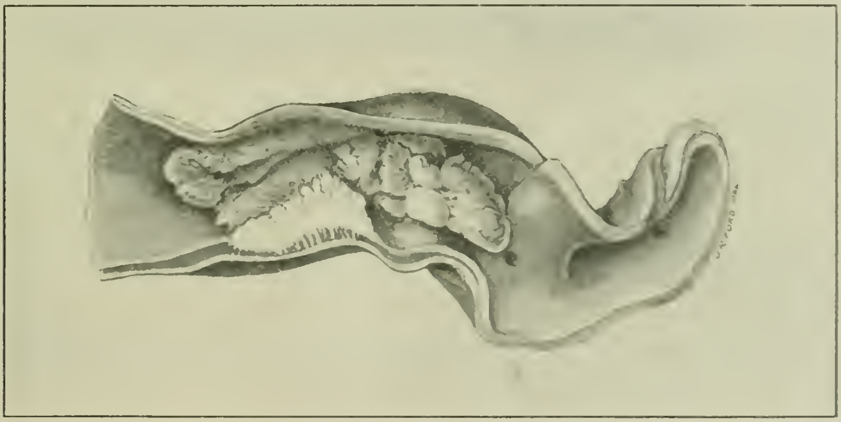

FIG. 11.

A vermiform appendix into the interior of which there projects a voluminous, necrosing tumour, which has invaded the muscular wall, and which microscopic examination shows to be a columnar-celled carcinoma. (Natural size.)

Figurae explicatio.

Appendix vermiformis cujus in cavitate projicitur tumor, pariete musculari invaso, quem scrutatio microscopica carcinoma esse demonstrat cellulis cylindricis constructum. (Magnitudinis naturalis.)

No. 66. Aged 53, male; a particularly well developed negro. A long and proportionally large appendix. Contents: an incomplete voluminous cylinder of white friable material (shed epithelium) admixed in the distal 5 cm. with pale yellowish brown fæcal material. Washings from the interior: a small quantity of very fine brownish particles, not sufficiently heavy to settle with any rapidity, and of the kind found to consist in other cases, of plant débris. Growing from the mucosa, near its middle, there are two small pedunculated tumours, like those at times found in the colon. Microscopic examination shows them to be simple adenomata. The intestine in this case was not slit up and examined at the autopsy. Whether it was similarly affected or not cannot, therefore, be stated. The cause of death was nephritis. The specimen is preserved in the Museum of the Royal College of Surgeons (1513·2).

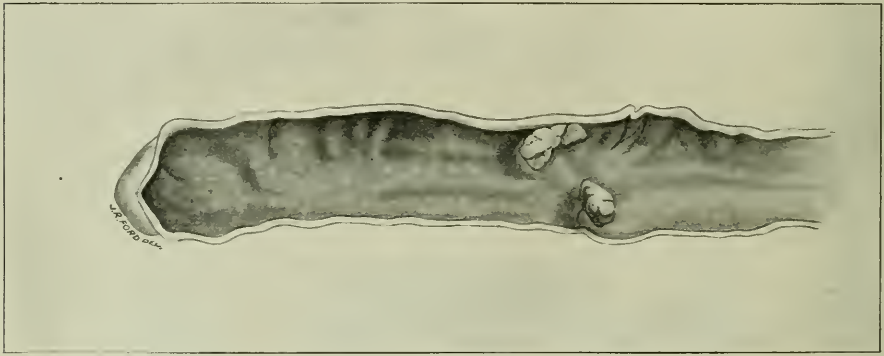

Fig. 12.

A vermiform appendix, showing two small pedunculated adenomata growing from the mucosa. Natural size.

Figurae explicatio.

Appendix vermiformis cujus in cavitate projiciuntur duo tumores glandulares quorum uterque pedunculo tenui praeditus est.

An Examination of the Appendicular Contents from Twenty-five Subjects between the ages of 15 and 25 years inclusive, dying from Causes other than Disease of the Appendix.

This decade was selected since it is that in which appendicitis is most common. The contents comprise shed epithelium admixed with fæcal material; or either of these singly. In three cases, small, more resisting pellets, or scybala, were present; these on being crushed in glycerine and examined microscopically, were found to consist of the usual disintegrating plant structures, without any differentiated centres;

they have been already referred to under the heading of Appendicular Scybala. A microscopic examination of the contents was made in every case. It disclosed nothing new, and nothing, therefore, that needs detailing, except that in one instance there were a few microscopic angular scales of reddish brown colour, which slowly disappeared on running HCl under the cover glass, and may be taken as rust, for like some forms of ferric oxide they presented a subcrystalline structure, and became paler before their disappearance.

Conclusions.

Promising as the suggestions are, viz., that the ingestion of particles of the enamel detached from cooking utensils, or of particles of steel from flour milled with steel rollers, is causally related to the production of appendicitis, the verdict is against both. In an examination of the contents of 100 appendices taken from persons over 40 years of age dying from disease unconnected with the appendix, I have not encountered either a macro- or a microscopic particle of either; and the same is true, with one exception, of the contents of twenty-five appendices taken between the ages of 15 and 25 years, the decade in which appendicitis is most common; in the case referred to, the contents included a few microscopic flakes of ferric oxide.

Moreover, a critical microscopic examination of the centres of twenty-five appendicular concretions revealed no particles either of enamel or of steel, which might have acted as nuclei in their production.[1] Whatever may have been the case in the past, flour as at present prepared with steel rollers, is free of visible metallic particles.

This conclusion is confirmed by the chemical examination of roller-milled flour, for inorganic iron—i.e., iron not organically combined with it, the well known presence of which latter is demonstrable only after incineration. The finest roller-milled flour is passed between no fewer than thirteen pairs of rollers; and this, if any did, should contain inorganic iron in conspicuous amount. Between the processes of rolling, the bran or pericarp of the "berry" is removed by means of silk sieves, and the use of air-currents, until there remain only single and clustered grains of starch, with fragments of the cells which contained them. In such finely prepared flour, nevertheless, a minute examination (made by adding glycerine and distributing the mixture beneath a cover glass) will reveal the persistence of a certain number of minute fragments of pericarp, together with pieces of the stiff, thick-walled hairs which grow from its outer layer. The embryo or "germ" is completely removed in the early stages of the process. The presence of pericarp in the finest white flour proves that this source of the "accessory factor" or vitamine is never *completely* removed, although the other source, the germ, is. Some of these fragments include cells of the aleurone layer, with their contained granules; some the coloured film and cells external to it; and others the coloured cells only.

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[1] Mr. Wilfrid Trotter tells me that he once removed a flake of such enamel, as large as a sixpence, from the anus, where it had produced a fissure.

I may here point out that in appendicular contents minute scales are not infrequently found, which at first suggest enamel. The scales are translucent, angular, sharply fractured, of buff colour and, curiously enough, often include blackish brown conidia and short fragments of mycelium. They are unaffected by the strongest HCl. On gently warming with HNO_3, however, they rapidly disappear, whilst an oily looking scum rises to the surface of the acid, and a microscopic examination shows that the scale has been replaced by groups of colourless, transparent droplets. These puzzling objects clearly consist of a soap, or complex body from which the HNO_3 has split off some form of fat.

Dr. H. Maclean tells me that the same reaction is given by certain of the fatty substances once known as Protagons. In the form of compact tresses of remarkably long acicular crystals, a chemical substance giving the same reaction may be met with in appendicular contents; the droplets into which the crystals are transformed by means of HNO_3 have all the microphysical aspects of fat; and after being washed, are colourable with scharlach solution.

The following tests for inorganic or uncombined iron were carried out upon two samples of stone-milled flour obtained from different sources; and one of the best white, roller-milled kind. The action of dilute hydrochloric acid was allowed to proceed for ten days at the room temperature, the flasks being closed with rubber stoppers, and the contents swirled daily. Of each sample of flour, 7 grm. were used, and to each flask were added 40 c.c. of a mixture of one volume of HCl and two volumes of distilled water. The filtrate gave no immediate reaction with freshly made solution of potassium ferrocyanide. Other portions of the filtrates were tested with the extremely delicate reagent, ammonium sulphocyanide, 2 c.c. of a solution being added to a volume of the filtrate that was equal in each case, the test tubes used, moreover, being of the same calibre. In all three, a pale pink was produced, showing the presence of a minute trace of iron. The tint was decidedly in excess of that hardly recognizable when the reagent was added to a mixture of one volume of HCl to two of distilled water; it being well known that the acid frequently contains a faint trace of iron. A critical inspection, made by viewing the contents of the tubes from above, showed that the tint in the case of the steel-milled flour was very slightly deeper than in the stone-milled; this minute excess is, of course, attributable to the action of the rollers.

The presence of this trace of inorganic iron in flour is explicable, I believe, in the following manner: On submitting *intact wheat* to the action of HCl, using a similar weight, viz., 7 grm., and 40 c.c. of the same dilute HCl, I found that the filtrate after the same ten days' maceration, gave as pink a tint with ammonium sulphocyanide solution as the flour. The wheat was crushed in the acid solution in a chemically clean glass mortar. Furthermore, the same weight of *bran* gave a deeper pink than the wheat. The whole of these observations were carried out contemporaneously, the series of colours being compared side by side.

The conclusion to be drawn is that the trace of iron in the different kinds of flour is already present in the untreated wheat, and this can only be derived from inorganic dust. Any sample of earth, or indeed of dust (even that deposited on the shelves of a pathological laboratory), when boiled with dilute HCl will yield a filtrate which gives a well pronounced blue colour with potassium ferrocyanide. The source of the iron-containing dust in the grain, it may safely be assumed, is the wheat-field.

Observation: July, 1920. A small amount of the soil from a Surrey wheat-field (some way from a main road) was dried on glass, and powdered with the finger. Of the finest portion, 2 mg. were boiled with 8 c.c. 25 per cent. HCl, and filtered. The clear filtrate yielded a well-marked blue with potassium ferrocyanide; with potassium ferricyanide, no trace of colour. The acid solution itself gave no reaction with either.

Some such dust may be carried by the wind; but the corn, as it falls, on being cut, and during its stacking, will be readily contaminated from the soil. The actual grain is, it is true, protected by its glumes and paleæ; but in the process of thrashing, the pericarp will be freely exposed to the dust already on, and raised from the outer coverings, as well as from the stalk, splashed, too, it may be, with soil from heavy rain. The reason why a more pronounced reaction is given by the bran (or pericarp) is that this would naturally, weight for weight, represent a larger area exposed to contamination.

A microscopic study of the pericarp by means of longitudinal and transverse sections made after the dried, ripe grain had been soaked in water, and then slowly hardened in increasing strengths of alcohol, the sections being mounted in glycerine, shows its structure to be, briefly, as follows. Proceeding from without inwards, it consists of: (1) Two layers of flat, much elongated, and longitudinally disposed cells; (2) a single layer of somewhat similar cells, the long axes of which cross the preceding exactly at right angles; (3) a thin sheet or film of homogeneous, orange-coloured material, around which no enclosing cell walls can be discovered ($\frac{1}{12}$ oil), the material lying simply between (2) and the aleurone cells which succeed (this is the source of the familiar colour of bran); (4) a single series of thick walled, cuboidal cells, each of which encloses a compact mass of aleurone granules (as viewed on the flat, these cells are polyhedral): to this succeeds the larger, more thinly walled cells of the endosperm, filled with starch grains.

Is the coloured substance the holder of the water-soluble vitamine? Dr. Gowland Hopkins, to whom I made this suggestion, writes to me, that most persons "think in particular of the aleurone layer" (as the seat of the vitamine) "but on no direct evidence."

If bran is soaked, or boiled in distilled water, the filtrate is of a faint yellow tint, whereas if the bran is treated with absolute alcohol or with xylol, no trace whatever of colour is extracted. This may be worth notice, since plant vitamine in other cases appears to be associated with colour-substances. The determination of this point, however, as Hopkins admits, would be exceedingly difficult.

The presence of inorganic iron in *Appendicular Concretions is readily* demonstrable; but in this they do not differ from ordinary fæcal material. Of three full-sized stercoliths, one yielded no iron; the second a trace; the third a well-pronounced amount; a fourth, of smaller size, gave as good a reaction as the last. The four concretions were from different cases.

In *Normal Fæces*, iron in an inorganic form is of constant occurrence, but the following observations may be cited in connexion with the subject under consideration.

Observation: The material examined, which was, of course, from a healthy person living on a regular and ordinary diet, was treated (when quite fresh and in small bulk) many times with alcohol, and kept at 37° C., in order to remove as far as possible the stercobilin; and afterwards dried in the incubator. The bulk finally used was that of a moderately sized concretion. This was ground in a glass mortar with 25 per cent. HCl and filtered. The filtrate was clear, but of a moderate yellowish-brown tint. With this, potassium ferrocyanide solution gave an immediate blue, which appeared as green owing to the coloration of the filtrate, showing the presence of a ferric compound: with potassium ferricyanide, a marked green, indicating the presence of a ferrous compound derived from the ferrous sulphide, the presence of which appeared from the evolution of H_2S on the addition of the HCl during the preliminary trituration. A second sample, of a different date, yielded precisely the same results.

That the source of the inorganic iron is not attributable solely to the ingestion of flour was proved as follows:—

Observation: The use of flour in every form (bread, pastry) was discontinued for a week, the starch consumed being limited to potatoes, rice (in grain), and oats (unrolled). A sample was taken on the fifth, and a second on the seventh day; and in conjunction with these a sample was examined which was obtained the day before the change of diet. These were treated with alcohol, and afterwards dried as already described. An accurate comparative study was made of the three, by taking 2 grm. of each sample of the dried material (somewhat more than that composing a large stercolith), rubbing it in a glass mortar with 20 c.c. 25 per cent. HCl, and filtering. From each sample the HCl at once liberated H_2S, showing the presence of iron combined as ferrous sulphide. The amount was equally marked in all. The filtrate in each was clear, and of moderate yellowish-brown tint. Equal quantities were put into six test-tubes of the same calibre, and tested, three with potassium ferrocyanide solution just made, and three with potassium ferricyanide. With the ferrocyanide each gave an olive-green colour; i.e., Prussian blue masked by the yellowish-brown tint of the filtrate. (If the ferric reaction is carried out in fluid tinted with picric acid this modification of the blue is very obvious.) With the ferricyanide a very pronounced, deep bluish-green resulted; thus proving that the iron present was chiefly in the form of ferrous chloride derived from the ferrous sulphide by the action of the hydrochloric acid. On the closest scrutiny I could detect no difference in the depth of coloration of any of the three tubes of each group: there was as much iron in the two samples where the use of flour had been strictly excluded, as in that taken the day before its exclusion.

The presence of iron in the fæces, in an inorganic form, chiefly or entirely combined with sulphur, has been long known to physiological chemists; and with the remark that the knowledge antedates the rise of appendicitis I may conclude.

> "In den Fäcalstoffen ist das Eisen meist hauptsächlich oder ganz als Schwefelverbindung enthalten."[1]

---

[1] Hoppe-Seyler, "Physiologische Chemie," 1881.

Some Experiments bearing on the Local Formation of Antibodies.[1]

By W. W. C. Topley, M.D., F.R.C.P.

The experiments here briefly reported were undertaken in order to see whether any evidence could be obtained of the local formation of antibodies or of possible intermediary substances, at the site of inoculation of a relatively large dose of a killed bacterial suspension. Since the results were uniformly negative they will be dealt with very briefly.

The method adopted was the preparation of an extract of the connective tissue, mainly muscle, into which the suspension had been previously injected, and the examination of this as regards (*a*) its power of inducing phagocytosis of the homologous bacteria, and (*b*) its influence (if any) on the phagocytosis of the same bacteria in the presence of normal and of immune serum. A similar tissue extract from a part remote from the site of inoculation was examined as a control.

The animals employed were rabbits. The bacterial suspension was prepared from a strain of the *Staphylococcus aureus*, and the same strain was used throughout, both for the inoculations and for the phagocytic experiments. The injections were made in all cases into the thigh muscles of the left leg. The dose given contained in most cases 1,000,000,000 killed cocci per kilo body weight of the animal employed. In a few cases it contained ten times this amount. In certain experiments a second inoculation was made after a varying number of days, at the same site as the first. In every case a control inoculation, consisting of the same volume of saline solution was made into the thigh muscles of the right leg.

The extracts were prepared as follows: The animal was anæsthetized and the subclavian artery was cut across. A sample of blood was retained and the animal was then allowed to bleed to death. The abdomen was opened and a canula inserted into the descending aorta, and the animal was perfused with one litre of sterile saline. As soon as the perfusion was completed, a portion of the tissues from each thigh in the immediate vicinity of the inoculations, was removed aseptically. The tissues were finely minced, while kept at a temperature of 0° C., and were then ground with sterile sand at the same temperature. The pulverized mass was then transferred to sterile glass tubes and submitted to high speed centrifugalization. In most cases the extracts were prepared one day and tested after being on ice for twenty-four hours.

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[1] At a meeting of the Section, held April 20, 1920.

The rabbits were killed and the extracts prepared at varying intervals after the inoculations. These intervals actually varied between one and a half hours and nine days.

The bacterial suspension used in the phagocytic experiments was in all cases prepared from a twenty-four hours' agar growth of the same staphylococcus as was employed for inoculation. Washed human leucocytes were used in all cases. Incubation was carried out in capillary tubes for fifteen minutes at 37° C. Films were prepared in the usual way and the bacteria contained in 50 cells were enumerated in each film, the number of phagocytic and non-phagocytic cells being also noted.

In every experiment the extract from the site of inoculation and the control extract from the tissues of the opposite thigh were tested for the presence of opsonin. They were also tested in the presence of one or more specimens of serum from the animal from which they had been prepared, and in many cases against the serum from a normal rabbit. It seemed possible, in view of our knowledge of the properties of exudates resulting from bacterial injections, that the extracts, even if devoid of any power of inducing phagocytosis, might either increase or diminish the opsonic action of the blood serum; and that any influence of this kind might vary, in degree and perhaps in direction, with the interval elapsing between the inoculation of the bacterial suspension and the preparation of the extract.

There are certain well-known sources of error involved in experiments of this type. Such tissue extracts have, in the great majority of cases, the power of binding complement; and their chemical and physical constitution is so complex that the nature and extent of their reactions with serum, apart altogether from any antibodies which they might contain, is at present largely unknown.

All the extracts studied in these experiments had the power of binding complement. On the other hand the routine control test with an extract prepared from a similar tissue remote from the site of inoculation, as well as several control experiments carried out with similar extracts prepared from normal rabbits, showed no evidence of any influence on the degree of phagocytosis induced by normal or immune serum. Titration of the extracts as regards their anti-complementary power showed, in almost every case, that this was abolished by diluting them ten times with normal saline, and in many experiments observations were made on the effect of extracts, diluted to this and to higher degrees, on phagocytosis.

The whole series of experiments gave uniformly negative results. The extracts themselves never induced more than a minimal degree of phagocytosis. They neither increased nor decreased the degree of phagocytosis occurring in the presence of normal or immune serum. The extracts from the tissues at the site of inoculation of the bacterial suspension differed in no demonstrable way from those prepared from other situations in the same animals, nor from similar extracts prepared from normal animals.

While these results are thus only of negative interest, they form one more instance of failure to obtain evidence of the local formation of antibodies, and to this extent may perhaps render us still more critical of the somewhat slender evidence on which the view that such local production does occur is based.

The experiments most frequently quoted in support of this contention are those of von Dungern, since these constitute one of the few instances in which definite evidence of local antibody production seems to have been obtained. This investigator, while engaged in immunizing rabbits to majaplasma, made the observation that the aqueous humour showed no trace of precipitin for this substance, although the blood serum contained large amounts. He further demonstrated that simple removal of the aqueous humour caused no subsequent passage of antibodies into the anterior chamber of the eye; and concluded that, so long as the tissues of the iris remained intact, the anterior chamber was cut off from all precipitin produced elsewhere. Several rabbits were taken and the aqueous humour was withdrawn from the right eye of each and replaced by majaplasma diluted four times with water. In several rabbits suppuration supervened and so spoiled the experiment. In one rabbit, however, the eye healed rapidly and after eight days showed no signs of inflammation. The aqueous humour withdrawn from it remained perfectly clear, and when mixed with diluted majaplasma produced a heavy precipitate. The aqueous humour from the other eye and also the blood serum were examined without finding any trace of precipitin. A similar examination made on the following day gave exactly similar results. Later, the blood serum showed the presence of precipitin, and after four days the precipitin content of the serum and the aqueous humour from the right eye became equal. Von Dungern concludes from this experiment that we have the best possible proof that not only a few organs but every type of cell can produce antibodies.

Römer (1901) immunized rabbits against abrin by way of the conjunctiva, and found as a result that the spleen, the bone-marrow and the conjunctival tissue of the eye exposed to abrin contained anti-abrin, while the conjunctiva of the other eye contained none.

Hektoen (1911), however, points out that at the time when Römer made his examinations the blood was also antitoxic, and since the eye treated with abrin had been the seat of several attacks of inflammation, while the other eye had remained in a normal condition, there is a distinct possibility that the anti-abrin in the conjunctiva of the treated eye may have been derived from the blood and lymph.

Wassermann and Citron (1905) estimated the amount of lysin for the *Bacillus typhosus* in the blood serum and in the serum of peritoneal and pleural exudates after single injections of typhoid bacilli, given intravenously, intrapleurally and intraperitoneally. The exudates were produced by aleuronat injections. They found that in certain cases the serum from the pleural and peritoneal exudates of animals, which had been given intrapleural and intraperitoneal injections, produced lysis in higher dilutions than did the blood serum; and they consider that this fact may be explained by assuming the local formation of antibodies by the cells of the pleura and peritoneum.

Hektoen (1911), in an extremely careful study on the question of the local production of lysins, agglutinins and opsonins for rat and goat corpuscles by the tissues of the anterior chamber of the eye and of the pleural and peritoneal cavities of dogs, was unable to find any evidence of such formation. These antibodies never occurred in these situations in higher concentration than in the blood; often the antibody content of the aqueous humor and the pleural and peritoneal exudates was considerably lower than that of the serum; while immune bodies of this type could never be demonstrated in the fluids at the site of inoculation earlier than in the blood stream. Again, there was no difference in the antibody content of the blood serum and pleural exudate, as between those dogs which received the injection of antigen intrapleurally and those which received it intravenously.

Wassermann and Citron (1905) injected killed typhoid bacilli into the ears of rabbits, afterwards ligating the ear for two hours. On the tenth day they determined the amount of lysin in the serum, and then amputated the ears of certain of the rabbits. In one of these animals, the subsequent fall in the antibody content was decidedly more rapid than in the control animals.

Hektoen (1911), in the paper referred to above, describes several experiments made by him in investigating the rôle of the subcutaneous tissues in antibody production. He argues that, if antibodies are produced locally at the site of inoculation, it would be reasonable to expect that subcutaneous injection of antigen into many places simultaneously would give rise to a large production of antibodies. Two dogs were injected with 1 c.c. per kilo weight of a 10 per cent. suspension of foreign corpuscles in ten different places, using goats' cells in one case and rats' in the other. In neither case did the resulting antibody curve exceed, or differ materially from, that which was produced by a single subcutaneous injection of rat or goat blood. Acting on the suggestion of Wright, that massage, after injection of vaccine into a limb, might give rise to a sufficiently marked increase of antibodies in the blood, to prove definitely the local production of these substances, the following experiment was performed. Rat or goat corpuscles were inoculated into the muscles of the foreleg in dogs, and when antibody production was well under way, the injected tissues were subjected to skilled massage for twenty minutes. In no case did the antibody content of the blood, carefully tested at hourly or half-hourly intervals after the massage, show any such decided rise as to indicate any production of antibodies at the site of injection. Finally, the following experiments were undertaken: 1 c.c. of a 10 per cent. suspension of goat corpuscles per kilo weight of animal was injected subcutaneously into the lower part of the right foreleg of each of five dogs. Two days later, the right foreleg was amputated in two of the dogs, and the left in two, while in the case of the fifth dog nothing was done. In all the stumps some suppuration developed, in two cases quite insignificant and in no case sufficient to affect the general health of the animals. The development of the specific hæmolysin ran a practically identical course in the two dogs whose left forelegs were amputated, and in those whose right forelegs were amputated, while all four showed distinctly less antibody development than did the fifth dog which was left undisturbed.

Several workers, including especially Forsmann (1905), have shown that the antibody curve varies somewhat according to the site of injection, and this latter investigator suggests that each tissue may have its own antibody curve. Hektoen (1911), however, points out that this phenomenon may depend simply on differences in the rate and concentration in which the antigen is carried from the places of introduction to the centres where antibodies are produced.

With the exception of von Dungern's experiment, and possibly that of Römer, there is, then, no striking evidence of the production of antibodies by the tissues at the site of inoculation, and these solitary and uncorroborated results can hardly stand against the far greater weight of negative evidence.

BIBLIOGRAPHY.

Von Dungern. "Die Antikorper," Jena, 1903.

Forsmann. *Centralbl. f. Bakt.*, 1 Orig., 1905, xxxviii, p. 463.

Hektoen. *Journ. Infect. Dis.*, 1911, ix, p. 104.

Römer. *Arch. f. Ophthal.*, 1901, lii, p. 72.

Wassermann und Citron. *Zeitschr. f. Hyg. u. Infekt.*, 1905, l, p. 331.

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Section of Psychiatry.

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Observations on the Psychogenetic Psychoses.[1]

By HENRY DEVINE, O.B.E., M.D.

IT sometimes happens that when individuals are faced with a situation of a strongly affective character, instead of making an efficient adaptation to the situation, they retreat from reality and develop psychotic or neurotic symptoms. Such psychoses have been variously grouped under the terms "situation," "emotional," or "psychogenetic" psychoses, because the symptoms are definitely related to and provoked by some particular psychic experience. The situation is usually disagreeable in character, but not necessarily so. Thus cases are reported in which persons who have learned that they have suddenly become enriched have reacted by developing a psychosis, and even in every-day life it is not at all unusual upon the receipt of good news, or when congratulated upon some achievement, for an individual to develop feelings of depression, uneasiness and apprehension. Perhaps the contrast mechanism in such instances is beneficial, as it inhibits undue egotism and elation, and thereby maintains the proper balance of the personality. However that may be, the point to be emphasized is that any emotional experience may upset the mental balance, and that the psychogenetic manifestations which occur range from the quasi-normal reactions of daily life to the gravest psychotic episodes.

Considerable attention has been given to the psychogenetic mental disorders by German writers, and in America Bernard Glueck has produced work of considerable interest, especially in relation to the so-called "prison" psychoses which manifest themselves in prisoners as a result of stress and confinement [1], whilst among French observers the effect of an emotional situation in the production of mental disorder has been carefully studied by Séglas and others [2]. The recognition of this group is important from a purely clinical standpoint, but perhaps it is from the psychological viewpoint that their study is especially of value. The mental mechanism in these cases is often quite clear, the conflict is on the surface as it were, and the significance and purpose of the psychosis is readily understood. By recognizing, therefore, the psychogenetic mechanisms in these more or less transparent cases it helps in some degree to an understanding of those more obscure cases in which the conflict is more hidden, the content of the psychosis less comprehensible, and the psychogenetic factors instrumental in their production less easy to elicit.

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[1] At a meeting of the Section, held December 9, 1919.

As a characteristic example of a psychogenetic psychosis the following case may be cited [3]:—

A young girl was engaged to be married. One day she complained of sickness and malaise to her mother, and she became so poorly that she took to her bed. Examination revealed the presence of an abdominal tumour, and from the history it appeared that she had not menstruated for some time. She confessed that she might be pregnant, and her fiancé was approached upon the subject. One or two days after her lover disappeared, and the girl, after a period of anxiety not unnatural in the circumstances, became insane.

When admitted to the asylum she was in a state of the greatest elation, buoyantly happy, extremely restless, and completely disoriented. Her delusions were all about her marriage, which she believed was about to be celebrated, and she gave invitations to all the nurses to be present at the ceremony, which was to take place at White Lodge, Richmond Park, which she thought she owned. She wished every one to share in her happiness, and she was overflowing with sympathy and pity for her fellow patients, and she regarded herself as a ministering angel come to look after them. The tumour rapidly increased in size, but none of the other signs of pregnancy appeared, and it was found that she was suffering from an ovarian cyst. This was successfully removed, and the patient made a rapid and uninterrupted recovery, both in respect to her physical and mental condition.

The clinical features to be observed in this case are as follows: (1) The reaction of the patient to life had been normal, in a general way, before the outbreak of the psychosis; (2) the illness was directly the result of a painful situation; (3) the delusional content was obviously related to the experience which provoked it; (4) when the stress was removed the patient made a rapid recovery. These four features are characteristic of a psychogenetic psychosis, and thus the case may be regarded as typical. Naturally all cases are not so clear cut, but these features are always more or less in evidence. The mental mechanism in this case is also characteristic. The patient meets with what is to her an insurmountable obstacle in life, and one to which she is unable to make a normal adaptation. Her interest retreats from reality, and she develops a phantasy which transforms the environment, and in which the mechanism of "wish-fulfilment" is evident. The mechanism is one of regression, the patient adapts herself to the situation on an infantile level, and makes her life a fairy story as it were. When the obstacle is removed the interest of the patient flows in higher channels and she again regains contact with reality.

The war has furnished a most fruitful field for the study of psychogenetic mental disorder. It is perhaps true that a few neurasthenic individuals improve under army conditions, no doubt because this particular atmosphere affords, in such instances, a more complete expression of the personal trends than was possible in civil life, yet in general it is more true that military life does not constitute a favourable environment for neuropathic individuals, and the conditions in the Army result in the mental breakdown of a number of individuals who have been able to make a fairly effective adjustment to civil life, which makes less strenuous demands, and only requires more or less routine adaptations.

The fact that the recovery-rate is so much higher in insanity occurring in the Army is no doubt largely due to the fact that a considerable proportion of the cases belong to the group of the psychogenetic psychoses, and they tend to recover upon removal from the stress which has produced the failure in adaptation. If we view the neuroses and functional psychoses as a method of retreat from reality, as a failure of adaptation which serves unconsciously to protect the individual from a painful situation, it is interesting to trace the development of the various mental symptoms from the milder neurasthenic syndromes up to the more severe manifestations as observed in the psychoses. It is often possible to observe the gradual onset of protective nervous symptoms in a patient who has recovered from a war neurosis as the time comes for his discharge from hospital, when he will again have to face reality. Dreams, dizziness, dyspepsia, headaches, tremors and the like begin to develop, all of which indicate a regression of the libido from the work of adaptation to reality, and the re-animation of what Janet describes as inferior functions or modes of adaptation. Three cases will now be described in which mental symptoms followed a traumatic episode. The first is an instance of a severe neurosis which assumed psychotic intensity following an emotional experience.

While in camp, not long after he had been recruited, a soldier witnessed a distressing accident in which a man was killed. He became greatly disturbed, could not eat nor sleep, found himself unable to do any work, and eventually had to be sent to hospital. He was extremely restless, he sat up in bed wringing his hands and rubbing his hair, and continually muttering, and at times shouting to himself. When he was allowed up he spent his time pacing rapidly round the garden, all the while rubbing his hair frantically with both hands in a regular stereotyped fashion. He made a great noise, roaring out continually, "See, see, I ought to have told them, see," and at times muttering to himself in disjointed phrases. When questioned it was found that he could readily pull himself together, and he was able to give a perfectly coherent account of himself. There was a rather curious click of the tongue in speaking, as well as a tic of the neck. He said he had been able to carry on quite well in the Army until he had seen this accident, which had continually haunted and obsessed him since. He now felt himself to be a nervous wreck, he could not bother to keep himself clean, he felt he must be moving all the time, could not concentrate, nor interest himself in anything, nor keep up at all. He said he was sorry to create such a noise and disturbance, but he really could not help it; he had no control over himself, and must give way to the feelings which possessed him. However, he promised to do what he could, and think of other people. His promises were, however, of no avail, and as soon as he returned to his ward his disturbing activities recommenced, if anything with increased intensity.

It soon became apparent that associated with these crises were quite characteristic psychasthenic ruminations, which absorbed his conscious mental life. He explained that the constantly recurring term, "See," meant "The whole situation, the war and myself in it, my being in this game of killing people." He then elaborated the following series of obsessional ideas: "I ought to have told them before I joined up; I didn't understand; they didn't know the sort of person I was. It's the whole situation, I can't understand my being in it. I joined up in a dream. It wasn't real, and I didn't understand. When the man was killed I woke up. This war game means killing people, and everyone ought to live as long as possible. I don't mind being killed; it's being in something that means killing people. I know now I am in a game I can never bear up in. This war game, killing. It's all a mystery to me. I did not know what I was doing when I joined up. I did not realize I was in a war. If I saw a street fight I always walked away. I ought to have felt like this before. I knew I might be killed, but I did not realize I was in the game of killing. I'm all in, I can't do anything. It seems funny I am in a war, I don't understand how I can be in a war. It's quite right that the country is in a war. But it's me, how can I be in the game of killing? I ought to have told them. When I go home I shall be worried because I have been in it. I must try and forget that I have been in this thing which means killing people. When I go home I must try and forget it. Perhaps I shall forget I have been in a war. How can I be in a war? It can't be me in it. I must have been asleep to be in a war now. Why didn't I tell them? I am a person taking part in a war. I don't think I really knew it was a war, I didn't understand. The man was killed. That's being in a war: people are killed. People ought not to be killed. I'm in something in which things like that happen. I'm part of a war which kills people. It's not being killed which bothers me, it's being in something, war, which means killing. I must have been asleep."

After a short time, with reassurance, explanatory conversations and so on, the patient improved and regained his normal mental level.

In this case the history, which for the sake of brevity I will omit, showed that the patient was of a definitely psychasthenic make-up, and he had been accustomed to react to the difficulties of life by means of various nervous symptoms. The deeper conflicts in this case were not elicited, but yet it forms an interesting study of one type of maladaptation to a painful situation. In this instance the retreat from reality did not reach the depth of the other cases I shall refer to, and the symptoms though severe are of the same character as those which occur in a lesser degree in the mental life of most people in lowered mental states. The patient shows clearly what Janet [4] calls the loss of *function du réel* and its substitution by exaggerated and inferior mental operations in the form of indecisions, crises of agitation, defects of attention, obsessions and ruminations. The symptoms appear mechanically when the psychological tension is lowered, when the capacity for adjustment to a new and painful situation fails. The mechanism is one to which the term "regression" may be applied. The interest retreats from reality and flows into more primitive channels with the substitution of rudimentary and instinctive reactions in the place of efficient action in relation to the situation.

The actions of the patient are on an infantile level. They are banal, futile, ridiculous, they accomplish nothing, have no bearing on the situation, they appear unreal, out of proportion, overdrawn, exaggerated. As the patient paces frantically round the garden he looks curiously like a child reacting to some thwarting of interest by means of a "tantrum." His thoughts are on the same level. They relate to the past and bear no useful relation to the present; they do not progress, arrive at a solution; they are ineffectual, obsessing, without real value, and issue in no kind of useful activity. Such ruminations are on the same intellectual level as phantasies, day dreams, lethargies and serve the same biological function. Being on an infantile level these actions and thoughts are easier than an energetic application to reality was to the patient; they are mechanical, they make no demands and require no effort. This elementary reaction is the only method of adaptation of which this patient is capable in the situation he has had to meet.

The next case, a man, aged 24, employed on a farm previous to enlistment, contracted a mild attack of influenza and was sent to hospital. The patient made a good recovery and was not at any time very ill. When the time came for his discharge to duty he accompanied his comrades to the office to receive his kit, and whilst waiting his turn he fell down in a "fit" and had to be put to bed, where he remained unconscious for a time. He soon recovered and it was again decided to discharge him. A similar incident occurred on this occasion, however, and he was therefore transferred to the neurological ward for further observation. When admitted to this department on October 25 he stood in a fixed and rigid position, and when put to bed he remained staring fixedly in front of him, would say nothing when questioned beyond his name, and exhibited very definite cataleptic phenomena. He was, briefly, in a state of stupor. In the evening the patient was more responsive and began to regain contact with his surroundings, though replies were only elicited after much delay. He answered a few questions as to his occupation, country and parentage, but soon became dazed and stupid when asked about recent happenings. The following questions and replies were noted after the above general information had been elicited:—

Q.: "What month is it?" A.: "Don't know."

Q.: "What time of year?" A.: "Winter."

Q.: "What kind of place is this?" A.: "Don't know."

Q.: "How old are you?" A.: "Don't know."

Q.: "When were you born?" A.: "1894."

Q.: "What country is this?" A.: "Don't know."

Q.: "Is this France?" A.: "Don't know."

Q.: "What have you been doing for the last six months?" A.: "Don't know."

Q.: "How long have you been in bed?" A.: "Don't know."

Q.: "How did you get here?" A.: "In a boat."

Q.: "What have you been doing to-day?" A.: "I got up, made my bed, washed and my head got . . . " (became very emotional).

After this, no more could be elicited from him. The above shows a somewhat patchy and inconsistent amnesia and disorientation. The dullness and attitude of refusal were obviously instigated by questions relating to recent events.

October 26: Attitude infantile. Kept saying childishly, "I'm hungry, I want my breakfast," though he has already had it. Later on he cried, and gave as an explanation the fact that he wanted his dinner. He had to be soothed and reassured. Gave silly replies to questions. Thus, asked who the orderly was, replied, "He is a nice man." He obeyed simple requests and showed no resistance except in respect to the recall of memories, but there was a tendency to perversation—e.g., kept his tongue out when asked to show it. Later on in the day he was weeping profusely, and when he came into the office for examination, he lay back in the chair sobbing violently. Asked what he was crying for, he replied, "I don't know what it is. I'm just bothered." I asked him if he had a nice dinner and he said "Yes, nicer than I am" . . . howls . . . "I want a clean, I want to wash." His memory reaction was much the same as the day before. He gave a few replies to general questions, then said he knew nothing, everything round bothered him, he wanted to go home, and asked me to help him to go.

During the next few days he wept frequently, said he wanted his wife, he was frightened, he wanted to do right and did not know how to begin, &c. At times his mind seemed clearer but at others he was dull, dazed and stupid, and he would sit on his bed for hours gazing vacantly in front of him. It was possible to elicit some facts of his previous history and to gain some knowledge of his intellectual status. The latter was poor and the patient showed some degree of congenital mental defect. He had not received much schooling, and he had led a wandering existence, supporting himself by farm work and general labour. On one occasion when a lad he had been treated rather roughly and this may have accentuated his natural timidity. Recently he had married, and his wife was pregnant. Any reference to the circumstances of his present illness provoked an emotional reaction; he would become muddled, nervous, frightened, perplexed and lachrymose. The following conversation is characteristic of his mental attitude.

"Can you remember things." "Yes, sir."

"Why did you come here?" "I had hurtings all over."

"Do you remember coming with the others?" "I do not remember, I got awful sick."

"What has happened in the hospital?" "I don't know, my stomach worried me."

"Did you refuse to go with the others?" "I don't remember, I want to help to fight, I'm worried about my wife, I don't know what's happened."

"Why did you have a fit?" "I'd love to know, I'm bothered, I don't remember all."

"What happened when you went for your clothes?" "I didn't get no clothes. I want to do right, that's all."

"Why did you fall down?" "I just want to do right, you aren't going to bother me, are you?"

"What worries you now?" "I've done everything wrong. I don't know how to tell you. Its these orders"—he picks up a bit of notepaper on which he had written some general orders as to his conduct on guard, &c., and upon which I had often seen him gazing vacantly, "I want to obey orders, I've got 'em wrote down and I've never understood. I don't understand these words and how they are fixed. Have I got them right? I can read but I don't understand, there's words in there I can't pronounce."

It is thus apparent that while the patient was well oriented and was able to furnish a relevant account of his past history, there was yet a marked resistance to the recall of recent events. This was expressed by an attitude of stupidity, an emotional reaction, irrelevant replies, or a frank statement of inability to recall. It is also to be noted in the above replies a reiteration of the desire to do right. This attitude underwent considerable development as is shown in the following characteristic episode. One day I found him sitting on his bed and gazing in a puzzled fashion at his locker. "Come, sit here," he said, "I want help to do right, I just don't know how to begin, I don't know where I am and I want to straighten things up. Here's some money I've got, I don't need it, I want to do right and don't know how, you take it all and keep it." After staring at the money for some time he continued, "I don't know what to do, I want to make a start. I want to help to clean up and put things straight. I'm worried." Looking at his little box —"I want to wash, clean up"—took up a box with cigarette ends in it— "Look at this, it's no use. I want to clear it all up. All the boys want things cleared up. I won't smoke this pipe any more, it makes my mouth dirty. I want to wash. I want to clean up." I took him to the ward sister and he explained his wishes to her in a bewildered fashion, and when he was shown how he could tidy up the ward he became childishly pleased and feverishly occupied himself with "cleaning up."

The patient made a fairly rapid readjustment, though subject at times to periods of perplexity and depression largely as a result of his anxiety in regard to his wife, and he needed frequent help and reassurance.

Much has been written about hysterical "twilight" states of which the second case is an instance. There is nothing in the symptoms of this case which adds much to our knowledge of this condition, but perhaps its chief interest is the fact that it reveals several mechanisms in a rudimentary form and transitory in character, which in other cases are much more prolonged and exhibit more elaborate organization and development. In this case the conflict was produced by the instinctive tendency to self-preservation, the desire to escape from a situation the patient could not face, reinforced by the special circumstances in regard to his wife. In so far as the patient was of inferior intellect and had only imperfectly developed social sentiments, the personality could exert but little inhibition upon his fears in respect to the situation.

The ground was prepared for the definite psychotic episodes by the constant fear that he would be unable to carry out his instructions, the significance of which he was barely able to grasp.

The patient first developed a condition of cataleptic stupor, but the disturbance of consciousness was not very profound and impressionability to things in the environment was retained. With widening of the scope of consciousness the patient became more accessible but exhibited amnesia in respect to recent happenings, and his answers to questions showed the tendency to "by-speaking" described by Ganser. The genuine character of this type of amnesia has often been questioned, but the explanation which is no doubt correct is that the desire of the patient to forget things brings about a condition in which this actually occurs. Combined with this amnesia, at times, were transitory phases of "puerilism" in which the attitude of the patient resembled that of a child of three or four. In many instances this symptom-complex has assumed a highly developed form in the war neuroses.

With the increasing pressure of reality, and as some realization of the situation began to dawn on the patient, another mechanism began to operate in his mind. He developed a mania for tidying up, he felt himself unclean, the ward seemed to him untidy, he wanted to put everything in the right place, and was most anxious to be kind and help people. These symptoms were of course the expression of what may be termed a "guilt" complex. Underneath was a feeling that he had shirked his duty, that he ought not to have broken down, and that he should have accompanied his comrades when they left the hospital. His desires to put things right, to "clean up," were methods of symbolic compensation and remind us of the well-known "hand-washing mania," which has a similar, though often more obscure, origin.

Here again we see a clear instance of the mechanism of regression. The interest, libido or energy could not find an adequate outlet, the patient could not make a proper adaptation to the reinforced and stressful reality of the moment and the interest is withdrawn from the environment altogether. The mental attacks are the final break at the moment of a too severe tension, and they are to be regarded as a primitive method of escape from an intolerable situation in which the patient shuts out reality by ignoring its existence. From a stage of stupor, comparable to an early infantile condition, the patient gradually returns to reality by progressive stages of childishness as previously described. As in all these cases, however, the interest is only able to flow back to higher channels, to get back into contact with reality as it were, when the external situation is changed to one in which the patient is capable of making a normal adjustment.

The third case is that of a soldier who suddenly developed an acute psychosis when his division was ordered to proceed to France. He became extremely excited, refused to leave the camp and made a violent attack on his superior officers. So uncontrollable did he become that it became necessary to restrain him for a whole day. When sent to hospital he was found to be in a dull and confused condition. He said his head felt "funny," and he was only able to express himself in fragmentary phrases. He rambled much about the Red Cross, saying, "It's a good thing for helping people, it means sacrifices, it is what I learnt at home how to do right." He was hallucinated and said he could hear the angels saying to him, "Do this!" and "Don't do that!" He said also he was in communication with his mother and was telling her all about himself. To the recent events in the camp his mind seemed a complete blank. Later on he became quite stuporose for a time, then showed signs of excitement, walked up to a patient and said: "Are you my friend, are you a working-man?" He had to be put into a side-room, shouting loudly, "My country for Jesus Christ," and then walked rapidly about his room, spitting on the walls and acting as if he were engaged in painting them. After a time he quieted down but two other episodes occurred before his recovery, each being instigated by the sight of the Red Cross emblem on the uniform of a nurse. This particular stimulus invariably provoked a condition of extreme terror. The patient became violent, made wild rushes out of his room, and tried to hide himself under the bed. He was controlled with considerable difficulty.

Within a few days the patient made a good adjustment and regained his normal mental level. He was quiet and reserved in character, somewhat shy and seclusive, but made himself liked by the orderlies and nurses and was anxious to be useful. He liked to remain in the ward, where he felt at home and was understood. After a time when his confidence had been gained it became possible to learn his story. He was of foreign birth and had been brought up by his mother in X., a country against which he was now fighting. He had helped his mother look after her little farm but had emigrated ten years before, joining his brother who had preceded him in this course. He felt that his prospects were poor in X., and he realized that without a certain amount of capital he would have but little opportunity to improve his position in his own country. He had hoped that in time he would be able to save enough to make a home for his mother in his new country. Hitherto he had not been able to realize this wish, but he had sent his mother money and comforts from time to time and had kept up a regular correspondence with her. When his native country went to war his mother wrote and described how her horse and cart had been taken by the military, and expressed the view that he ought to return and help his motherland. The patient did not feel he was called upon to act upon this suggestion. He told me in his simple fashion that though he still loved his own country, yet he felt she was in the wrong, that she was fighting against those things he had learned to value in his adopted country of which he now felt himself to be a citizen. All he wanted was for his mother to share with him the liberty he had experienced since he emigrated, and for his own country to reach a similar state of enlightenment. He was a Catholic and had kept up his religion, but it seemed different in a free country where there was no compulsion to follow any particular creed, nor was he oppressed if he showed any laxity in religious practice.

In the course of time the patient was drafted into the Army, and he came over to England in a troopship. He felt quite happy to fight for the country to which he felt he owed so much and experienced no anxiety until he reached England where he met with a somewhat disquieting experience. He was passing the camp church where Mass was being celebrated and he caught sight of the priest making the sign of the Cross in the fashion peculiar to the country of his birth. This incident made him extremely uneasy and he felt disturbed and unhappy. He was unable to shake off these disagreeable impressions and he found that as he passed through England in the train that the countryside reminded him strangely of his native land. Indeed at times he actually had the illusion that he was there in reality. He began to get increasingly out of contact with his environment and he felt that people were looking queerly at him and regarding him with suspicion. He was unable to throw much light on his delirious episodes but said that at times he thought he was dead and that his mother was with him.

The patient made quite satisfactory progress and responded to treatment.

In this case we see a young man who had emigrated from his own country some years before and had learned the ways and acquired the sentiments of his adopted country. When he came to England a conflict arose between what may be termed the "motherland" complex and the war situation in relation to himself. This complex was stirred into activity by a chance religious symbol obviously associated with elements in his complex. The complex began to invade his personal consciousness, producing vague anxiety, illusory perceptions and vague delusions of reference, obviously a projection of his own fears to other people. At the traumatic moment when he was called to France the complex surged into the personality, complete dissociation occurred and he retreated into an infantile phantasy, in which he was controlled by his mother and supernatural beings in whom he had believed in his early days. The mechanism is again one of regression. The mechanism is simple enough, but the question naturally arises as to why the patient could only solve the conflict by means of a psychosis. Exactly the same subjective and objective situation has occurred in numerous instances during the war, and a perfectly efficient adjustment has been made. The situation and the complex were in obvious ways capable of co-ordination with the retention of a perfect grasp of reality. The explanation is, I think, to be found in the character traits of the patient. He was of a simple type and had always been unconsciously dominated by the motherland complex. It had inhibited his mental development and had been the undercurrent, the impulse of his life, which had impelled it along a certain narrow groove and had prevented any wide social excursion. The patient had always been controlled, as it were, by his childish influences. All his actions had always been determined by what his mother would think, and his one anxiety had been that she might know that he had not forgotten her early admonitions. Thus, I think, it is permissible to suggest a certain abnormal arrest or fixation of interest in this case which rendered him shy and seclusive, dependent and childish, and dependent for his comfort on a friendly atmosphere. The complex dominated the personality rather than the personality the complex. As long as the patient was in his adopted country and he could communicate with his home his dominating complex found adequate outlet and expression and could be perfectly co-ordinated with his new sentiments and ideas. There was no conflict. As soon, however, as a new situation arose, one which thwarted his unconscious life motive, adaptation failed. This question of fixation of libido, interest or psychic energy is of course one of considerable importance in relation to the psychoses.

In suggesting a few comments on this group of cases it is first to be noted that all these psychotic episodes were provoked by definite situations of a strongly affective character, and the psychoses were thus definite reactions of the individual to particular experiences. The trauma is psychic and there is here no question of any physical traumatic factor which may complicate cases from the front line. In the second place the cases recovered when the painful situation was removed and the psychic irritant ceased to be in operation. Whilst, however, these two features are common to the whole group the form which the regression takes differs in each case, and we have a widely differing clinical picture according to the reacting type of the individual—viz., a frank "wish-fulfilment" delirium, a severe psychasthenic crisis, an hysterical twilight state, and an acute hallucinatory confusion. This point is especially important in so far as many cases belonging to the category of the psychogenetic psychoses, in which the prognosis is good, present a clinical picture closely allied to the acute phases of dementia præcox especially of the acute hallucinatory or katatonic type. It has frequently been said that many war psychoses have been diagnosed as dementia præcox, and yet they have made a good recovery, with result that discredit has been thrown on the acumen of the clinician, and criticism has even been directed to the conception of dementia præcox itself. Unfortunately the fact of dementia præcox is only too true, as every asylum ward abundantly testifies, but there is no doubt that it is often an extremely difficult matter in the acute stages of a psychosis to diagnose a deteriorating psychosis, or to predict the degree to which deterioration will occur. I think we may state quite definitely that reliance in making a diagnosis must not be based on symptoms, but that each case must be regarded from all angles and points of view, and especially in the light of these cases must we look for some traumatic situation which may have been instrumental in the production of the psychosis and removal of which may effect a cure. The stress of environmental conditions is of course intuitively recognized by psychiatrists, and the fact that many of our cases improve rapidly when removed from their home to a mental hospital may, in some instances, be explained on the grounds that the home atmosphere has a traumatic significance. The trauma may not be so immediately in the foreground as in our cases, but still it may be there and should be sought for before venturing on a prognosis. Glueck quotes Risch in this connexion as follows: "Risch speaks of a certain condition in young girls, which he was wont to consider as katatonia, because of their symptomatology, but the later course of which convinced him beyond a doubt that he was dealing with purely psychogenetic disorders. These patients were frequently taken home from school against the advice of the doctor, and promptly recovered their normal health upon reaching home. Hysterical features were wanting in those conditions, and in all of them he could find some sort of mental anxiety or shock concerning some love affair as of etiologic importance. The anamnesis frequently revealed that the withdrawal of a betrothal, or any other unpleasant termination of a love affair—sexual traumata, &c.—stood in immediate relation to the psychosis. Upon experiences of this sort he bases the belief that in many psychoses of young girls, which are being so readily diagnosed as dementia præcox, a much more correct prognosis could be rendered if their true nature were recognized through a more careful attention to the psychogenetic factor.

These observations are not, of course, meant to imply that a differential diagnosis is not in many cases possible between a psychogenetic psychosis of the hallucinatory type and dementia præcox. Apart from other considerations one clinical point which often serves to differentiate the two conditions is the impressionability to influences in the environment in cases of the psychogenetic group. As an instance may be cited the reaction of the patient to the Red Cross emblem. Colonel Hurst showed me a case some time since with extreme inaccessibility and negativism, but he exhibited a very strong terror reaction directly the word "bomb" was mentioned. I do not know the subsequent history, but I thought then that this impressionability to a quite understandable external stimulus, justified a favourable prognosis and placed the case in the category of the psychogenetic psychoses. The symptom no doubt depends on the depth of regression and the extent to which subjective interests predominate.

It is not, however, merely from a survey of the symptoms and a study of the precipitating factors of a psychosis that we are able to estimate its meaning and future course; each case must be regarded from the wider view of personality or the soil on which the symptoms develop. One of the chief problems of psychiatry is to determine the defects of personality which result in abnormal reactions in conditions of stress. If we study the cases completely which have been described, review their previous history, and estimate their mental make-up and character traits, it is found that all of them had exhibited evidence of inadequacy, so that it is justifiable to come to the conclusion that the actual psychosis is no more than an episode in the life of an abnormal person, and that this particular reaction to stress was, in the circumstances, the only one of which each patient was capable. In collecting, then, this group of cases together there is no attempt to describe in a clear-cut fashion a form of mental disease or entity, but rather to indicate how certain individuals of defective make-up react to situations which they are called upon to meet. This view cannot here be developed at any length, but I would suggest that when we speak of dementia præcox, paranoia, and so on, we do not so much speak of a disease or morbid entity but rather the tendencies of certain, in a wide sense, abnormal individuals to develop a certain attitude to life which we call mental disorder. To understand all these conditions I think it will be necessary to study much more intensively the personality of our patients and the influences which have been brought to bear upon them from early days. In gathering together certain cases under the heading of "psychogenetic psychoses" we merely emphasize the fact that the external stress is of a particularly severe and obvious nature. I would suggest that the term is in a wider sense equally applicable to dementia præcox and other psychoses, but that in these cases the defect of personality is more fundamental and the individuals are in consequence unable to make an efficient reaction to the demands of everyday life. A stress is not merely a stress because of its intrinsic difficulty but because of its personal significance. Thus a quite ordinary occurrence may have a traumatic significance to certain individuals, as the following cases show :—

A single lady, aged 43, lost her mother. She immediately became very worried and peculiar, and she began to develop delusions to the effect that she had no money and that her brothers were ruined. The condition became rapidly worse; delusions of negation appeared, and she thought she was dead and all her relatives, and eventually that nothing existed at all. For some months now she has been entirely inaccessible, mute and resistive, but given to severe onanistic tendencies. It is only when the circumstances of the patient and her personality are taken into consideration that it becomes apparent why the death of her aged mother should be endowed with such traumatic significance. All her brothers had gone out into the world but she had devoted herself with passionate zeal to the care of her mother. Her whole libido or interest was almost exclusively devoted to this maternal complex, and at her mother's death it was not free to energize the personality for the new adaptations which were demanded of her. The delusions which she expressed, that she was dead and so on, were in a symbolic sense true—for her nothing did exist, her world of reality had departed. The libido was arrested, fixed on the mother, and she retired from real life into phantasy. The incident had a personal traumatic significance.

A woman, aged 22, married, and a few months after her husband, to whom she was greatly devoted, died. A child was born after his death. After a time she married happily again, but she always kept the memory of her former husband alive by excessive devotion to her child and preoccupations with his photograph, letters and the like. Seven years after her second marriage she became pregnant and as her time approached she became fussy and cantankerous. She wanted everything carried out in exactly the same way as had been done at her first confinement, and she became cross and irritable when it was not possible to do so in accordance with her desires, and she began to think she was ill-used and neglected. After the birth of her child she became actually insane, the content of her delirium relating to her past life with her first husband, conversations with him and a journey to Italy which he had promised her.

Here again is seen a partial fixation of the libido on a complex in relation to the first husband of the patient, a complex which found adequate expression until her second pregnancy which made new demands on her interest which she could not meet owing to its antagonism to her predominant complex. As in the other cases she meets the difficulty by regressing back into phantasies of her previous marriage. The childbirth, an ordinary physiological occurrence, is to this woman a severe psychic trauma.

Furthermore, in adolescent cases of insanity we find our cases developing dementia præcox without any ascertainable external stress whatever. Is there not in these cases a still more fundamental defect of the personality, an arrest or fixation of the libido, which inhibits the patients from making an energetic adaptation to the new demands of life which adolescence implies? In a case of paranoid dementia præcox which I submitted to this Section on another occasion [5], the analysis revealed most clearly the fixation of the libido at an infantile level which rendered the patient incapable of adapting himself to the ordinary routine situations of life, and led to the development of a fantastic delusional scheme which was his method of evading the claims of real life.

In the light of these observations then, I would suggest that we pay much more attention to the personalities on which psychoses are engrafted and of which perhaps they are the inevitable outcome. What is the fundamental difference, what is the underlying defect which makes one person react to life as a paranoic, another as a precocious dement, and another as a psychasthenic? Can we detect, modify, or prevent these tendencies to morbid reaction? Are they the result of faulty habits originating in early life, or are they the inevitable outcome of heredity? All these questions require an answer, and though much has been done by modern methods of investigation to illuminate these problems yet they still suggest a wide and important field of inquiry.

There is at present, as a result of experiences gained by the war, an attitude of considerable optimism in regard to the treatment and cure of the neuroses and psychoses. A high recovery rate has been the rule in the subjects of mental disorder in military hospitals, and many of the neuroses have responded in a gratifying way to treatment. I think it must be frankly recognized that these encouraging results are in a measure due to the nature of the clinical material which has been under treatment. Many of these mental illnesses have occurred in individuals who have been quite capable of making an efficient adjustment to the demands of ordinary civil life, but have succumbed to the quite abnormal stresses of military service. They therefore belong to the category of the "psychogenetic psychoses" which have been the subject of discussion in this paper, and when the situation which has produced the nervous and mental symptoms has been removed, readjustment has readily taken place.

It cannot be said that those of us who have been accustomed to deal with civil psychoses have been so successful. To some extent, perhaps this is due to conditions which it is hoped will soon be remedied, but it must also be recognized that we have, in the main, been dealing with cases in which the mental symptoms are the expression of a much more serious defect of personality than is the case with those of the psychogenetic group. This is so equally in the case of most civil neuroses in which the illness has much deeper roots than in the war cases. It is not only a matter of altering the law and improving the conditions of treatment in insanity, but we need a thorough intensive study from every standpoint of these cases in order to arrive at a solution of the problems which they present. From the point of view of personality in relation to mental disorder, not only would it seem necessary to get our cases early, but I think we shall perhaps have to extend our studies to the personality traits before the outbreak of the actual psychosis, in the school, nursery and even cradle before we shall be in a position to understand the development of many forms of mental disorder.

REFERENCES.

[1] GLUECK, B. "A Contribution to the Study of Psychogenesis in the Psychoses," *Amer. Journ. of Insanity*, 1911-12, lxviii, p. 368.

[2] SÉGLAS, J., et BARAT, L. "Le Rôle de l'Émotion dans l'Étiologie des Maladies mentales," *Journ. de Psychol.*, 1913, x, p 81.

[3] DEVINE, H. "The Significance of some Confusional States," *Brit. Med. Journ.*, 1911, ii, p. 747.

[4] JANET, P. "Les Névroses," 1909.

[5] DEVINE, H. "The Biological Significance of Delusions," *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Psych.), p. 194.

Section of Psychiatry.

President—Dr. WILLIAM McDOUGALL, F.R.S.

The Nature of Epilepsy.[1]

By E. W. SCRIPTURE, M.D.

A PECULIARITY of speech observed in the study of epileptics some years ago has found no satisfying explanation in any of the usual theories of epilepsy. Quite recently a new theory has been proposed with which the speech observations accord completely.

In a study of inscriptions of speech in various nerve diseases records of thirteen cases of epilepsy were made and analysed. The results were presented before the New York Academy of Medicine. At a later date the work was continued at Claybury, at the National Hospital in Queen Square, at the West End Hospital for Nervous Diseases and at the Maida Vale Hospital.

The inscriptions are made by speaking into a wide tube ending in a flexible membrane. The vibrations and puffs of air pass down the tube and move the membrane. These movements are enlarged by a light lever and recorded on a surface of smoked paper on a revolving drum. The record sheet is afterwards removed and varnished. To the eye the inscriptions by epileptics do not differ from normal inscriptions; it is only when measurements are made by a micrometer microscope that a peculiarity appears.

The vowels and vibrating consonants appear in the inscriptions as series of fine waves. The length of each wave is measured and the results are plotted on cross-section paper. A long wave is the registration of a low tone of the voice and a short wave of a high tone. A line drawn through the series of dots thus gives the rise and fall of the voice, or the melody of the words spoken. It is termed the "melody plot."

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[1] At a meeting of the Section, held January 13, 1920.

The melody plots for normal speech show that the voice is continually rising and falling; it is never steady on the same pitch for an instant. This is quite independent of the general melody of the phrase; whether the person speaks with rising or falling inflection, or with great or little modulation of the general melody, he always shows this finer fluctuation or flexibility of melody.

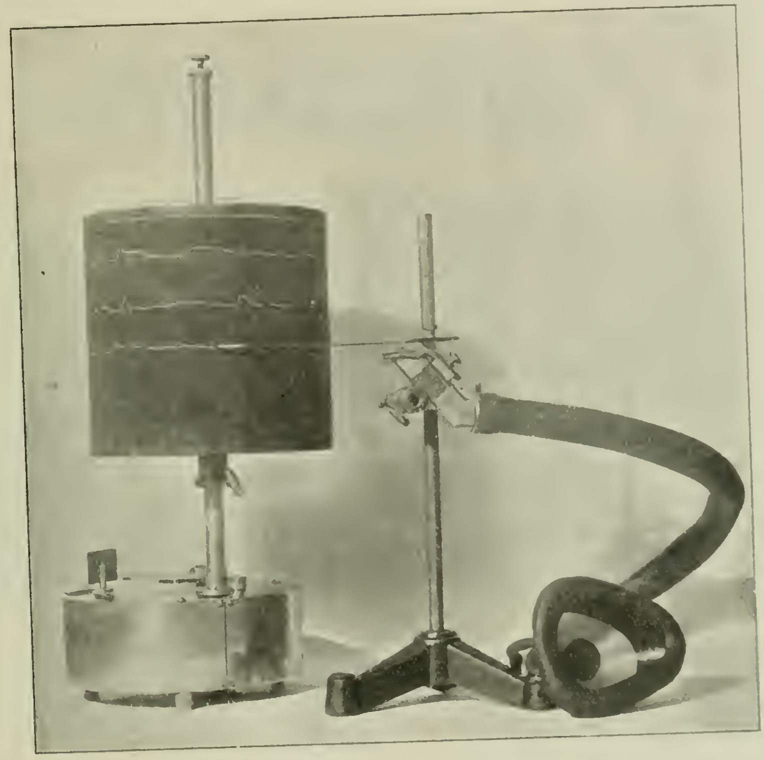


FIG. 1.

Apparatus for making speech inscriptions. The waves and puffs of air from the mouth pass along the wide tube to a flexible membrane whose movements are enlarged and registered on a moving surface.

The melody plots for epileptic speech, however, show a diminution or an absence of the finer fluctuations. The melody lacks flexibility.

To the ear the speech sounds expressionless or wooden. With little difficulty the ear can be trained to detect this peculiarity. I have been taken through the wards of a large children's hospital and have been able to tell whether or not I was in an epileptic ward by the way in which the children said "Good morning." During the war many men came to me with stories of fits in order to secure certificates that would get them exemption from service. More than one had been carefully trained to tell a story that was typically epileptic. Although relatives and witnesses might be brought, my final judgment on the genuineness of the case was based largely and sometimes entirely on the melody of the person's speech. If the patient did not have the typical epileptic inflexibility, I could not bring myself to believe him to be a genuine case. In such cases this sign has a special value. In the first place the patient knows nothing about it. In the next place this peculiarity cannot be imitated voluntarily. Apart from the fact that years of attempts have not enabled me to imitate it, the very nature of the sign as an expression of the epileptic temperament—as will be explained in a moment—makes it impossible to produce it artificially.

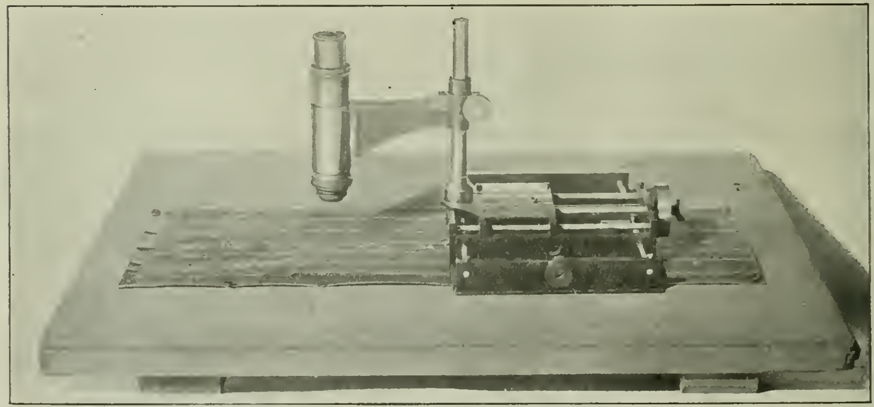
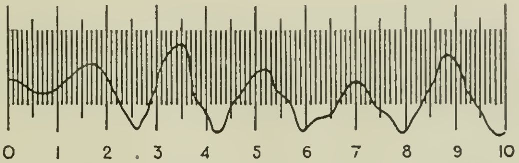


FIG. 2.

Micrometer microscope for measuring speech inscriptions. The record sheet is removed from the drum, varnished and fixed on a board. The travelling microscope is placed upon it. Measurements are made with an ocular micrometer or by moving the microscope by a micrometer screw.

Inscriptions of speech have been made for various nervous and mental diseases such as the spastic conditions, general paralysis, hysteria, melancholia, mania, &c. In the conditions so far studied there occur either no changes from the normal melody or else changes unlike those of epilepsy. Hysteria, for example, is characterized by exactly the opposite condition of over-flexibility. For purposes of differential diagnosis this inflexibility of epileptic speech is perhaps more to be relied on than an actual fit. An hysterical fit often so closely resembles an epileptic fit that the most careful observer cannot declare it to be non-epileptic until he has a most intimate knowledge of the person's life.



Readings	18	35	52	70	88	scale units
Length	18	17	17	18	18	scale units
Duration	0.0088	0.0083	0.0083	0.0088	0.0088	sec.
Frequency	113	120	120	113	113	

Fig. 3.

Waves of an inscription seen through the microscope with an ocular micrometer. The positions of the summits of the waves are read off on the micrometer scale. Since the speed of the drum is known, the readings can be turned into time; the duration of each vibration is then calculated. The frequency, or number of vibrations per second, is found from the duration.

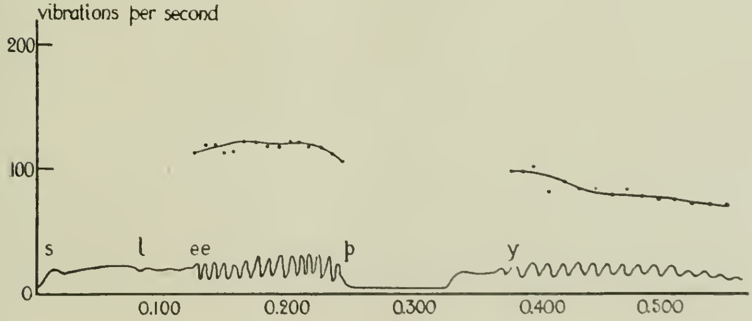


Fig. 4.

Melody plot of an inscription of the word "sleepy." The original inscription is supposed to be laid along the horizontal axis. Above each wave a dot is placed corresponding to its frequency. The rise and fall of a line drawn through the dots indicates how the voice changes in pitch.

This peculiar inflexibility of melody may throw some light on the nature of epilepsy. Inflexibility, apart from the rigidity of monotonous speech, does not occur in any organic condition yet studied. On the other hand variation in melody is one of the ways in which a normal person expresses a mental attitude. Inscriptions of the word "oh" spoken with various attitudes of admiration, astonishment, doubt, &c., differ only in the melody plots. The inflexibility, or woodenness, of the epileptic melody may perhaps be the expression of some peculiarity of the epileptic character. Epilepsy itself may possibly be a disease of character, or mental make-up.

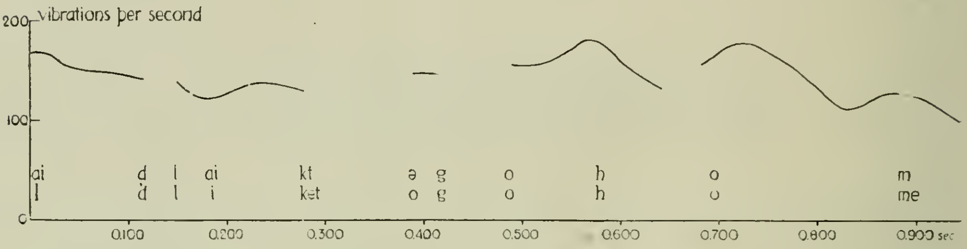


FIG. 5.

Melody plot of "I'd like to go home" by a normal voice. The voice continually rises and falls in small fluctuations. Whatever may be the general course of the melody in words spoken by a normal voice, these minor fluctuations are never absent.

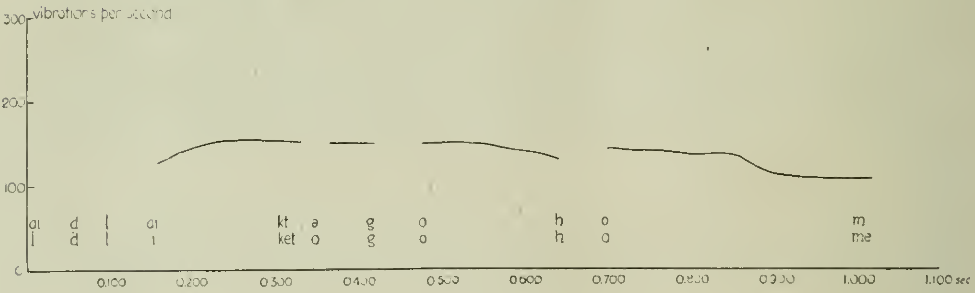


FIG. 6.

Melody plot of "I'd like to go home" by an epileptic. The small fluctuations in melody are absent. This loss of the small fluctuations is not found in any other abnormal conditions except where monotony is present, as in spastic conditions. Epileptic speech, though it may not show the wide range of normal speech, is not monotonous.

In recent years there has been a tendency to study the make-up of the epileptic's mind. According to L. Pierce Clark[1] there is substantial agreement that there is a more or less constant defect in all epileptics, and that this defect consists in ego-centricity, with a highly developed sensitiveness. Clark's careful studies of the history and mental constitution of a large number of epileptics leads him to his first conclusion that this temperament of extreme hyper-sensitiveness and egotism makes the person incapable of proper social adaptation. Clark's next conclusion is that the very essence of the epileptic character is an emotional attitude of a stubborn, unyielding demand that the environment shall conform precisely to the person's wishes. Clark's third thesis is that the epileptic seizure is a reaction to the strains of an intolerable environment by a lapse into unconsciousness. We might say that when the epileptic's mind finds the demands on it too great, it puts out the sign "not at home." We might add that the difference between epilepsy and hysteria arises from the fact that the epileptic mind maintains an irreconcilable attitude towards the situation, whereas the hysterical mind effects a compromise. Clark asserts that the actual or potential epileptic child shows bad adaptive tendencies, even in the first adjustments called for. Sucking, biting and chewing are often imperfectly performed. There is often a lack of smoothness in movements of the tongue and hands. Such children are often incoordinate, and slow in learning to sit, creep and walk. Such facts point to some fundamental biological inferiority in the mechanism of adaptation as the basis of epilepsy.

This new view of epilepsy is as remarkable as it is revolutionary. According to it, epilepsy is a disease of character that may perhaps be modifiable or curable, or even preventable. According to this theory the inflexibility of the epileptic melody would be an expression of the epileptic's attitude towards his environment. As speech is the most direct form of expression, and is the one most immediately concerned with the environment, it is natural to expect the epileptic character to reveal itself just in this way. To the clinical facts supporting this theory there may be added the laboratory fact of the speech peculiarity.

It gives me great pleasure to use this opportunity to express my obligations to Dr. Clark himself, with whom I was associated for several years, and to Sir Frederick Mott, Sir Robert Armstrong-Jones, the late Dr. F. E. Batten, Dr. A. Feiling, and to the hospitals mentioned above for facilities and aid in the speech investigations.

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[1] "Clinical Studies of Epilepsy," State Hospitals Press, Utica, N.Y., U.S.A.

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Section of Psychiatry.

President—Dr. William McDougall, F.R.S.

Studies in the Pathology of Dementia Præcox.[1]

By Sir F. W. Mott, K.B.E., M.D., F.R.S., LL.D.Edin., F.R.C.P.

Introduction.

For some years past I have been interested in the pathology of dementia præcox. First I investigated the reproductive organs, and I have already published in recent numbers of the *British Medical Journal* a paper on "The Investigation of the Testes in 100 Cases of Deaths in Hospitals and Asylums at all Ages from Birth to 80 Years and over." In this paper I have shown that in a large proportion of the cases of dementia præcox there is a complete arrest of spermatogenesis, and in all cases a more or less regressive atrophy is shown, the degree of change as a general rule depending upon the age at which the mental symptoms were first manifested and the time elapsing between the onset and death (*vide* Plate VI, figs. 1 and 2). In some cases in which the clinical history showed that mental symptoms had occurred in the prepuberal or puberal periods spermatogenesis may never have occurred. Now, in spermatogenesis we have a most active nuclear proliferation going on continually, and the histological evidence shows that the nucleic acid, which is the principal substance of the nuclear matter, is formed from a phosphorized lipoid contained especially in the cells of Sertoli.

The head of the spermatozoon consists of a protamin nucleinate and the tails, which are formed in these nurse cells of Sertoli, consist almost entirely of lecithin and cholesterol.

I will not go into further details on this point, but I should like to point out that where active spermatogenesis is going on there we find abundance of the lipoid granules; the same regions which show the existence of these lipoid granules also show the oxidase reaction; by this I mean that these granules play an important part in the chemical processes connected with the function of spermatogenesis. The oxygen on the surface of the lipoid granules is molecular oxygen, but in order to become nascent, chemically active oxygen, the molecule of O_2 must be converted into atomic oxygen, and this is effected by the catalytic action of the phosphorus and iron contained in the nuclei of the spermatic cells. A continual process of decomposition and recomposition, in which oxygen plays an important part in building up the fresh nuclear matter, takes place.

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[1] At a meeting of the Section, held November 11, 1919.

Nucleic acid which forms the main constituent of the heads of the spermatozoa, as was shown by Miescher and Kossel's experiments, contains a large amount of organic phosphorus, iron and calcium. All nuclei contain these three important elements in addition to the elements of a proteid. The arrest of spermatogenesis may then be due either to a failure of one or more of these elements in the circulating blood or to an inherent biochemical lack of durability of the germ plasm.

The reason why I have given this short account of the failure of nuclear matter in the reproductive organs in dementia præcox (for it occurs in the ovaries as well as in the testes) is that the essential histological morbid change in the nervous elements of dementia præcox, as Nissl and Alzheimer pointed out long ago, is one of nuclear decay.

Nature is unmindful of the individual, mindful only of the species, for the body is but a vehicle for the germ plasm. During fœtal life internal secretion of the sex determinant cells is active in establishing sex characters; after birth till puberty all the productive vital energy is concentrated on the somatic cells to build up a body capable of fulfilling nature's supreme biological end—reproduction. I have shown that the interstitial cells forming a well-marked gland in the testes at birth in a few months shrink and become quiescent functionally; the spermatic tubules at three months have grown to twice the size of those at birth and no further change takes place till puberty, when the interstitial cells reappear and are filled with lipoid substance and active spermatogenesis begins. Simultaneously with this concentration of productive energy on reproduction, the sexual internal secretion passes into the blood and produces the secondary sexual bodily characters: at the same time it effects a complete mental revolution. At first a vague ill-defined longing occurs, and this develops sooner or later into an all-prevailing desire arousing new sentiments, passions, and behaviour, presenting many biological characteristics peculiar to each sex.

The critical periods of life as regards mental disease are adolescence and the climacteric periods of life when the sexual function matures and wanes. This affords *a priori* evidence of the important relation between the sexual functions and disease of the mind. This question naturally arises: Is the regressive atrophy of the reproductive organs the cause of the mental changes? (*a*) By its disturbing influence on the whole endocrine system of glands; (*b*) by the suppression of the normal sexual impulse, *libido*, of attraction to the opposite sex; (*c*) or is it a concomitant evidence of an inherent lack of vital energy germinal in origin? I incline to the last explanation as being the most probable although I consider the other two may in certain cases act as important contributing factors for the following reasons:—

(1) There is a general lowered vital reaction of the tissues of the body.

(2) The morbid changes are most marked in the cortex cerebri and primarily and especially affect the nuclei of the neurones.

(3) A number of the cases of dementia præcox show prepuberal clinical signs with history of arrest of development of mind which can be explained by the arrest and failure of development of cortical neurones: and at puberty when the stress of productive energy of the reproductive organs occurs nuclear neuronic decay sets in. This decay is probably owing to a germinal inborn lack of durability, for there is simultaneously a progressive failure of nuclear proliferation in the organs of reproduction.

At what period in early life this nuclear decay of the brain and of the reproductive organs begins it is difficult to say in individual cases; but I am of the opinion, from histological observations, that it may begin in some cases before puberty—even long before puberty. Kräpelin points to the fact that in many cases clinical symptoms occur in pre-puberal life, so that it is legitimate to conclude that there are a number of cases which might be termed dementia præcossissima.

Having given this brief introduction it is necessary now to lay down certain physiological and anatomical premises.

THE OXIDASE REACTION.

If a portion of brain or a section of spinal cord be stained for the oxidase reaction the grey matter is coloured blue, the white matter is unstained.

Marinesco has demonstrated that histological examination of the sections shows that the cytoplasm of the ganglion cells, as well as their protoplasmic prolongations, are studded all over with fine blue-stained granules. Neither the nucleus nor the axis cylinder process contains any trace of granules. This agrees with the observations made by this author and myself in respect to the absence of refractile granules in the nucleus and axon when a living nerve cell is examined with the ultramicroscope. Marinesco found these granules in the cells of the choroid plexus but none in the neuroglia cells. Marinesco remarks it is probable that these oxidase granules belong to the neurone terminals which constitute the synapse and establish connexions between the different neurones.

> "It is especially in the plexiform layers, as is the case for the so-called granule layer of the cerebellum and of the brain, that these oxidase granules are very numerous."

These facts clearly indicate that oxidation processes take place in the grey matter and that the intercalary cells, Type II Golgi (granules), which form the physiological link in systems of neurones of the first type, play an important part in the chemico-physical processes of the synapses.

Now, if oxidation processes which occur in the grey matter are essential for all nervous activity (including psychical), and if it be that molecular oxygen of the oxidase granules must be converted into free atomic oxygen, to ensure neuronic function, it follows that deprivation of oxygen carried in the circulating blood will cause suspension of function. This is the case, for Mosso has shown, in a patient of his who had been trephined, that unconsciousness invariably occurred six seconds after pulsation in the brain had been caused to cease by compression of both carotid arteries.

It is calculated that there is six times as much blood in the grey matter as in the white matter. It was asserted by Leonard Hill that the oxygen content of the blood leaving the brain was as great as that of the blood entering. Bayliss gives reasons why this conclusion is not valid. Moreover, it must be remembered that the amount of grey matter to white matter in the brain is relatively small, and we know that no active oxidation processes take place in the white matter. This fact also disposes of the argument that Battelli's observations show that relatively to other organs in the body the peroxidase reaction of the brain is feeble.

It may be concluded therefore that oxidation processes are essential for neural function and that oxygen for cerebral activity is continually being used up and replenished by the blood. I have shown that there is no free oxygen in the cerebro-spinal fluid, but there is as much CO_2 free and combined as in the lymph. If the neurones are bathed by the cerebro-spinal fluid the sugar in the fluid may be a source of energy for the nervous system and be continuously undergoing a process of oxidation.

Now to continue the argument we may assume the following premises:—

All psychical processes are subordinate to physiological processes, that is neural activity.

All neural activity is dependent upon oxidation processes. These oxidation processes occur in the grey matter.

Oxidation processes are dependent upon the liberation of atomic oxygen from oxidase granules.

The oxidase granules are found on the body of the cell and the dendritic processes. They are especially abundant in the granule layers of the brain and the cerebellum. These granules are the intercalary neurones, second type of Golgi, and where they form definite layers they are interposed between radial fibres and the dendrites of neurones of the first type of Golgi forming a synapsis.

Granted Marinesco's observation, that in the granular layer the oxidase granules are especially abundant, it will be here especially that the atomic oxygen will be liberated under the influence of a catalase. Now the intercalary neurones have relatively only a small amount of cytoplasm compared to the size of the nucleus.

If the iron or phosphorus or both of the nucleus acts as a catalyser, as there is reason to believe, it may be hypothecated that when a stimulus arrives at the layer of intercalary neurones a catalase is liberated from the abundant nuclear substance; this acts upon the molecular oxygen attached to the oxidase granules, converting it into free atomic oxygen whereby physical and chemical changes occur, which either result in a physiological junction of the processes of the intercalary neurones by amœboid movement and multiple contacts with the processes of the first type of neurones, or a process of combustion occurs whereby the sugar is converted into energy which serves as a source of stimulus to the next system of neurones.

Whatever it be, it is significant that these intercalary neurones should exist in well-defined layers and in such abundance in cortical situations where radiating and association tracts of fibres are ending—e.g., the calcarine region where there is a double layer of granules.

Again the great abundance of granules in the cerebellum is very significant for the following reasons:—

The cerebellum is an organ of uniform structure; it is present in all mammals. In fact, in fast swimming fishes and mammals, e.g., seals and sea-lions, it is very large; it is very large in all fast flying birds, especially birds of prey. This biological fact, together with the results of experiments on animals and clinico-anatomical observations in man, show that this portion of the brain is an organ of reinforcement of muscular action, and continuous tonic contraction of the muscles depends upon its functional activity. Under the influence of stimuli from the skin, especially the soles of the feet and all the structures of locomotion, the eyes and particularly the semicircular canals, a continuous discharge of neural energy along the rubro-spinal and vestibulo-spinal tracts takes place, reinforcing with varying degrees of intensity as required, the discharge from the spinal motor neurones to the muscles. It has been shown by Luciani that removal of the cerebellum in animals causes asthenia, astasia and ataxia. The layer of granules which form such a striking feature of the cerebellum may be assumed to provide an abundance of catalase for the conversion of the molecular oxygen into atomic oxygen and thus effect a continuous flow of neural energy. These and other considerations show that the oxidation processes necessary for neural activity take place at the junction of the intercalary neurones with the neurones of the first type. It is here that delay occurs in the transmission of a stimulus along a system of neurones. It may be assumed that either the chemical changes connected with oxidation are transformed into a physical stimulus which travels along the next neuron of the first type, or the oxidation processes cause either an alteration of the surface tension or an amœboid movement whereby a physiological junction is effected at the synapse so that the original impulse can be transmitted. The extent, character, and intensity of the stimulus and its motor reaction are largely dependent upon the biological quality of the stimulus, for groups and systems of neurones are attuned by an instinctive memory or bio-rhythm to react to a biological stimulus with a specific rhythm with maximum intensity, but owing to the fact that a system of neurones with a special function is attuned to a specific biorhythm, a stimulus of any kind will give rise to a specific effect. This fact is proved by a simple experiment. A faradic current applied to the skin causes an uncomfortable vibration sensation; applied to the tongue, a taste sensation; to the eyeball, a sensation of light; to the drum of the ear, an ill-defined auditory sensation. So that owing to this inherent specific bio-rhythm of the special sense neurones, the same stimulus produces the specific effect. It may be presumed that a common mode of motion has been transformed to a special mode of motion at the central receptor.

Oxidation processes do not take place in nerve, and it is incapable of fatigue. The nervous impulse as it travels along the axon is a physical disturbance unattended with any chemical change, but when the impulse reaches the terminal brush of fibrils in the grey matter it liberates a catalase and oxidation processes occur.

NISSL GRANULES, AN ARTEFACT, BUT THEIR PRESENCE OR ABSENCE IN DEAD CELLS OF GREAT SIGNIFICANCE.

The living nerve cell does not contain Nissl granules but a number of minute oval or spherical granules, which, if they escape from the cell remain discrete. By dark ground illumination they reflect the light and appear white, by direct illumination they appear dark and very much like an emulsion. When the cell dies these disappear, and if certain fixing reagents, such as alcohol, formol, or sublimate solution be employed, a coagulation of the proteid constituents of the cytoplasm occurs causing the formation of the Nissl granules. These granules, as shown by their staining reaction with basic dye, consist of a basophil substance.

Significance of the disappearance of Nissl Granules.

The cytoplasm of all nerve cells contains this basophil substance, and inasmuch as it diminishes in amount or even disappears in exhausted cells it has been supposed to be the energy substance of the cell—hence has been termed kinetoplasm. Consequently, although the basophil substance does not exist in the form of Nissl granules in the living cells, yet a comparison of the histological appearances, as to its distribution and amount in normal cells, with the appearances presented by similar cells in morbid conditions, affords a reliable method of determining functional or organic changes of the neurones.

The Nissl granules are especially abundant and form a tigroid pattern in the large motor and sensory cells of the stem of the brain and the spinal cord; they also exist in the Purkinje cells, the large pyramids of the cortex, and especially are they well seen in the Betz cells. In the smaller cells they are not distinctly seen, but their cytoplasm contains basophil substance in all normal cells of Golgi type I.

Morphological changes in the cytoplasm as regards the amount, distribution, and arrangement of the basophil substance (chromatolysis), may therefore be correlated with disease and functional disorders of particular systems and communities of neurones having specialized functions.

Still more important are biochemical changes indicated by an alteration in the staining reactions of the cell, for they may point to a morbid state or to the death of the cell. Whereas the morphological change previously referred to leads to depression or *suspension* of function—a biochemical change indicates a *suppression* of function. If polychrome or toluidin blue and eosin dyes be employed for staining sections we may obtain a basophil and acidophil reaction; thus a motor cell which is dead, e.g., from experimental anæmia or hyperpyrexia, stains a diffuse dull purple, the processes as well as the body of the cell having a homogeneous instead of a differential reaction to the dyes.

Macallum has shown that the Nissl granules contain both iron and phosphorus; the basophil substance is therefore a nucleo-proteid and, as I pointed out in my Croonian Lectures in 1900, diminution of this substance may be "an expression of the diminution of the vital interaction of the highly phosphorized nucleus upon the cytoplasm."

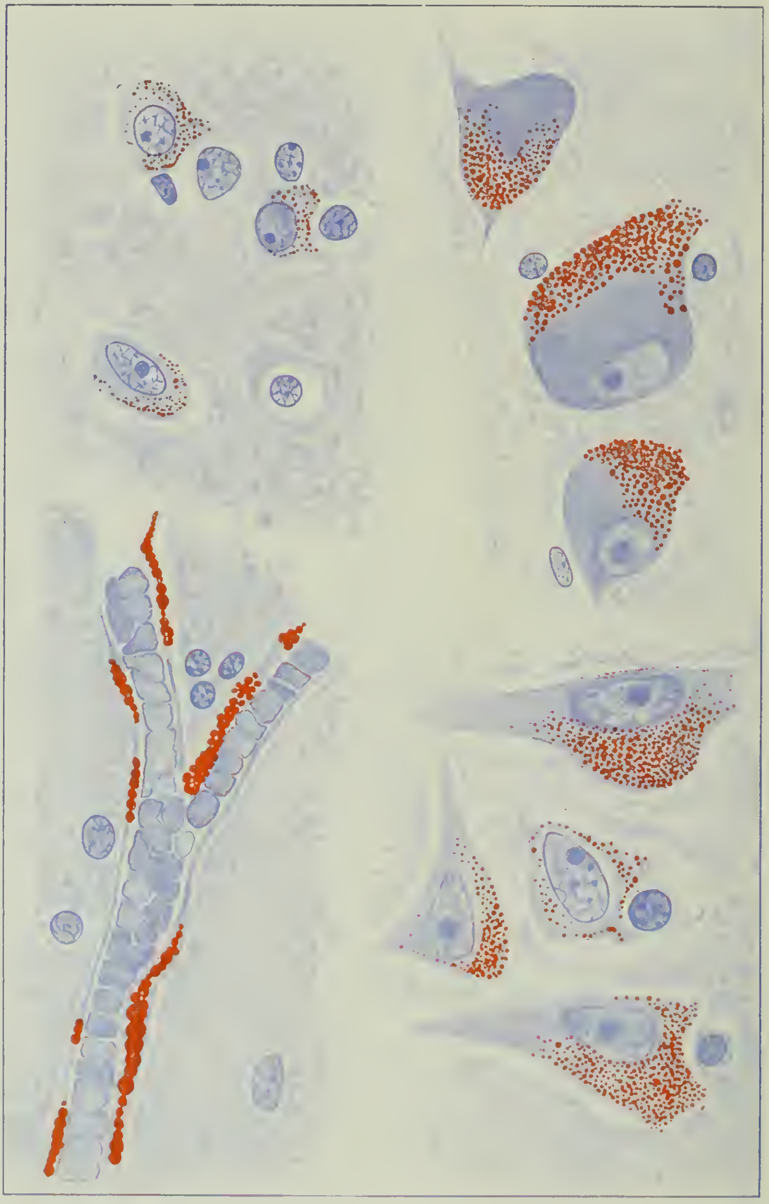
The abundance of nucleo-proteid, with its important iron, calcium and phosphorus constituents in the cytoplasm of the large multipolar motor cells, may be correlated with a greater chemico-physical change in large cells with large and relatively long axons.

The small intercalary neurones (second type of Golgi) have little or no basophil substance in the cytoplasm; the nucleus is relatively very large, but we have seen that these cells enter into the synapse of neurones of the first type.

LIPOID GRANULES IN THE CELL AND THEIR SIGNIFICANCE.

In healthy young animals lipoid granules are not seen in the cytoplasm of the neurones; they are found in the cells of old animals. These granules are also found in varying amount in the cells of the brain and spinal cord of human beings dying of various diseases. Thus I have found them in abundance in myasthenia gravis, in death from shock caused by compound comminuted fracture of the thigh from

PLATE I.



Frozen sections of formalin-hardened brain of Case I, stained with scharlach R. and hæmatoxylin mounted in glycerine. To the right are seven large cells of the optic thalamus showing a large number of lipoid granules in the cytoplasm. (Magnification 600.) On the left is a capillary with lipoid granules in the sheath and in the endothelial cells. Above are three cortical cells with lipoid granules and several neuroglia cells. (Magnification 600.)

gunshot wound. They are found in old people dying of various diseases, but I have found them especially abundant in dementia senilis, general paralysis, dementia præcox, and amaurotic idiocy.

Pighini has made a special study of the cause of their appearance, and has shown that if pieces of the brain of a healthy dog are kept in Locke's fluid in a warm chamber for twenty-four hours, these granules appear and increase in number with the length of time the tissue is kept under these conditions. Controls showed that these were not present in the tissue before they were placed under these conditions.

The facts seem to show that up to a certain point the presence of these lipoid granules may be within physiological limits and be only a sign of deficient metabolism, incidental to a failure of complete oxidation processes; and their existence therefore is not inconsistent with a normal, although probably lowered, functional activity of the neurones so affected. But when the cytoplasm of a large number of cells contains abundance of these granules the physiological limit has been passed and they are an indication of a pathological condition associated with a depression of function sufficient to give rise to impairment or even cessation of function. In amaurotic idiocy the whole of the neurones throughout the cell body are filled with scarlet stained globules. In dementia præcox the cells of the cortex, the basal ganglia, and the medulla exhibit this change. Especially does the process affect the cells of the frontal lobes (*vide* Plate I). Pighini concludes that these fat granules are unsaturated phosphatides for they yield a blue-violet colour with Nile blue sulphate.

These granules stain red with Sudan III, and according to Pighini this would indicate a participation of cholesterin or of its esters with cerebroside. The tendency of the granules to stain a violet-blue colour with Nile blue also suggests the presence of cholesterin. He believes that Marinesco and Obersteiner were correct in considering that these lipoid granules in the nerve cells of old people are products of regressive metabolism, and that hypo-oxidation and pathological processes in which katobolism exceeded anabolism would account for their occurrence—they are probably therefore an expression of anabolic hypofunction.

Having thus considered in detail certain anatomical and physiological facts concerning normal neural structure and function we are prepared to estimate the significance of the morbid changes observed in dementia præcox and see how far they could account for the fundamental disorders of the mind in this disease. I will commence the subject with a brief reference to the supposed causes.

CAUSES OF DEMENTIA PRÆCOX.

With regard to the cause of dementia præcox Kräpelin states that, in the absence of recognizable external causes, it is possible only to think of auto-intoxication which may have some remote connexion with the sexual organs. Kräpelin has pointed to the fact that in a number of cases of dementia præcox the degenerative process began before puberty. He states:—

> "The conclusion seems to me justified that the psychic abnormalities which precede the real onset of dementia præcox already represent, in fact at least, the action of the cause of the disease, even if they can be traced back into the first years of the patient's life. The commencement of the malady would be, if this view should be confirmed, moved back into childhood, for a considerable number of the patients we should have to assume a longer or shorter preparatory stage, in which without noticeable progress, but already in indications, the disease develops certain results such as we find again most strongly marked at the height of its development and in the terminal phases. This interpretation finds essential support 'as it appears to me' in the dementia præcox of children and in engrafted hebephrenia."

My observations show that the appearances of the testes in those early cases of dementia præcox, in which there is a complete abolition of any sign of spermatogenesis, would rather lend support to the view that the regressive atrophy may have commenced before puberty in some cases. For I have observed that in some cases even after months or even several years of mental symptoms pointing to dementia præcox, and, in the notes of which, in not a few, excessive masturbation has been recorded, there exists with the signs of regressive atrophy in many of the tubules still evidence in other tubules of active spermatogenesis. So also in some females the ovaries show Graafian follicles and corpora lutea together with atretic follicles and degenerated ova. Kräpelin asserts that 14 per cent. to 15 per cent. of all inmates of the asylums suffer with this form of mental disease. He dismisses the idea that masturbation is the cause, for reasons which are apparent, viz., masturbation occurs in other mental diseases. Dementia præcox is as common in women as in men and therefore the loss of seminal fluid by the latter, as a result of masturbation, cannot be the cause.

Kräpelin accepts Bleuler's distinction of *fundamental disorders* and *accompanying phenomena* of the disease. *The fundamental disorders* are those which occur in dementia simplex and in the terminal state of *simple weak-mindedness.* From this point of view the weakening of judgment, attention, of mental activity and of creative ability, the dulling of emotional interest, and the loss of energy, lastly the loosening of the inner unity of the psychic life would constitute the fundamental disorders while all the remaining morbid symptoms, especially hallucinations and delusions but also the state of excitement, depression and stupor, further the manifold disorders of volition, negativism, automatism, stereotypy, automatic obedience, mannerisms, grimacing, outbursts of meaningless laughter, &c., would be regarded as secondary *accompanying phenomena* due to disruption of harmonious psychic interconnexions by the degenerative process affecting functional systems of neurones in varying degrees of severity.

The fact that primary dementia of adolescence occurs in uncivilized races as well as civilized, supports the notion that this disease is of endogenous origin. The nature of the accompanying phenomena probably largely depends upon the evolution of the individual psychic personality ; and in this connexion it is interesting to note that Kräpelin found that auditory hallucinations were rare among the native Javanese. Doubtless word symbols play an unimportant part in their mentality as compared with civilized Europeans.

HISTOLOGICAL MORBID CHANGES IN DEMENTIA PRÆCOX AS DESCRIBED BY VARIOUS AUTHORS.

Histological investigations hitherto, with the exception of the examination in a few isolated cases of the sexual organs, have been devoted to the central nervous system, especially the cerebral cortex.

Macroscopically, the brain and the spinal cord in these cases show little evidence of any disease.

Microscopic investigations by numerous neuro-pathologists, notably Nissl, Alzheimer, Duston, and workers (Rae Gibson and Harper Smith) in my own laboratory, have arrived, broadly speaking, at similar results.

Kräpelin sums up the changes thus :—

> " Nissl, in the cases of chronic evolution, has noted profound modification of the cells which he has described under the name 'Destruction of the Nucleus.' A considerable number of cells appeared to be destroyed but there is no atrophy of the cortex. The deep layers contain numerous and large neuroglial cells."

Alzheimer, studying histological lesions in acute cases of catatonia, has described grave alterations of the cells of the cortex, especially of the deeper layers, notably swelling of the nuclei, infolding of their membrane, the cellular body retracted and on the way to destruction, and neoformation of neuroglial fibrils which surround the cells.

In the old chronic cases, Alzheimer found widespread changes in the cells which may be regarded as the terminal state of grave disease which has run its course, in particular sclerotic forms. Very frequently deposits of lipoid products of decomposition were found in the various cells, even in quite young persons. Strikingly frequent were groups of nerve cells in which the basal processes appeared to be swollen and deformed by accumulation of fat. Lastly, diffuse loss of cortical cells could be observed. These severe morbid changes affected especially the second and third cortical layers.

The observations of Klippel and L'Hermitte have shown that these primary degenerative changes of the neurones may affect the whole central nervous system; and they consider that they are either due to an inborn biochemical deficiency or to the effects of an autotoxin circulating in the blood after the reproductive organs have arrived or should have arrived at maturity.

Klippel and L'Hermitte have estimated volumetrically the number of cells affected in different parts of the cortex. Their conclusions are of importance and may be thus summarized:—

(1) The lesions rarely affect the vessels of the brain and spinal cord, and only in restricted points the neuroglia, and there exists neither diapedesis nor lesions of the endothelial walls of the vessels nor of the connective tissue cells.

(2) The lesions of the neurones can be divided into three categories corresponding to the chronological order of their development.

(*a*) Pre-existing lesions, not constant and of congenital origin, constituted by anomalies of development.

(*b*) The lesions of the neurones developed at the same time and in the course of the disease, and consist in atrophy of the neurone with early granulo-pigmentary degeneration.

(*c*) Consecutive lesions marked by arrest of growth affecting the neurone, but also in different degrees the whole organism.

Whereas diffuse lesions are localized in the centres of association, the integrity of the constituent groups of the projection systems of the neurones was in general preserved.

Klippel and L'Hermitte have since called attention to the lesions of the spinal cord in dementia præcox, in particular to degenerative lesions of the posterior column.

Wada states that he has found the large pyramidal cells are comparatively less affected.

Sioli remarks upon the accumulation of lipoid disintegrated material in the tissues especially around the vessels. Most authorities make little mention of extensive fibril destruction, although some authors note changes, and Goldstein asserts that in particular the coarser fibrils are damaged. De Buck, and others, refer to attenuation of the fibres in the supraradial network.

All investigators refer to glia cell proliferation. Alzheimer, in acute cases, describes amæboid hyperplasia of neuroglia, accumulation of glia cells round the nerve cells and morbid new formation of fibres which surround the cells in a particular manner. Nearly all authors refer to the large swollen pale nuclei of the glia cells, many of which can be seen adherent to, or even penetrating into, the decaying nerve cells.

None of the authors refer to the change in the granule layers.

The histological investigations of the central nervous system in dementia præcox of all these eminent authorities are in agreement on certain fundamental points, viz., that there is a parenchymatous degeneration of the neurones, and that the affection of the mesodermic vascular and supporting tissues are slight and of little importance; contrasting therefore most distinctly with the meningo-encephalitis of dementia paralytica and sleeping sickness, both of which are due to a parasitic infection of the central nervous system.

The results of these observers clearly point to a primary parenchymatous degenerative process of the neurones and are in favour of the unicist conception of Kräpelin.

SUMMARY OF PERSONAL HISTOLOGICAL OBSERVATIONS.

None of the eight cases examined showed any thickening of membranes or obvious naked-eye change of the brain; in that respect contrasting plainly with dementia paralytica. Five of the cases died in pre-war times. Paraffin block sections of various portions of the brains were prepared of $5\,\mu$ or $10\,\mu$ in thickness, stained by polychrome methylene blue or methylene blue and eosin. The remaining three were cases that have died within the last year or two. In these the brain was systematically examined both by paraffin block sections, stained in the same way as above, and frozen sections of formol-hardened material stained with scharlach and Nile blue for lipoid granules.

The sections which had been prepared in pre-war days had retained their colour, and the results of the histological examination of these sections appended to the clinical notes of each conform, so far as they go, with the results obtained in the recent cases.

Broadly speaking, the morphological changes correspond with those described by the previously mentioned authorities. They confirm the results obtained by Klippel and L'Hermitte regarding the extension of the degenerative cell changes to the whole brain, including in two cases the cerebellum. They also show that there are two types: (1) In which there has been a congenital arrest of many neurones, especially of the small and medium sized pyramids in the frontal lobe (amentia, admirably described many years ago by Shaw Bolton) and in which later at puberty or adolescence, a widespread neuronic degeneration occurs. (2) The degenerative cell process is not associated with any congenital or prepuberal arrest of development.

The glia cell proliferation is general, and occurs especially in regions where the cell degenerative process is most marked. The neuroglia nuclei are pale in colour, and seen in groups around the degenerated cells, sticking to them or penetrating the cytoplasm. I could see no spider cells, and only slight evidence of neuroglia fibril formation by Ranke's Victorian blue stain, so characteristic of general paralysis.

The vessels show no changes: there is no perivascular cell infiltration, nor endothelial proliferation of the capillaries, so characteristic of the meningo-encephalitis of dementia paralytica. Around the small vessels and in the endothelial cells, especially of the frontal lobe, fatty lipoid granules are found (*see* Plate I).

There is little evidence of nerve-fibre atrophy and sections of the brain stained by the Weigert-Pal method would afford but little explanation of the dementia. Such fibre atrophy or deficiency as occurs could be explained by the destructive decay of the cortical cells, or the congenital cell deficiency. The fibre deficiency or atrophy is most apparent in the frontal lobes.

But the amount of fibre atrophy, due to cell destruction in those cases in which the demential symptoms came on first at puberty or in early adolescence, does not show a correspondence as in dementia paralytica. That being the case, we must assign the major part of the fundamental symptoms of dementia præcox to the functionally incapacitated or degenerated neurones. By this I mean the greater part of the neurones are living, but so biochemically altered that a progressive disorder and loss of function results.

My observations show that, besides the morphological changes in the cells affecting the nucleus and the cytoplasm of the cells, which have been described as occurring in the cortex by all the before mentioned authors, there are, in addition, similar, though not as intense, morphological changes in the basal ganglia, the stem of the brain, the medulla oblongata, and in severe cases of the cerebellum, but the cortical cells are most affected.

No author has paid attention, so far as I can find, to the marked nuclear and cytoplasmic changes in the layers of granules (intercalary neurones) of those regions of the cortex where these cells are so aggregated that they can easily be differentiated from the scattered pale nuclei of the neuroglia cells.

The swelling of the nucleus and infolding of the membrane of great numbers of the cells in the cortex, and to a less degree in the other regions of the brain mentioned, is well established, but no author has pointed out that numbers of the nuclei of these cells show, in varying degrees of intensity, a biochemical change by the fact that the nucleoli, which in normal cells are stained a deep blue (basi-chromatin) reaction, are stained purple or even reddish-pink (oxychromatin reaction). According to Heidenhain this oxychromatin colour with eosin and blue dyes signifies a diminution of organic phosphorus (*see* Plates). This diminution of organic phosphorus may be associated with diminished function in relation to oxidation processes.

Besides the biochemical and morphological changes of the nucleus, there are morphological and biochemical changes observable in the cytoplasm and its processes. Under a low-power magnification the cortical cells, especially the small and medium-sized pyramids, are seen to have their processes broken off, and the regular linear arrangement into the columns of Meynert may be more or less destroyed according to the advance of the disease.

There may in some cases, owing to congenital deficiency, be small places where the cells are absent. Examined with an oil immersion, normal Nissl granules are seldom seen, even in the large Betz cells or cells of the optic thalamus, corpus striatum, pons and medulla oblongata, but they are seen fairly normal in the anterior horns of the spinal cord.

Generally speaking, the Nissl pattern is not seen in the cells and dendrons, but the cytoplasm and processes are stained a dull diffuse bluish-purple, and scattered throughout are vacuoles.[1] These vacuoles are caused by lipoid granules, which have been dissolved out in the process of preparation of the sections (*see* Plates III and V). As can be seen, frozen sections stained by scharlach or Nile blue show the cytoplasm more or less filled with fat granules (*see* Plate I). I have already given reasons for supposing that the basophil substance, which is the antecedent of the Nissl granules, is a product of the vital action of the nucleus on the cytoplasm, and the lipoid granules are evidence of hypofunction, which, when it is abundant and affecting many cells, may be regarded as a pathological process. So that the cells in all the regions mentioned, in all the cases more or less, exhibit direct evidence of hypofunction.

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[1] Since writing this paper I have examined the inferior cervical ganglion, together with the structures named, from a recent fatal case of dementia præcox. The cells of the sympathetic ganglion showed abundant lipoid granules so that it is probable that this change may be more or less universal in well marked cases of this disease.

No author hitherto has directly investigated the condition of the intercalary cells, although reasons have been given for supposing that they play an important part in the formation of the synapse in all systems of neurones and in the cerebellum. Microscopic examination with an oil immersion of regions of the cortex in which these cells form definite layers visible with a very low magnification—e.g., the ascending parietal or occipital convolutions in the region of the line of Gennari and other areas, shows that these cells are profoundly modified. The nucleus is swollen with pale blue or purple staining, and often pink—indications of a biochemical change. The cytoplasm is hardly visible in these stellate neurones of the plexiform[1] layer (granular) owing to the swelling of the nucleus; fine vacuoles are, however, seen in it, and when these cells are stained for lipoid, fine orange-coloured granules are observed corresponding to the vacuoles.

Similarly in these cells this reaction constitutes evidence of a hypofunction and deficient oxidation. Having this in mind, I felt it would be desirable to see if this failing of the basophil substance could be demonstrable to the naked eye. I took, therefore, three pieces of tissue from three separate brains that had been hardened in formol. I selected the cerebellum because its structure is uniform, and therefore comparisons would be more reliable.

Portions of the cortex of the lateral lobe of a case of tetanus, a case of senile dementia, which showed by the usual staining methods marked changes similar but more extensive than dementia præcox, and a case of dementia præcox were taken. These were washed free of formalin and simultaneously passed through the various processes for blocking in paraffin. The three pieces in one block were cut simultaneously, the sections containing the three tissues were placed on cover glasses stained and mounted; so that the conditions in no way varied

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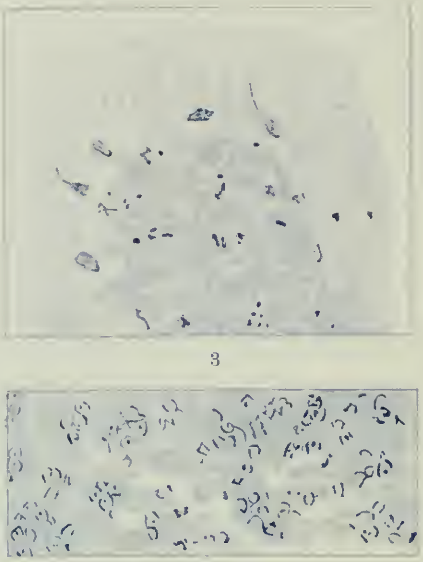
[1] The term used by Cajal and Campbell for the granule layer.

PLATE II.

B



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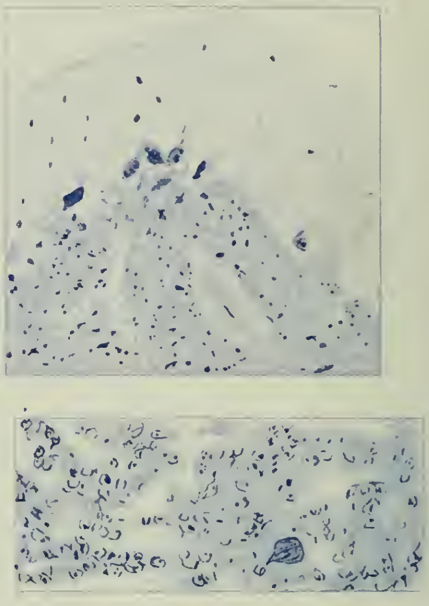

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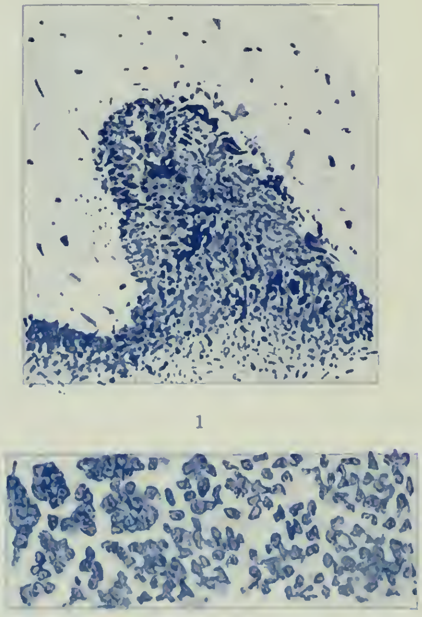
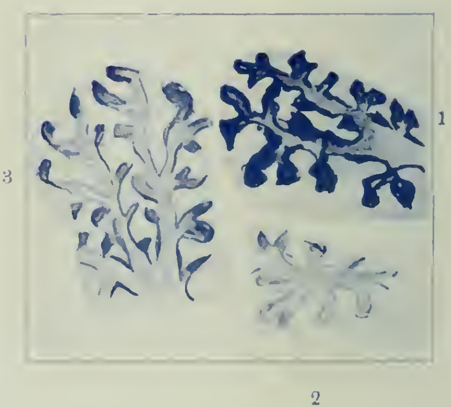


1

A



A, the three sections of cerebellum mounted on one slide (all submitted to the same conditions after removal from formalin), as seen with the naked eye; slightly magnified. Fig. 1, tetanus brain; fig. 2, senile dementia; fig. 3, dementia præcox.

B (1, 2, 3), each of these as seen under the microscope, the upper figure with a low power magnification. The lower, with a high power magnification, shows the granule layer only. Observe the difference in the basophil staining reaction. The only fallacy is the possibility of difference in the formalin employed. Further experiments of a similar nature are being conducted, combined with chemical analysis of the tissues. A more positive conclusion regarding this finding will then be possible; at present it is only very suggestive.

for each section. The results are shown in the accompanying Plate II. It will be seen how much deeper blue the granules of the tetanus case are stained than are the sections of the brains of the two cases of dementia.

This observation, in conjunction with the previous microscopic observations, points to a deficiency of basophil substance and a diminution of the organic phosphorus in the cells of the brain of dementia præcox; a fact which may be correlated with the evidence of a failure of nuclear phosphorus in the reproductive organs. Further observations by chemical analysis of the cerebellum in dementia præcox and normal are in progress. But the following chemical investigations, although by no means conclusive, tend to support the microscopical investigations:—

CHEMICAL QUANTITATIVE ANALYSIS.

Waldemar Koch and Sydney Mann, in my laboratory, made a number of careful analyses of the brains of persons dying of different diseases, including six from cases of dementia præcox:—

> "Compared with the normal, the amount and distribution of phosphorus shows no marked change, but the neutral sulphur shows a great diminution, while the inorganic and protein sulphur is greatly increased. Thus so far nine cases in all have been examined and found to give results which, although varying among themselves, all tend in the same direction—i.e., *a diminution of the neutral sulphur.*[1] This variation appears to be independent of the cause of death, and so far has not been found in other forms of insanity. It does not then seem unreasonable to suppose that the subject of this mental disorder may possibly *possess a general bodily deficiency* for oxidation processes. Some support is lent to this by the fact that Pighini has found an increase of neutral sulphur in the urine in this disease."

These experiments are valuable inasmuch as they indicate deficient oxidation processes. The fact that these observers were unable to detect any diminution of organic phosphorus is easily explicable and in no way contradicts the micro-chemical staining reactions indicative of a deficiency of nucleic acid phosphorus. The reasons I would adduce are these:—

---

[1] An explanation may not be out of place for the term "neutral sulphur." It represents an intermediary stage between unoxidized protein sulphur and oxidized ethereal and inorganic sulphates. It does not split off sulphuric acid on prolonged boiling with hydrochloric acid, neither does it form lead sulphide on boiling with alkali and lead acetate. The term *neutral* is used in contradistinction to sulphates or sulphuric acid.

(1) Not more than 2 per cent. of the brain consists of cells in the grey matter, and only a fraction of this consists of nuclear matter.

(2) The great bulk of the brain is white matter, and of this myelin, which contains a large amount of organic phosphorus is the largest constituent.

(3) There is no appreciable change in the myelin in dementia præcox; consequently, although the nuclear phosphorus may have diminished considerably in amount, it would be only a small, almost inappreciable amount of the total organic phosphorus in the brain.

Pighini has carefully studied metabolism in dementia præcox. He gives the following summary of his results:—

> "With a view of throwing light on the metabolic change associated with the profound symptoms of dementia præcox, I have selected four typical cases of the disease in the acute and eight in the more advanced stage, and in them I have studied the various food elements by means of numerous analyses of the food administered and the excretions. Each case gave results of interest, which may be summarized as follows:—
>
> (1) In the dementia præcox of Kräpelin the acute phase and the advanced phase each present different modifications of altered metabolism.
>
> (2) In the acute phase, as evidenced by motor restlessness, sitophobia, violent impulsiveness, slight elevation of temperature, &c., *there is a negative balance (increased excretion) of nitrogen (urea, uric acid xanthin bases) of phosphorus and sulphur, indicative of a marked dissolution of the phosphorized and sulphurized proteids of the organism.*
>
> (3) In the advanced phase, as evinced by dementia, negativism, tics, grimaces, katatonia, &c., there is a proportionate retention of phosphorus and nitrogen, a loss of sulphur proportionate to these elements, and an independent loss of calcium.
>
> (4) In the two phases investigated there is an altered water metabolism, and a relaxed excretion of chlorine."

The great difficulties attending chemical analyses of the brain and the many sources of error in estimating metabolism in this class of patients makes one careful in drawing conclusions. Still such evidence as exists supports the view that there is a deficiency in the oxidation processes in the brain. Seeing that the microscopic investigations related tend to prove that the oxidation processes are deficient, these findings support the general premises deduced.

THE CORRELATION OF THE MORPHOLOGICAL MICRO-CHEMICAL AND CHEMICAL INVESTIGATION OF THE BRAIN AND REPRODUCTIVE ORGANS WITH THE FUNDAMENTAL CLINICAL SYMPTOMS.

A certain number of cases of dementia præcox occur in congenital aments, or imbeciles. These might be termed dementia præcossissima and from early childhood there are clinical indications of a failure of the higher neural functions. It is quite probable that not only the brain is affected by a developmental deficiency, but that there is a general deficiency of the *élan vitale* and this is manifested by the reproductive organs at puberty, which either fail to develop or an early arrest of spermatogenesis occurs. In this connexion it is interesting to note that I have shown a complete arrest of development of spermatogenesis in several cases of imbecility.

Cases in which clinical symptoms first manifest themselves at puberty or early adolescence exhibit a progressive failure of the *élan vitale*, which may be correlated with the regressive atrophy of the testes, organs in which it can be easily demonstrated that productive energy is most active. But the active nuclear proliferation continually going on in the testes is a synthetic process requiring active oxidation processes to build up a complex organic phosphorus compound, protamin nucleinate out of simple phosphorized lipoid substances. The fact that these synthetic processes rapidly fail points to a germinal defect.

The *élan vitale* so poetically described by Shakespeare in "Romeo and Juliet":—

> "My bosom's lord sits lightly on his throne
> And all day long an *unaccustomed spirit*
> Lifts me above the ground with cheerful thoughts."

is a sign of an outburst of productive energy connected with the supreme function of reproduction.

There is also evidence of germinal defect, for many cases of dementia præcox are congenital aments as shown by the fact that a number of the higher cortical neurones do not develop.

The fundamental clinical disorders of dementia præcox are a weakening of judgment, attention, of mental activity and of creative ability, the dulling of emotional interest and the loss of energy, lastly the loosening of the inner unity of the psychic life. Now if we assume that the neuronic changes show (1) a progressive *suspension* of function of some neurones associated with (2) such intense bio-chemical and morphological changes in other neurones as to indicate suppression of function we are able to explain remission or partial remission of some of the symptoms, and sudden changes from stupor to impulsive behaviour.

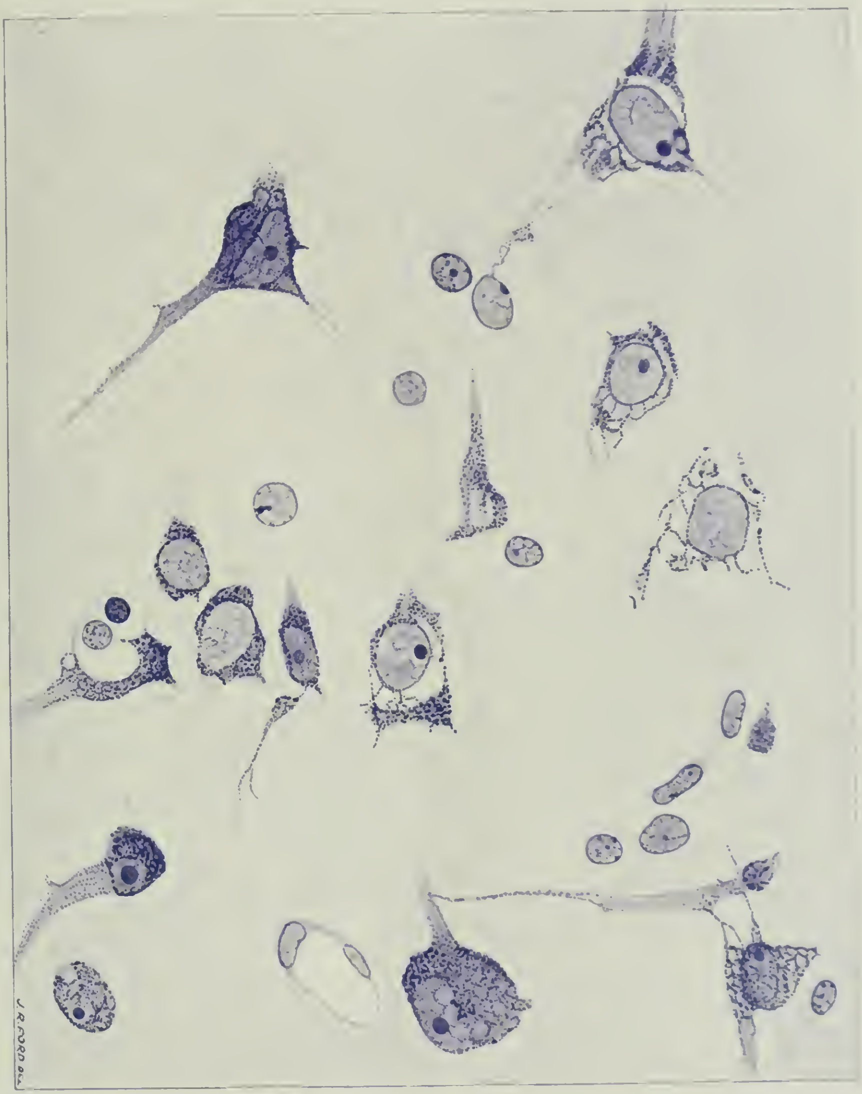
Suspension of neuronic function due to hypofunction from defective oxidation processes caused by auto- or hetero-toxic conditions may vary in intensity and degree, but *suppression* of function owing to germinal lack of durability is incapable of any remission but is progressive, so that even when a remission of some of the symptoms occurs there is a residuum of weak-mindedness, *dementia simplex*, which is progressive and continuous. It should be mentioned that the neurones are in the normal individual permanent cells adapted for a prolonged life and protected by special anatomical conditions from injury and disease. Any form of stress, using the term in the wide sense employed by Mercier, will contribute to lower the durability of the neurones.

Now it is known that some cases which at first clinically appear to be cases of dementia præcox recover. Moreover, some cases of confusional insanity may present a clinical picture of dementia præcox and recover completely. It must be supposed that these cases are due to a hypofunction, and we should probably find a general condition of lipoid granules in the neurones with basophil chromatolysis and disappearance, or partial disappearance of the Nissl granules.

But evidence of a biochemical and morphological degeneration of the nucleus points to a condition which would end in suppression of function, although this condition does not necessarily imply death of the neurone and atrophy of the axon. The morphological changes implying *suppression* of function are found especially in the cortex and particularly the cortex of the frontal lobe in which neuroglia proliferation is most marked. Associated with this are universal changes in the various regions of the brain pointing to hypofunction, viz., lipoid granules in the cytoplasm and in many cells an oxychromatin or a tendency to an oxychromatin reaction of the nucleus (*see* Plates III and V).

The affection of the stellate intercalary cells which enter into the synapse, and the evidence I have adduced of the importance of these cells in connexion with oxidation processes productive of neural energy and transmission of nervous impulses, suggest that a hypofunction or suspension of function of these neurones would lead to synaptic dissociation and thereby account for psychic dissociation and the coming and going of symptoms; or where there is a permanent morbid change, to a suppression of their function with permanent dissociation.

PLATE III.



Section of cortex of angular gyrus (Case I). Degenerated pyramidal cells. Observe the swollen nucleus with intranuclear network. There are no Nissl granules seen, only a purple dust and vacuoles. The nucleolus is stained a reddish purple instead of blue. The neuroglia cells are faintly stained in comparison with the normal. Some of the pyramidal cells are undergoing autolysis, and one shows two neuroglia cells within a hollow space of the cell. Near this is a small stellate cell with greatly swollen nucleus. (Magnification 4, ocular, and $\frac{1}{12}$ oil immersion.)

We have thus two morphological conditions which will account for the fundamental disorders, and the nature of these disorders will depend upon the cerebral structures affected whether in such a way as to produce suppression or suspension of function. Naturally the nature of the mental disorders will also depend upon the localization and the relative intensity of the hypofunction, suspension, or suppression of function of the neurones.

It is quite probable that there is a hypofunction of the whole of the bodily tissues; there is certainly a diminished vital resistance to microbial infections. A large percentage of these cases of dementia præcox die of tuberculosis, but my observations show that exactly the same neuronic changes can be found in dementia præcox cases that have died of acute pneumonia. So that although it is common to find stupor in patients affected with active tuberculosis and although the absorption of toxins may, therefore, have played a part in the production of some of the symptoms, yet I have formed the conclusion that the essential cause of this disease is an inborn germinal defect, the nature of which is unknown. When we come to consider that in a normal seminal emission there are about 226,000,000 spermatozoa, it is probable that some are of greater vitality than others, and that by the law of chance a weak spermatozoon may occasionally reach the ovum, or the ovum itself may be deficient in vital energy. Again the combination may not be favourable to the production of a sound and durable organism. That this chance of a spermatozoon being vitally deficient would be increased by hereditary taint, or by any of the causes which would lower the vitality of the parents, seems probable.

Nature is unmindful of the individual, mindful only of the species, and by early mental decay and arrest of spermatogenesis, reproduction of a degenerate being is thereby prevented.

CASE I.—DEMENTIA PRÆCOX.

W. H. *Previous History.*—According to the statements of the patient he joined, as a boy, the Somerset Light Infantry in November, 1912, and was in the band. After two years and fifty-six days' service he was discharged for deafness. He rejoined the Army in March, 1915, and was sent to France. Whilst at Dickabush in November, 1915, a shell burst near him and he was sent to the base, suffering from shell shock. From there he was sent to Faversham, Chatham, and Mill Hill, and finally discharged as unfit for further service After leaving the Army he apparently did no good. He had many jobs, but admitted he could not keep them, and had been in prison for being in possession of matches whilst on munition work. He was admitted to Long Grove Mental Hospital on June 3, 1918, aged 20.

Condition on Admission.—Physical condition: Good; comparatively normal and of average development.

Mental Condition.—He was found to be suffering from recent melancholia and was an evident case of dementia præcox. He had several mannerisms and laughed and grinned inanely, was full of all sorts of strange fantasies and ideas and said he felt he was under some influence or spell. He was very solitary and uninterested in anything except his own peculiar ideas. He himself thought he had always been strange, even before joining the Army, but that he had been much worse since sustaining shell shock. He was evidently hallucinated and sometimes very confused and contradictory. He accounts for his fits of depression in a very inconsequential way, and even when saying that he had asked the attendants at the infirmary to poison him he began to smile in an imbecile way.

In July, 1918, his mental condition was deluded and hallucinated, restless, impulsive and resistive, with occasional faulty habits. On one occasion he butted his head against the wall and at another time he threw himself about the floor. This condition continued with lapses into a mildly stuporose condition.

On October 12 he threw a flower pot at an attendant, and on November 1 he was noted as being in an unsettled condition, aggressive, impulsive and intolerant of discipline.

On November 11, 1918, he was transferred to Hanwell Mental Hospital where he was diagnosed as dementia præcox, his mental condition being one of moroseness and confusion, refusing to speak, but with outbursts of aggressiveness and violence.

On December 6, 1918, he was noted as showing no improvement in his mental condition, but his physical condition was good and he was clean in his habits.

Throughout the later part of January and February, 1919, he was troubled with a low form of pneumonia and a persistent cough. This condition however cleared up and his health was good. His mental condition showed no improvement and he continued to be dangerously impulsive, sullen, obstinate, and quite devoid of interest in himself and his surroundings.

On October 31, 1919, he developed a pyrexia with symptoms of influenza and on November 5 he was found to be suffering from lobar pneumonia, from which he died, at 1.20 p.m., on November 6, 1919—seventeen months after admission to Long Grove.

Post-mortem Examination.—State of nutrition: Good. Brain: Weight, 1,158 grm.; convolutions irregular; badly developed. Right lung: Solidified throughout; red hepatization. Left lung: Solidified and red hepatization at base. Heart: Walls dilated; blood-clot in auricle. Liver: Enlarged and fatty. Kidneys: Congested. Testicles: Right, 13·8 grm.; left, 15·8 grm. Other organs normal.

Cause of death, acute lobar pneumonia.

Microscopic Examination of the Brain.—By hæmatoxylin and scharlach and Herxheimer scharlach methods for fatty granular degeneration: Comparatively to the dementia senilis there are few of the cortical cells which show the granular fatty degeneration in the motor area. In the frontal region, however, a large number of cells, small, medium and large, of all the layers, show fatty granular degeneration. The nuclei of the cells are swollen, the nuclear membrane infolded or irregular in outline and the contents often show fine orange-stained granules. The nucleolus may be stained purple or orange red.

Medulla Oblongata.—The cells of the ventral nucleus of the vagus and the nucleus ambiguus show the fatty granules in them. There is very little evidence of change in the hypoglossal. In some of the cells the nucleus is obviously swollen and shows degenerative changes. This applies to all cells including those of the olivary body, although the pigment in this kind of cell is finer. The large cells show the lipoid granules better.

Unstained Specimen of Medulla.—Many of the cells show fine granules faintly but not nearly so many as in the stained specimens. The conclusion is that this is a fatty degeneration as the granules stain with scharlach "R." Very few cells of the pons show any fatty change at all.

Paraffin Sections.—By eosin and toluidin blue and toluidin blue alone.

Cortex, Motor Area and Ascending Parietal.—There is a deficiency of basophil substance generally. The Nissl granules in the majority of cells where these granules are well seen in the normal, are in the sections deficient, and there is a tendency, in a varying degree, of conversion of the granules into a dust-like condition. Many of the large motor cells show yellow pigment granules. In some the nucleus is eccentric. As regards the medium and smaller pyramids, many show swollen nuclei, chromatolysis, shrinking of the cell and disappearance of the dendrons or dendritic processes. The cytoplasms often show yellow or unstained granules due to the lipoid substance having been dissolved out. The most marked changes are seen in the deep polymorphic layer where there is hardly a normal looking cell to be found. The nuclei are swollen, in many instances they occupy nearly the whole of the cell. The membrane is often infolded and the nucleus itself very frequently has a pale granular appearance as if it had undergone fatty degeneration and the fat had been dissolved out. The cytoplasm contained but little basophil substance and is filled with minute granules or vacuoles, due to the lipoid having been dissolved out. The processes of the cells are either not seen or are broken off. There is a proliferation of neuroglia nuclei, faintly stained with fine blue chromatin granules. These are seen at all levels, but especially where the cell changes are the most marked. In the ascending parietal convolutions, where the intercalary cells form a definite layer of granules it was observed that these cells have large swollen nuclei, faintly stained blue or pink, according to the stain employed and with but very little cytoplasm. It was observed that the cytoplasm contained little or no chromophylous substance and was filled with fine vacuoles.

Cerebellum.—The cerebellum is well stained; both the cells of Purkinje and the granules give a good basophil stain and this structure might almost pass for normal.

Medulla Oblongata.—Most of the cells of the various nuclei have abundant basophil substance. The Nissl granules however are not so sharply defined as in the normal. Some of the cells show deficiency and a tendency of the granules to break up into dust. A few of the cells show swollen nuclei and vacuolation of the cytoplasm of the cells. These early changes are best observed in the ventral nucleus of the vagus. There is some proliferation of the neuroglia cells especially about the floor of the fourth ventricle.

Pons.—The pontine cells of the first type show a diminution of basophil substance and disintegration of Nissl granules to a variable extent; lipoid granular change in the cytoplasm, especially in the cells with a swollen and often irregular shaped nucleus. The small intercalary cells have very pale nuclei and are difficult to differentiate from the glia cells which are proliferated and exhibit a similar pallor and lack of basophil substance.

Optic Thalamus.—In the thalamus the large medium and small sized cells are all affected with the nuclear and granular change described (*vide* Plate I). The small intercalary cells are similarly affected to those in the pons and one can see the nucleus surrounded by a finely granular cytoplasm.

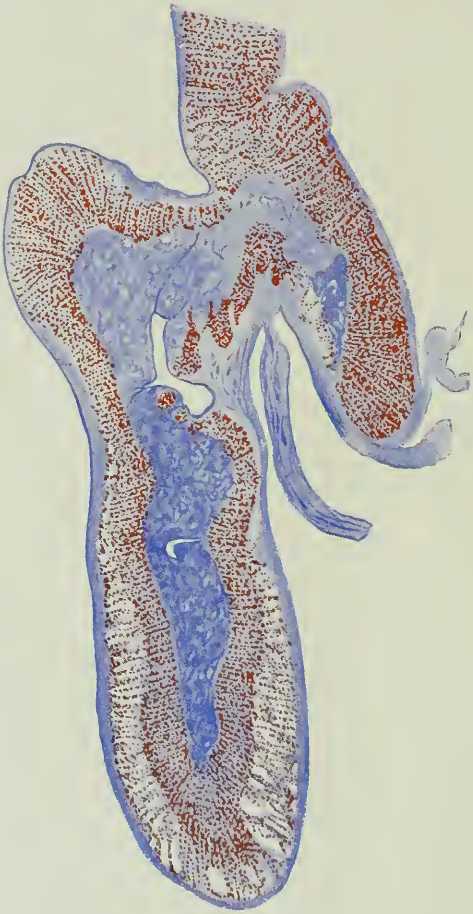
Corpus Striatum.—The large cells are far less numerous. The changes are the same as have already been described in other cases. Even more marked nuclear and cytoplasm degeneration is to be seen. Oxychromatin reactions in the nucleolus and substance of the cell after staining with toluidin blue and eosin, the granular change in the cytoplasm is seen and the processes of the cells are broken off.

In the Island of Reil the nuclear and cytoplasm changes are well marked especially in the polymorph layer. It is evident also in most of the cells of the pyramidal layer. There is a proliferation of glia cells throughout the cortex. By Victorian blue there is no evidence of proliferation of neuroglia fibrils.

Angular and First Temporal.—Both the temporal and angular regions of the cortex show little difference in the number of cells or their arrangement in columns from the normal. The granular layer is well marked. Stained with toluidin blue and eosin all the cells, under low power magnification, seemed to be stained purple. The same is seen in the angular cortex, though perhaps not quite so marked as was found in the auditory. The large and medium-sized pyramids seem to be less affected but the granular change is quite evident in the smaller pyramids and well marked in the infragranular polymorph layer (*vide* Plate I).

Calcarine Fissure.—Under a low power the cell arrangement appears normal. Although most of the small and medium-sized pyramids of this region show a granular change in the cytoplasm and a swollen nucleus, the most marked change is found in the intercalary cells; which when stained by Nissl method are quite a pale blue. With toluidin blue eosin the nuclei are stained pink and the nucleoli also.

PLATE IV.



Section of adrenal gland (Case I) hardened in formalin and cut with freezing microtome. Stained with scharlach R. and hæmatoxylin. (Magnification 12.)

Occipital Pole.—Exactly the same change is observed here as in the calcarine fissure.

Superior Frontal.—Rather a patchy arrangement is observed under a low power in the cells of the supragranular layer, as if there were a deficiency of development. The regular arrangement in columns is not maintained. The apical processes are obliquely set to the surface and the cells themselves seem to have their processes broken off. All the cells seem to take a pinkish tinge; this is especially noticeable in the small cells (*vide* Plate V, fig. 3).

The impression gained from this examination is that the frontal region is more affected than the other parts of the cortex examined, and there is a deficiency of development. Examination by the high power shows the same cell changes described elsewhere in the brain, but in a more advanced degree. In some instances the cells seem to be almost completely destroyed. The nucleoli all take the red dye. Here and there are patches where only glia cells are seen. With Victorian blue no definite evidence of neuroglia fibril proliferation can be observed.

Frontal Pole.—By low power there is less evidence of change than in the superior frontal, still there is not the normal arrangement in columns. Observations with high power magnification however show an advanced nuclear and granular change of the cells similar to those seen in the superior frontal. Frozen sections of the tip of the pole of the first temporal convolutions stained by scarlet and hæmatoxylin, show marked granular degeneration of the cells of the deeper layers. Fat granules are seen in the perivascular spaces of the smallest vessels; some apparently taken up by the endothelial cells (*vide* Plate I).

Adrenal.—By scharlach-hæmatoxylin. The cortex is narrower than normal. The greater portion of the cells have no lipoid content. Those that have are stained a deep red. It is possible that this deficiency of lipoid is due to the pneumonic condition from which he died, but the narrow cortex and low weight (right, 30 grm.; left, 45 grm.), for a young man is abnormal. The cortex seems to be more deficient than the medulla (*vide* Plate IV).

Testes.—No spermatozoa were found in films made from emulsion of the testes. Sections of the testes stained by scharlach and hæmatoxylin show some deficiency of lipoid in the Sertoli cells, and thickening of the basement membrane. There is no evidence of spermatogenesis, and many of the tubules are apparently atrophied.

CASE II.

Clinical Notes.—R. D. G., aged 20. Admitted Colney Hatch, April 27, 1914. Died October 20, 1916. Has a brother in Hanwell (said to be an epileptic), admitted June 10, aged 21. When admitted he was certified as suffering from delusional insanity. He adopts fantastic attitudes, gesticulates freely and hears voices of imaginary persons and claims to be in close communication with the Almighty. He says that he has been dead but has risen from the grave. He was in ex-7 standard at school. He had night horrors as a child and sleeplessness due to hallucinations. He has been a boot-repairer. In the frequent notes taken during the twenty-eight months of his residence in the asylum there is no statement that he masturbated. The diagnosis was changed in April, 1915, to dementia præcox. He is dull and stupid and very rarely speaks to anyone. He is disorientated for both time and space, extremely untidy and faulty in habits. Further notes: Nothing except that he is occasionally troublesome. June 2, 1915: First stage of tuberculosis. September 18, 1916: Another attack of so-called apical pneumonia. Death from acute pulmonary tuberculosis, October 20, 1916. Testes, 15 grm. and 16 grm. No spermatozoa seen by dark ground examination. Vesicula seminalis seen by dark ground examination.

Microscopic Examination of Testes.—Examination of frozen sections stained by hæmotoxylin and eosin. No spermatozoa seen in sections. Many of the tubes atrophied and only show sustentacular cells, the spermatogonia spermatocytes and their nuclei are absent; the basement membrane is thickened and the fibrous interstitial tissue increased; the interstitial cells are seen but they appear to be smaller than normal as if in the resting stage or partial resting stage. The majority of the tubes show spermatogonia and spermatocytes with active mitotic figures, but spermatids are rarely seen and nowhere could I find any spermatozoa.

Interstitial lipoid: hardly any lipoid seen. Very little in cells of tubes. Examined with high power, fine granules seen in syncytial (Sertoli) cells.

Examination of Brain.—Weight of brain, pons and medulla after hardening in formol until September 15, 1919, 1,210 grm.; deduct 10 per cent. increase due to hardening, 121 grm.; = 1,089 grm. net weight. The weight therefore is considerably below the average weight of the male brain.

Microscopic Examination of Brain: Frontal Lobe.—Toluidin blue and eosin. The majority of the cells are stained a rather faint purple. There appears to be a failure of cells groups in pyramidal layer as if undeveloped. In these regions the glia cells are in excess with very pale stained nuclei. The nuclei of the neurones are large, clear and irregular in outline. The nucleoli and intranuclear network are not so well-stained as normal. The intranuclear network is stained pink and the nucleolus is frequently stained purple instead of blue. The cytoplasm is either stained a diffuse purple or a faint pink. As a rule the larger the cell the more blue is seen in the stain. Nowhere is there evidence of Nissl granules. The granular and infra-granular layers show the most imperfect staining under a low power. With an oil immersion the nuclear change is most pronounced in these cells. A great proliferation of glia cells is seen, many of which can be seen adherent to the degenerating neurones—not a normal cell could be seen. The glia nuclei are as in other cases only faintly stained.

Superior Frontal Lobe.—Very marked nuclear change in all the cells both large and small. The cytoplasm of the large pyramids shows no Nissl granules. The edges are crumbling, the substance is diffusely stained. There are many neuroglia cells—some of them lying in the perineuronal spaces.

Motor Area.—A number of the large Betz cells show well marked nuclear changes. In others the nucleus appears normal, although there is usually a deficiency of the chromatin substance. A great many satellite cells are seen around the neurones. In other respects the changes in the cells of the various layers resemble those described as occurring in the frontal lobe—except that the change is not nearly so marked and there is no evidence of failure of development of cells.

Occipital Cortex.—Similar changes of nucleus and cytoplasm in cells observed only not so marked. Both granular layers show very pale staining. The cells have a large swollen clear nucleus, the whole stained a purple pink, the cytoplasm is more evident and exhibits less crumbling and disintegration than the cells in the frontal lobe.

Cerebellum.—Strikingly evident under a low power is the deficiency of chromatin in the granule cells. There are patches and streaks of faint blue or purple staining. These patches can be resolved under a high power and show in their midst very faintly stained nuclei. The cells of Purkinje are generally of normal shape, but exhibit as a rule only fine Nissl granules. Generally speaking, the cells are stained a diffuse purple with the polychrome or double stain.

Examination of Brain for Lipoid Granules in Cells.—Frozen tissue sections, stained by scharlach and hæmatoxylin. Cells of optic thalamus: A great many of the larger and medium sized cells show lipoid granular degeneration of cytoplasm. Frontal lobe: Nearly all the cells of the various layers show lipoid granular degeneration. Motor area: Little or no degeneration seen: only a few cells of deeper layers show some lipoid granules. Medulla: A few of the large motor cells show slight lipoid degeneration.

Case III.

History.—D. B., an unmarried labourer, aged 33. He was admitted to Claybury Asylum on May 15, 1903, suffering from melancholia. He was sullen, reserved and obstinate, seldom speaking except in answer to imaginary voices addressing him. At times he struggled with the attendants, but seemed to have no recollection of same. He had no idea of time or place and his memory was very bad, so bad that he did not know what occupation he had followed. He also suffered from marked delusions of persecution and thought his food was being poisoned. He stood for long periods of time in the same place and position. He was in a poor state of health, but his heart and lungs and other viscera were reported healthy. The progress notes up to August, 1908, when the patient eventually died of pulmonary phthisis, report a state of progressive dementia. In April, 1905, he was said to be simple, childish and laughing without cause and at other times suffering from great depression with aural hallucinations and delusions of persecutions.

Microscopical Notes.—Weight of brain, 1,250 grm. Nissl polychrome: Basal ganglia, small and large cells show characteristic nuclear change (nucleolus looks red). Considerable increase of neuroglia cells, pale colour (deficient nuclein), many in groups around degenerated cells.

Medulla.—Polychrome. Very characteristic nuclear and cytoplasm changes throughout. The large cells of the hypoglossal are least affected. The large cells of the ventral nucleus of the vagus are much affected, the small cells of the dorsal nucleus are much affected, and all the small cells of the medulla, in varying degrees, show the characteristic changes. The neuroglia cells are proliferated and although there is deep staining of the Nissl granules, especially in the large motor cells, nevertheless the nuclei of the neuroglia are very pale pink, giving therefore an oxychromatin reaction.

Cerebellum.—Purkinje cells are well stained with the basic dye blue, but the granules are poorly stained by the basic dye a purple pink. The intranuclear network is very distinct and is so stained.

CASE IV.

History.—J. G. H., admitted to Claybury Mental Hospital on May 24, 1904, said to be suffering from mania. He had been a clerk by occupation: his age was 27 and he was unmarried. He had led a temperate, steady life and shown considerable ability and aptitude for his work while at school. Fifteen months prior to admission he suffered from religious excitement; expressed desire to become a missionary and to reform the East End of London. On his father's side of the family there was a history of intemperance and mental weakness and one of his aunts was an epileptic. He began wandering aimlessly at nights and staying out late. When he was admitted to Claybury he suffered from delusions of persecution, aural hallucinations and expressed strong religious convictions. At first he was a very difficult patient to deal with, he attempted to escape and had to be shut in a strong room. Later he developed a suicidal tendency, but this soon passed off. He became much less impulsive and much quieter. In January, 1905, he was diagnosed as adolescent mania and was much more resistive and restless about this time. In 1907 he was for some time (months) very troublesome, striking and teasing other patients, but gradually he was becoming more simple and childish and grinned and grimaced in a meaningless fashion. After a few months of more marked dementia the patient caught cold, developed broncho-pneumonia, and after four days' illness died on September 25, 1908.

Microscopic Notes.—Weight of brain, 1,590 grm. Tip of frontal convolution, Nissl stain: Every cell shows characteristic degenerative changes. Swelling of nucleus, infolding of membranes, pallor of nucleolus or reddish colour, vacuolation, granulo-pigmentary degeneration, disintegration of cytoplasm, shrinking of cell, no great amount of neuroglia cell proliferation, pallor of glia cells. No vascular changes. The most marked cellular change I have seen. Nissl cerebellum: Granules faintly stained comparatively. Purkinje cells: Nuclear swelling and infolding of nuclear membrane; nucleolus pale or reddish colour. Chromatolysis more marked in some cells than others. Nuclear change marked in some cells and yet little chromatolysis.

CASE V.

History.—C. O'C., aged 22, employed as a domestic servant. Attack commenced about fourteen months previous to admission, but she had gradually been getting worse during the last three months. No further previous history obtainable. On admission to Horton Mental Hospital, April 16, 1903, she was diagnosed as a case of melancholia, her mental condition being very dull, with hallucinations and an impaired memory. Her bodily condition was fair. Both bodily and mental condition remained unchanged until March, 1907, when she was re-certified and diagnosed as suffering from dementia præcox. She was then stuporose, dirty in her habits, and showed marked katatonic signs. Her health continued fair. On February 14, 1908, she had a very violent outburst, developing a tendency to attack anyone in her vicinity. This was followed by a condition of stupor in which a foul stomatitis developed. She was removed to the infirmary. Her pulse became rapid and weak, and her temperature became subnormal. A cough and dullness at bases of lungs developed; also signs of peripheral neuritis of the legs. On March 8, 1908, she collapsed with a very subnormal temperature and extremely feeble pulse, and died at 9.35 p.m. of the same day.

Post-mortem Examination.—State of nutrition: Poor. Lungs: Œdema and congestion of bases with muco-pus in small tubes. Heart: Marked fatty degeneration with some infiltration and pigmentary change. Aorta: Slight atheroma. Liver, spleen, kidneys and small and large intestines were congested. All other organs normal.

Cause of Death.—(1) Fatty degeneration of heart; (2) bronchitis.

Microscopic Examination of Brain.—The following structures were examined: Medulla oblongata, top of first frontal, top of ascending parietal. The staining employed was polychrome. The sections were examined about ten years after they had been made. The tissue had been alcohol-fixed and the sections had retained their coloration perfectly. Medulla oblongata: All the cells show marked changes. Nissl granules are absent. The cytoplasm is shrunken and contains a basophil staining duct; there is vacuolation of the cytoplasm indicating that lipoid granules in abundance had been present but dissolved out. The nuclear changes in greater or less degree were present in the majority of the cells. In many (especially the smaller) cells the nucleolus is stained a faint pink. The nucleolus instead of giving a blue stain is either pink or purple. The same changes only marked if the cells are observed in the two cortical structures above named. The glia cells stained faint pink are proliferated. In the ascending parietal the granule layer is especially affected. The small cells show a large swollen pink nucleus with a vacuolated ragged surrounding of cytoplasm. The polymorph layer of cells show well marked changes of the cytoplasm and nucleus. The cortex only measures 1 mm. instead of 2 mm. in depth. The frontal top: The columns of Meynert are not disorganized. The pyramidal cells show the apical processes well, and under a low power the section might pass for normal. Under high magnification the most pronounced and typical changes in all the cells are seen, especially marked is the change in the polymorph layer. The following changes were observed: Complete absence of Nissl bodies; great diminution of basophil substance in the cytoplasm, leaving an irregular vacuolated structure. Some of the vacuoles contain yellow lipochrome granules, others obviously have contained a lipoid substance that has dissolved out in the preparations of the sections. Some of the cells are mere shadows. The nucleus is swollen irregular in shape, membrane often infolded or crenated. The nucleolus is stained pink or purple in many of the cells. There are large numbers of pale slightly pink-stained neuroglia cells. These can often be seen adherent to the dead or degenerating nerve cells. The dendritic processes are broken off and not a healthy normal cell can be seen. The changes are most marked in the polymorph layer.

CASE VI.

History.—M. R., aged 17. No history prior to admission given. Admitted to Horton Mental Hospital on August 29, 1907. She was in a stuporose condition and took no interest in her surroundings. It was with difficulty that she could be made to open her mouth or eyes. She was in a very weak state, with discharging sinuses round left shoulder and signs of consolidation of left lung. Pyrexia was maintained, and she became slowly weaker, developing signs of cavity in her left lung, and her right lung was also affected. Her mental condition was at first one of katatonic stupor. At night she became noisy and restless, later she was more or less stuporose. Patient became rapidly weaker, and finally died at 5.12 a.m. on January 4, 1908.

Post-mortem Examination.—State of nutrition: Very emaciated. Both lungs: Consolidated with tubercle: in both upper lobes there was a large cavity. Small intestines: Tubercular ulceration throughout. Organs of generation: Both tubes much enlarged, matted together and full of caseous material and slightly adherent to surrounding structures; caseous material in body of uterus indicating tubercular disease. Joints: Tubercular disease of shoulder joint; other organs comparatively normal.

Cause of Death.—(1) Tubercular salpingitis; (2) tubercular disease of lungs, intestines, and left shoulder-joint.

Microscopic Examination.—Polychrome and eosin. Methylene blue and eosin-stained sections of the cortex in various regions. The corpus striatum, optic thalamus, medulla oblongata, and cervical spinal cord were examined. The cells of the cortex in various regions, especially the frontal, show changes of a primary degenerative character. The columns of Meynert, as a rule, are seen, but the cells generally speaking are deficient in basophil staining reaction. The outline is irregular, the processes are short, and the apical processes are only clearly defined for a short distance. The nuclei show signs of degeneration. They are swollen, and the nuclear membrane is infolded or crenated; the nucleolus and intranuclear network is stained a purple or red. The deeper layers of polymorphic cells are more affected than the superficial

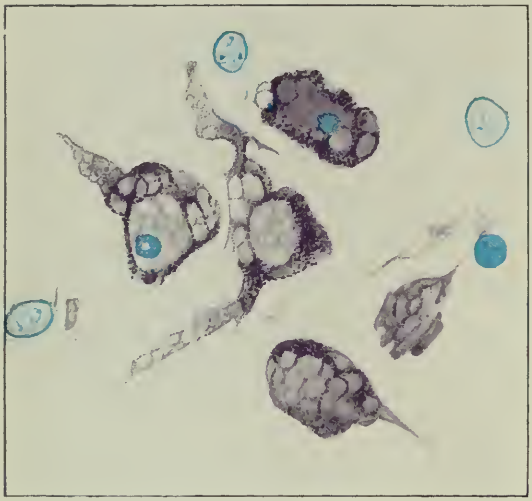


FIG. 1.

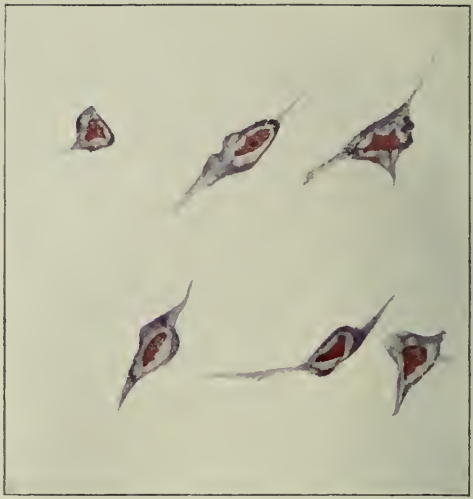


FIG. 2.

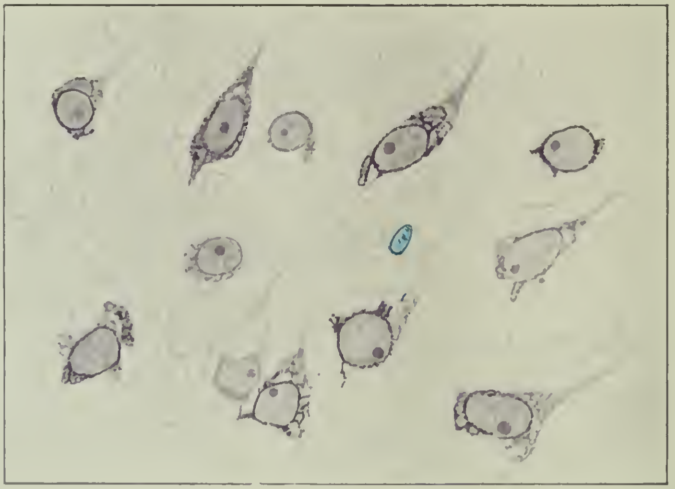


FIG. 3.

Sections of brain of Case VI. Fig. 1 stained with polychrome and eosin. 1, cells of optic thalamus; the nucleus is swollen, the cytoplasm is markedly vacuolated, the processes are distorted or broken off; the nucleolus is stained a reddish-purple. (Magnification 600.)

Fig. 2: Sections of medulla oblongata, degenerated cells of dorsal nucleus of vagus. The nucleus is swollen, and the nucleolus is stained red. (Magnification 600.)

Fig. 3: Section of frontal pole of Case I, stained with toluidin blue and eosin. Marked swelling of the nucleus; the cytoplasm is vacuolated. The nucleolus is stained pink. (Magnification 600.)

layers of pyramids. Both the corpus striatum and the optic thalamus show similar changes in a marked degree. The intercalary neurones of the cortex and the basal ganglia give an oxychromatin reaction. The neuroglia cells are likewise affected, the nuclei staining pink instead of blue. They are increased in numbers, especially around the more obviously degenerated neurones. Similar changes are found in the medulla oblongata, even the large cells of the somatic nuclei show a marked diminution of the basophil substance (*vide* Plate V).

CASE VII.

History.—C. J. H. Admitted to Bexley Mental Hospital on September 14, 1901, aged 23. No history, prior to admission, except that he cut his throat in an attempt to commit suicide; that he reached Standard IV at the age of 13; and that the onset of the mental condition occurred fourteen months previously.

Condition on Admission.—Poor physical development; except for a slight general bronchitis, the organs were normal.

Mental Condition.—Attention was difficult to obtain and retain, and he was intellectually dull. Comprehension was poor and reaction most erratic and absurd. His memory was also poor, especially for recent events. He was eccentric, self-absorbed and entirely controlled by his hallucinations and delusions. He apparently believed he was related in some mysterious manner to the Heavenly spirits, as he denied all relation to human social classes. He was not depressed, neither was he resistive or hostile. Diagnosed as a case of recent melancholia.

His manner developed into restlessness, noisiness and hostility at frequent intervals, when he attacked fellow patients and attendants. Between these relapses he was quite amenable, and did some useful work in the wards, but was never reliable.

On January 30, 1908, his mental condition became much worse. He was very dull, confused, emotional and depressed. At times very hallucinated, resistive, restless and inclined to be hostile. Health remained fair.

On February 23 he developed hyperpyrexia, and was found to be suffering with acute lobar pneumonia, from which he died at 8.12 a.m. on March 2.

Post-mortem Examination.—State of nutrition: Poor. Right lung: Normal. Left lung: Consolidation of the whole of the lower lobe; grey hepatization; recent dry effusion over the lower lobe and the base of the upper lobe. All other organs normal except for some congestion of the liver, kidneys, and small and large intestines. No tuberculous or dysenteric lesions.

Cause of Death.—Lobar pneumonia.

Microscopic Notes.—Polychrome stain tip of first frontal. Very marked granulo-pigmentary degeneration of all cells (both first and second type of Golgi), proliferated neuroglia cells with pale nuclei. Top of ascending parietal granular layer, swelling and oxychromatin reaction of nucleus, very marked characteristic degeneration of supra- and infra-granular pyramids, more marked in the infra-granular region. Extreme pallor of neuroglia cells, of which there is some evidence of proliferation. Basal ganglia: All stages of degeneration of cells up to disintegration and complete destruction, the same and as intense as in the cortex. Groups of neuroglia proliferated cells stained pale pink. Cells of second type of Golgi are undergoing degeneration, for they are studded with small pale granules in many instances.

Medulla Oblongata.—Although the Nissl granules are well formed in the hypoglossal cells, the nuclei are obviously changed; they are swollen up—the nuclei are pink or purple, the glia cells have undergone active proliferation but give a pale pink oxychromatin reaction. The cells of the vago-accessorius nucleus show the characteristic changes even more markedly, most of the cells exhibit marked chromatolysis as well as the nuclear change. At a lower level the cells of Burdach's nucleus show marked nuclear changes and chromatolysis. Most of the cells of the olive show a granulo-pigmentary degeneration of the cytoplasm.

CASE VIII.

History.—R. R. B., aged 21. Educated at a Board School, where he showed considerable literary ability but little mathematical ability. He was employed on the staff of several papers, in a minor position, and recently had been reporting for a sporting newspaper. As a lad he was excitable, restless and masterful. There is no history of fits, and the family history is said to be good; the father is intelligent and the mother appears to be a sensible woman. About four months ago patient acquired ideas of persecution on the part of other reporters on the staff of the paper, believing that they accused him of being illegitimate and the son of a member of a noble family. He suffered from aural hallucinations. Later his delusions developed; he become possessed of the idea that his home was an immoral house, that the infirmary was full of prostitutes, and that he himself was suffering from syphilis. He thought loafers in the street made remarks about him as he passed, and he frequently challenged men to fight. He became apprehensive and depressed, believing that his only relief would be either by committing suicide or by strangling some other person. He had never made any attempt at suicide but had frequently made homicidal attempts.

Admitted to Bexley on May 4, 1908. Certified as a case of dementia præcox. In his manner he was stereotyped and somewhat aloof, but he recounted his tales clearly and briskly. Consciousness was clear, apprehension was satisfactory. Intelligent and active-minded; memory good. His moral habits appeared to be unexceptionable. He made many violent attacks upon both patients and staff, and was confined to bed in a single-room during the greater part of his residence here. On several days he was in a state of great excitement, intermissions on these days being few and short, but for the most part he was quiet; his outbursts have been suddenly aroused and as suddenly subsided. He made one attempt at suicide after admission. During the past six weeks he was as a rule depressed, dull, uncommunicative and somewhat retarded, and during the last fortnight he became very feeble and emaciated. On the night of August 8, 1908, he suddenly collapsed, and died at mid-day on the 9th.

Post-mortem Examination.—State of nutrition: Slight emaciation. Left lung: Deeply congested and shows patches of broncho-pneumonia. Heart: Slight recent endocarditis on edges of left mitral valve; few patches of atheroma of aorta just above the valves. Adrenals: Enlarged. Large intestines: Enlargement of the mucous follicles, but no dysenteric lesion. Other organs comparatively normal. No sign of tubercular disease.

Cause of Death.—Broncho-pneumonia.

Microscopic Examination of the Cortex Cerebri.—By Nissl and hæmatoxylin eosin methods. The superior frontal, ascending frontal, orbital, lingual, Broca, tip of inferior frontal examined. All these structures were without noteworthy vascular changes, but they all exhibited marked cellular changes and neuroglia cell proliferation. The cellular changes were as follows: Deficient cellular development in the frontal lobe (excepting ascending frontal). And in this region the cells show more marked changes; the columns of Meynert are disorganized, the cells have their dendrons broken off, few show any apical pressure, the cytoplasm is vaccuolated, doubtless due to the lipoid granules having dissolved out in the paraffin embedding process; the nuclei are swollen and the membrane often infolded or crenated. These changes are seen in the other regions of the cortex but to a less degree. The cells are generally shrunken and show a marked degree of chromatolysis. Nissl granules are seldom seen even in the large pyramids.

CASE IX.

History.—E. C. Patient was admitted to Hanwell Mental Hospital on May 27, 1914, at the age of 73, suffering from senile dementia. Her condition on admission was one of feebleness, restlessness and depression. She was evidently in a state of considerable trepidation as to her future, and believed that dogs were to eat her. She was incapable of coherent conversation and dirty in her habits. Her memory was grossly affected, and consequently no previous history could be obtained. Her sister was a patient in the same Mental Hospital. Her physical condition was comparatively normal for one so advanced in years. She was very feeble, but there was no definite paresis and she was blind in the right eye from cataract. During her short residence at Hanwell she was very childish and had no idea of time or space. She was incoherent and restless and was constantly folding and unfolding her clothes. She was depressed and had some vague ideas of unworthiness, calling herself a wicked woman. She is noted as being the subject of many senile tissue changes. On June 5, she became rapidly weaker with laboured breathing and failed to rally under cardiac stimulants. She continued in a semi-conscious condition and finally died at 9.45 p.m. of the same day.

Post-mortem Examination.—State of nutrition: Wasted. Brain: Marked wasting; sulci well marked; convolutions rounded and atrophied. Thyroid: Somewhat wasted. Right lung: Lower lobe showed some patches of consolidation. Left lung: Some bronchitis and emphysema. Heart: Walls thin and atrophied; slight atheroma of valves; pigmentary degeneration of muscle. Aorta and coronary arteries: Atheromatous. Liver: Some fatty degeneration. Kidneys: Granular. Renal artery: Atheromatous. Organs of generation: Very distinctly atrophied. Other organs showed no changes worthy of note.

Cause of Death.—Secondary terminal bronchitis and broncho-pneumonia; pigmentary degeneration of heart; chronic granular nephritis.

Microscopical Examination of Brain.—Hardened in formalin, four years. Toluidin blue and eosin. Frontal pole. Cerebellum. Motor area. Medulla.

Frozen sections stained by scharlach and hæmatoxylin show very marked lipoidal degeneration of cortical cells with much lipoid in the perivascular spaces. This condition is especially marked in the frontal region of the brain. The small vessels of the brain do not show degenerative changes. The nuclear changes are not so marked as in the case of dementia præcox. The nucleoli do not give an oxychromatin reaction. A piece of cerebellum embedded in paraffin with a piece of a case of dementia præcox and a piece of a case of tetanus. The three in one block were cut into sections of 10 microns and stained. A naked eye examination showed a deficiency in basophil staining of the cerebellum in the cases of dementia senilis and dementia præcox as compared with the case of tetanus.

CASE X.[1]

DEMENTIA PRÆCOX; MONORCHIDY; ATROPHY OF SUPRARENAL ON SAME SIDE.

(*This case, being of special interest, is given in full.*)

G., aged 27, admitted to Claybury Asylum, September 6, 1906. No. 4,846. Single, no occupation. First attack at the age of 23. Duration about ten days; form—subacute mania; previously not in an asylum.

Medical Certificate.—"He fancies people follow him about the streets to annoy him. At times he becomes very violent and noisy, and he then fancies he is surrounded by men and he fights with them and becomes very dangerous. Sometimes he is very sullen and morose and will neither speak nor answer questions."

For the past four years he has shown an incapacity for study, a vacant expression and was careless in his dress. On 30th ultimo he threatened an imaginary person with a knife and then ran into the street. He became apathetic and careless in his work, and has not slept well of late. He had a very good appetite. He was cheerful, but rarely speaks now. He was temperate and an abstainer. Had no religious excitement and was until recently a member of South Place Ethical Society. He has an undescended testicle, which some medical men think has retarded his brain development. and others disagree. He passed the Civil Service Examination, but low down on the list. He did clerical work, mostly in accountant's department of G.P.O. He was born and lived at Hackney.

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[1] I am indebted to Dr. Sano for the notes of this case.

Previous Medical History.—He was not certified, but under the care of Mr. Corner, of Harley Street, from December, 1904, till June, 1905, and under Dr. Forbes Winslow from August, 1905, to February, 1906. He was in different private homes. He was at home since February and had numerous attacks which with some exceptions were not beyond home management. As a rule he was quiet, idle, avoiding exertion. The attacks commence with threats and swearing. He had occasional attacks of asthma.

Family History.—Father: Paralysis agitans past four or five years. Parents not related. No consumption, no intemperance.

Examination of Patient.—Height, 5 ft. 10¼ in. Weight, 11 st. Hair fair; patches of hair on sacrum. Absence of left testicle. Palate very narrow and V-shaped. Teeth carious. Bowels regular. Urine 1,008, acid. Pupils equal, react well. He is pale, anæmic and flabby, but otherwise well covered and in fair condition. Knee-jerks present.

On Admission.—To my questions he answers in an off-hand jerky way. There is no retardation. He holds himself in a stereotyped way and shows tendencies to quaint mannerisms. His expression is dull. He seems a little irritable but is otherwise unemotional. He confesses he has no will power. He cannot concentrate on his work. He describes that, as a result of insomnia, he would get up and shout and that he could not help doing this, and suggests that it was an uncontrollable impulse. He has some insight into his case but it is too self centred. Memory and orientation fair.

Subsequent History of G.—September 12, 1906: He is the subject of subacute mania. Is sullen and disinclined to answer questions. Believes that there is a conspiracy to persecute him and that he is followed about by people who are plotting to injure him. He is in good health and condition.

December 10, 1906: He is apathetic and has no initiative. He shows no disposition to improvement.

August 3, 1907: He is suffering from dementia. He is dull and confused, lacks all interest in himself or surroundings, is apathetic and has no initiative. Stands for a time in one position. Speaks in a drawling monotone; yawns frequently. He is in fair health and bodily condition.

November 2, 1909: Struck attendant in the mouth causing swelling and cut on inner side of the lip (upper).

August 3, 1910: He hears imaginary voices speaking to him. At times he becomes irritable and excitable and makes homicidal attacks upon those who are near him.

1913: He is suffering from primary dementia.

August 15, 1914: Positive Widal. Transferred to hospital: no symptoms.

May 14, 1915: He gives evidence of aural hallucinations and responds to a female voice he frequently hears. He is impulsive at times.

February 10, 1916: He has been failing gradually for the last four days. Temperature subnormal. Pulse weak and irregular. Died on this date.

Post-mortem.—Cause of death, broncho-pneumonia.

Left suprarenal: 3·5 grm.; very little lipoid cortex. Right suprarenal: 6·5 grm.; very little lipoid cortex (*vide* figs. 1 and 2).

Right testicle: 12 grm. Left absent. No spermatozoa in testes or vesicula seminalis. The left vesicula seminalis was very large as compared with the right: both contained a thin clear gelatinous fluid but no spermatozoa could be found by dark ground illumination. Testis: No spermatozoa found, tunica albuginea thickened, and the testis presented a whitish appearance. Examined with hand lens: There is excess of interstitial tissue. The white tubules are shrunken in some places, and not so distinctly seen as in others. There is clearly an atrophic process.

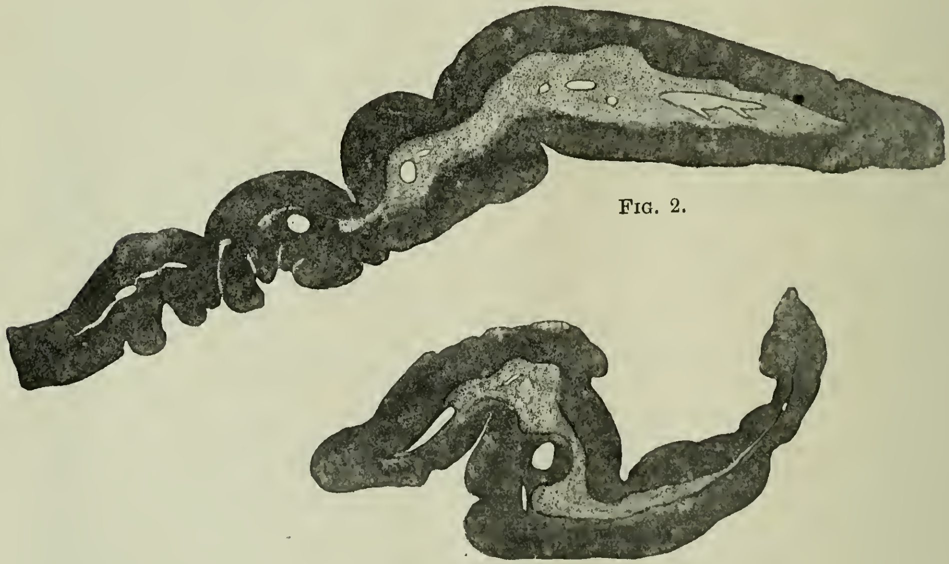

FIG. 2.

FIG. 1.

Sections of (1) left suprarenal, and (2) right suprarenal gland, to show comparative size. (Magnification 12.)

Microscopical examination shows a complete atrophic process of a very advanced nature; most of the tubules greatly thickened basement membrane with clear unstained vesicles filling the lumen. Others show cells filling the lumen, similar to those found in the fœtus. Nowhere can active mitosis be seen, nor any evidence of any stage of spermatogenesis (*vide* Plate VI, figs. 1 and 2). The appearances coincide with those found in other advanced cases of dementia præcox.

PLATE VI.

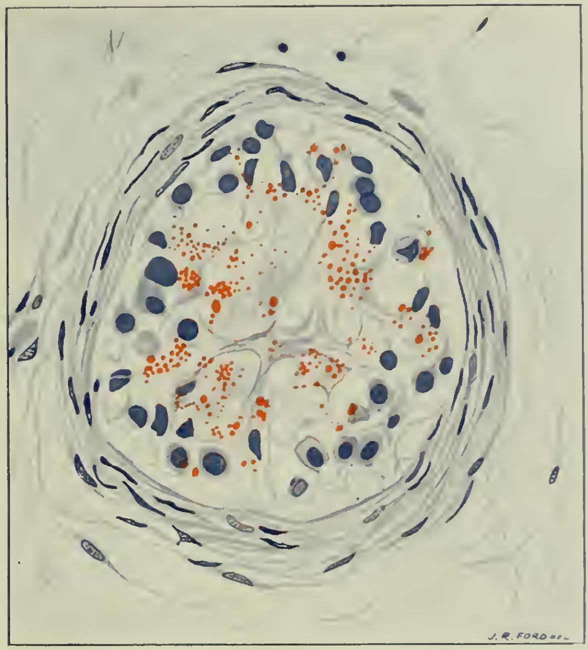

FIG. 2.

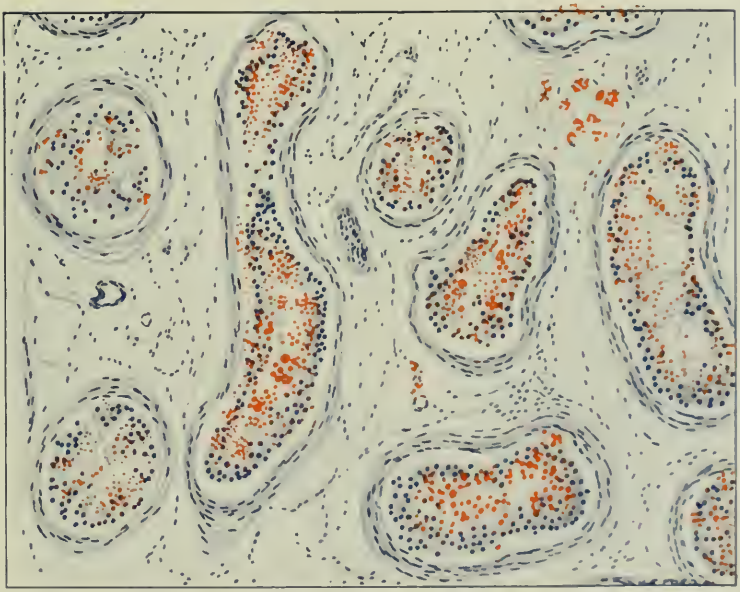

FIG. 1.

Third stage of testicular regressive atrophy. Section of right testis cut by freezing microtome after hardening in formalin. Stained with scharlach R. and hæmatoxylin.

Fig. 1: There is a great increase of the interstitial fibrous tissue; no interstitial cells are seen. The tubules are shrunken; the basement membrane is greatly thickened; there is no sign of spermatogenesis; the lipoid granules are only present in the interior of the tubule. (Magnification 115.)

Fig. 2: A section of tubule magnified 420 times. The basement membrane is greatly thickened; there is no sign of spermatozoa, spermatids, or spermatocytes; the nuclei of the spermatogenic and Sertoli cells present show no sign of mitosis. The lipoid granules are coarse and deeply red stained, and are contained in the Sertoli cells or between them in the sustentacular framework. They are not the normal fine orange-stained granules, and are probably a product of fatty degeneration of the spermatic cells.

Heidenhain stain: Tubules all show atrophy, but there is varying degree in the extent of the atrophy; there is no spermatogenesis observable in any of the tubules; in some the epithelium has entirely disappeared, leaving only the thickened basement membrane and hyaline rudiments of the epithelial cells; in other tubes there are epithelial cells, but none show active mitosis. The cells of Sertoli are seen distinctly in many of the tubules, as they form the only remains of the epithelial lining cells. No evidence of spermatocytes or spermatogenesis is observable in any of the tubules. Interstitial tissue (stained with hæmatoxylin and eosin) is increased but there is very little evidence of the existence of the interstitial cells of Leydig. Hæmatoxylin and scharlach stain shows very little interstitial lipoid; there is a considerable amount of dark red coarse lipoid granule substance within the tubules, many of these are in the syncytial cells of Sertoli. Scharlach stain, with a low power, shows that nearly all the lipoid is within the whole extent of tubules, but as if resulting from fatty degeneration of the epithelial cells. There is comparatively little in the interstitial tissue.

Suprarenal cortex stained by the same method shows a great deficiency of lipoid, in some places quite absent, in others in relatively small scattered patches.

Examination of several sections permits the conclusion that there is a marked deficiency of the cortical lipoid substance.

Other cases of dementia præcox showed a diminution of the cortex, and this case is of interest in the fact that the adrenal gland on the side of the absent testicle weighed one half that of the opposite side. The suprarenal gland is developed from the genital ridge, and the cause of the absence of the testis and the very small suprarenal gland on that side was doubtless an embryonic deficiency. There are many facts which show a correlation between the adrenal cortex and the sexual functions which I will briefly summarize.

THE INFLUENCE OF THE ADRENAL CORTEX ON THE GENITAL FUNCTION.

The cortex adrenalis is enlarged during pregnancy and is small in cases of deficient sexual development, and the fact that changes have been described (in the guinea-pig) by Kölmer in the cortical cells accompanying the phases of the œstrous cycle seem to afford evidence of some relationship between the functions of the cortex and the generative glands, but whether this is direct or operates through other ductless glands—such as the thyroid and pituitary—is not known. The occurrence of a connexion between the development of the sexual organs and that of the suprarenal cortex has been especially insisted upon by Glynn, who is of opinion that the cortex is concerned with the differentiation and growth of the sexual cells. Bulloch and others have recorded cases of adenoma of the adrenal cortex and precocious sexuality.

Analysis of the suprarenals of the pig by Biedl yielded: 74·61 per cent. H_2O, 25·39 per cent. dry residue; of the latter 61·12 per cent. consisted of proteins and 38·8 per cent. lipoids; as this included the medulla the proportion of lipoids must be very high in the cortex.

The darkly refractile lipoid contents are composed of lecithin and cholesterol, the latter in the form of esters. The suggestion that the cortex of the suprarenals may be a seat of manufacture of lipoids of the body and related to the formation of the myelin of the nervous system, is supported by the fact that there is a large development of lipoid in the cortex during the period of myelination of the nerve fibres of the brain which development is absent in anencephalous monsters. The superadded part of the suprarenal cortex, according to Elliott and Armour, in the fœtus does not contain darkly refracting lipoids characteristic of the ordinary cells.

Swale Vincent, in a very interesting paper upon "The Experimental and Clinical Evidence as to the Influence exerted by the Adrenal Bodies upon the Genital System," describes feeding experiments of animals with suprarenal gland and the stimulating influence upon the growth and activity of the testes carried out by R. G. and A. D. Hoskins upon white rats. Other glands did not show hypertrophy. The authors conclude that the testicular hypertrophy is due to the cortical portion of the gland. Swale Vincent is himself conducting experiments in which certain possible sources of error will be removed—viz., he is using only the fresh cortex of the adrenal gland. He points out that drying may de-grease the material and alter or destroy the lipoids.

BIBLIOGRAPHY.

ALZHEIMER. *Centralbl. f. Nervenheilk.*, 1900, p. 296.

BATTELLI, F., und STERN, L. "Die Katalase," *Ergebnisse der Physiol.*, 1910, x.

BAYLISS, W. H. "General Physiology," 2nd ed., 1918.

L'HERMITTE. "Les Démences Précoses," *Traité internat. de Psych.*, 1911.

KRÄPELIN. "Dementia Præcox and Paraphrenia," Translations from the eighth German edition of the "Text-book of Psychiatry," iii, part 2, Section in the "Endogenous Dementias."

KOCH, WALDEMAR, and MANN, SYDNEY. "A Chemical Study of the Brain in Healthy and Diseased Conditions, with especial reference to Dementia Præcox," *Arch. of Neur. and Psych.*, iv.

MARINESCO, G. "Recherches Histologiques sur les Oxydases," *Compt. rend. Soc. de Biol.*, 1919, lxxxii, No. 2.

MOTT, Sir FREDERICK W. "Normal and Morbid Conditions of the Testes from Birth to Old Age in One Hundred Asylum and Hospital Cases," *Brit. Med. Journ.*, 1919, ii, pp. 655, 698, 737; "The Cerebro-spinal Fluid," Oliver Sharpey Lectures, Royal College of Physicians, 1910.

PIGHINI, GIACOMO. "Chemische und biochemische Untersuchungen über das Nervensystem unter normalen und pathologischen Bedingungen: VII Mitteilung, ueber die Autolyse des Nervengewebes 1, von Carlo Fontanesi," *Biochem. Zeitschr.*, 1914, lxiii, p. 337.

Idem. "The Organic Metabolism in Dementia Præcox," *Arch. of Neur. and Psych.*, iv.

PIGHINI, G., und BARBIERI, P. "Chemische und histochemische Untersuchungen über die Lipoiden Abbaustoffe des Gehirns bei Progressive Paralyse."

RENAUDIÉ, HENRI. "Dementia Præcox Studies: A Contribution to the Study of the Pathological Anatomy of Dementia Præcox."

WADA. *Obersteiner's Arbeiten*, xviii, p. 313.

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Section of Psychiatry.

President—Dr. WILLIAM McDOUGALL, F.R.S.

Psycho-analysis of a Case of Early Paranoid Dementia.[1]

By DAVID FORSYTH, M.D., D.Sc., F.R.C.P.

I.

THE subject of this paper was a boy, Claude, rather more than 7½ years old, who was brought to me on account of his troublesome and eccentric behaviour. Except as a baby he had always been difficult to manage by either of his parents, though only in the last eighteen months or so had he become noticeably peculiar in his ways. But about this time he grew moody and depressed, often refusing to speak. In these moods, which became more frequent, never more than a day or two passing without at least one, he looked utterly miserable and would cry without any apparent cause; or he would be very cross, shouting at anyone who approached him, and arguing everything; or he went about in a kind of dream and had to be told just what to do: "Now open the door. Now go out. Now shut the door," &c. He became liable to sudden outbursts of violent temper when he would throw things about or kick the first person who came near; for which reason he could not be trusted alone with other children. On one occasion he was quietly watching his mother feed her cage birds when suddenly he ran at her, gave her a heavy thump and shouted "What do you mean by knocking me over?"

At other times he would pass into moods of quite another kind in which he withdrew into himself, oblivious of everyone and everything around. His stare was vacant but he could be heard talking to himself or even chattering, smiling often and apparently quite happy. To disturb him in such a mood was usually to rouse his keen resentment.

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[1] At a meeting of the Section, held June 1, 1920.

It was noticed that he became increasingly suspicious, at first of his friends and acquaintances, but finally of strangers; people speaking together in the street were telling tales about him, if they laughed it was at him; and finally he asserted that they were bent on injuring him somehow. For many months he had never said a nice thing about anyone, or done a kind action; he would never admit that he cared for anybody—at the most he would say he did not know—but he expressed his dislike openly and freely; "I hate you" came from him several times a day, and he had even gone up to strangers and shouted "I hate you" at them.

He was said to have always been a jealous child, but this side of him had so grown in recent months that he could never associate for long with other children without quarrelling. Provided they allowed him the leading place he was amiable enough, but once he was asked to stand aside to give one of them a turn he quarrelled. Playing cricket, for example, he usually contrived to be the first to bat, but as soon as his innings was brought to an end and he was called on to take a subordinate place in the field he would lose his temper and as often as not would attack his playmates.

In addition to these singularities which made the boy quite unmanageable by those responsible for him, he developed many nervous tricks. He fidgetted incessantly, made grimaces and twitched his face. He ground his teeth, sucked his fingers, bit his nails, rubbed his chin, plucked at his under-lip and ears, scratched his nose, picked at his eyelids (so giving himself a succession of styes) and fiddled with his jersey. He had a little cough or, rather, grunt, and frequently muttered to himself. In his sleep he groaned often and scratched his legs till they bled. These habits which on his bad days never ceased, made people look round at him in the street, the more so as he began to tumble about as he walked, knocking into his mother or governess as though he really wanted to trip them up. He became self-conscious, gave an impression of worrying intensely without, however, admitting it, and often expressed a dislike of changing his ways "because that would be silly and babyish." He bore really severe pain without a murmur and never acknowledged being tired or feeling ill. Nevertheless he was always pleased to be kept in bed by a slight illness; on these occasions he was happy and contended, his nervous habits completely subsided, and when the day came for him to get about again, he would cry and beg to be left as he was.

Such was Claude's condition when I first saw him. He was obviously a clever boy—he read Dickens and enjoyed it; he had taught himself his letters when a year old—and yet in some ways he was a baby—e.g., he always had his food cut up for him and he could not tie his boots or dress himself. He was a firstborn child, a sister, Maisie, being born sixteen months after him, and then two brothers, John and Dennis, 2½ years and 4½ years respectively younger than Claude. The father was well educated and clever, with strict religious views of perhaps a narrow kind; and, as he acknowledged, with some peculiar traits which had led people in the street to point at him as mad—at any rate he believed he had caught this word as he passed. He was too busy at his work to have time to spare for his children, and at week-ends his leisure hours were given to a boys' club which took him away from home.

The mother, equally orthodox, had been attached to Claude so long as he was the only child, but when his sister was born she found that a girl baby appealed to her altogether more strongly, and she transferred practically all her affection to Maisie. She explained this by saying that Claude, even before he was 2 years old, became naughty and difficult, and for this reason was no longer lovable. The true explanation, of course, lay in another direction and will be discussed later. Suffice it to say here that Claude, after an undisputed enjoyment of his mother's love for nearly two years, found himself relegated to second place—and a poor one at that—and later to third and fourth places as the family (and his naughtiness) grew. Yet another point in the boy's upbringing must be mentioned. Both parents were reserved to the last degree in speaking of sexual matters, and when Claude asked for enlightment on any topic of the sort which at the time perplexed him, it was refused out-of-hand and the child was reproved for being nasty-minded. This secretiveness on the part of the parents inevitably spread to subjects other than sexual as the next point in the history shows.

When Maisie was 4½ (Claude being nearly 6) she was taken suddenly ill and within a few hours an operation became imperative. Claude was at once packed off to the grandparents. Maisie was operated on that night but never recovered consciousness and died before morning. After the funeral Claude was brought home again, the parents having decided—*incredibile dictu*—to say nothing to him as to what had happened. He returned, ran up to the nursery (this from his own account) but no Maisie was there. He looked round and saw his own toys about, but none of Maisie's. He felt frightened and went downstairs again to ask, but the look on his parents' faces stopped him. He said nothing. They said nothing. And from that day until a year later when I talked to him Maisie's name had never been mentioned in his hearing. At this point in the history two facts need bringing together. Maisie had died nearly two years ago ; the boy's behaviour had first grown peculiar about eighteen months ago. It could not be doubted that the two were directly related.

II.

During the months that he was coming to see me, Claude, for reasons which will be more obvious later, lived, not with his parents, but at the home of his governess, Miss P. She was unusually intelligent and understanding with children, and at my request kept daily notes of the boy's behaviour. These were of great help to me throughout the progress of the case, though, on account of his intense suspiciousness, they had to be conveyed to me by stealth. Some extracts from this diary will bring out still more clearly the main points in Claude's psychology.

First with regard to his attitude towards strangers or new acquaintances. With these, if grown up, he always became excited and ended by getting rude. If he was introduced to a circle of child playmates he would be friendly at first, but within a day or two was sure to quarrel violently. He seemed to take it for granted that no one could like him once they came to know him. "Of course I don't expect anyone to love me. Why should they?" he once said. Another time he had been taken to task for his rudeness to some visitors. "Well," he replied, "everybody hates me so I might as well be rude." "But people can't like you if you are rude." "What is that to me? . . . . I want everybody to be killed that I don't like. It's not wicked of me because I can't help it."

He would frequently express himself on these lines: "I don't trust anybody, they all tell me such lies." "Everything and everybody makes me cross." "People who laugh at me shall be kicked." "Why are you staring at me? Everybody stares at me." "Other people want to kill people as well as me." One night he called to Miss P. from his bed, and when she came near he sprang up and hit her with all his might. "That's what I've wanted to do to you all day. . . . . Sometimes in the night I sit up and watch you asleep, and I feel I want to hurt you. But it will be worse than thumping. I couldn't kill you, could I? If I could I would. I hate you and you hate me too." A week previously he had told her he would like to put his fret-saw in her bed, "and if it hurt you I should be glad. . . . I simply love doing wicked things, only I can't think of anything wicked enough." At another time, "I'm thinking now I want to bite you."

His jealousy was provoked on every possible occasion. A friend of Miss P.'s came to stay; Claude made faces at her every time she looked his way, and was rude and cross all day. At bedtime he said, "I hate——. How can you love her and me too? All day I've wanted to kill her and you as well, because you love her more than me. . . . But I will be good when she is gone." On another similar occasion he volunteered, "I'm always jealous when people come because you love them better than me and that makes me hate you."

For the most part he showed little fear, though he was once strangely terrified after turning on the bath taps and finding he might not be able to turn them off fast enough to prevent the water running over. A constant fear he had was of being left behind when he was out-of-doors or in a strange house, and he once or twice spoke of being afraid of coming to see me the day after he had told me a specially big lie. A more significant example is the following. Miss P. went to help him off with his jersey. He flinched and started away. "I thought you were going to hit me," he said. "Everybody hits me. If I stay in this house everyone in it will hit me. You said you wanted to bang me on the head with a poker." Another time he said, "Whenever anyone puts out their hand I think they are going to hit me."

His attitude towards his parents was significant. During the months he was away from home staying with his governess he practically never referred to either father or mother. Only on four occasions did he break this silence. Once, the day after his mother had been to tea, he began, à propos of nothing, "Mother tells me such lies. She called me her pretty boy and I knew she can't mean it because once she said I was too ugly to kiss (this was when his eye was bandaged on account of a stye). Am I ugly? Come here and look quite close." "No, you are not at all ugly." "I don't believe you; its too dark to see properly; you must look again in the morning." Another time, "Mother doesn't love me a bit. I asked her who she liked best, and she said first Dennis, then Maisie, and then John, and me last. . . . But I don't care. I want to be ugly and stupid and no one to like me. That is the best way—ugly as ugly, and stupid as stupid." Later in the treatment, when he was beginning to understand his feelings, he admitted, "I used to worry about mother, because I thought she hated me. . . . I feel miserable because often

I've tried to think how I could get all of them out of the way and there would be only me." But a few days later his old feelings came over him again. He had been to an outdoor inspection of the boys' club by his father. He was very "nervy" all the time, looked abjectly wretched, and could not find a word to say to his father, even when he was told he would shortly be going away for a holiday with him and his mother. That night he protested, "Why should I go away alone with Mummy and Daddy? They'll only be nasty to me as they were before, and then I shall be nasty too. . . . I hate Daddy's club boys. Why shouldn't I kill them? . . . I don't want Daddy to love them; he's to love only me."

Just a couple more extracts from the diary. "He (the doctor) says I'm clever, but he doesn't really think I am. Do you think I shall be when I grow up? No, you'd be glad if I was quite stupid, because you don't like me. You say you do but I don't believe you. Everyone tells me such lies." Again, he had spent a week-end with friends, and was told that his hostess so liked him that she wanted him to come again. He looked incredulous. "Did she say that? When did she?" Then he burst into tears and sobbed out, "I like her; I do like her." This was the only occasion for many months at least that he had admitted liking anyone. "It's silly and babyish saying you like anyone," he would sometimes declare.

It should be added that very occasionally there were hallucinations of hearing. For example, at tea with his mother, when no one had been speaking, he shouted at her, "You're not to scream at me like that. I'm not deaf if you are." An hallucination of the same kind seems to have been responsible for an excited outburst, again à propos of nothing, "I can't sing. You know I can't sing. I tell you I can't sing. Everybody knows I can't sing."

III.

From the particulars already given it is not difficult to recognize something of Claude's mental affliction. He was a boy who, except on rare occasions, expressed no affection for anyone, not even his mother or father, and would defend himself on this score by protesting, "It's silly saying you like anyone." On the other hand, he freely and repeatedly showed hatred of everybody with whom he came in contact. He was convinced that others hated him and this he held to be the cause of his own hostility. Latterly he had come to think that when he saw people talking or laughing together in the street, he was the subject of their conversation, and still more recently that they were planning injury to him (delusions of reference and persecution). Further, he was a boy with strong jealousy, which he freely indulged. These, of course, are features of paranoia. This state, however, is often characterized by a delusion of a third kind—namely, grandeur—the patient holding exalted ideas of his position, powers, &c. In Claude no such delusion was found, and though, as will been seen later, his imagination was rich in fantasies, self-glorification was not a feature of them; on the contrary, he seemed to indulge few or none of the fantasies of ambition which fill so large a part of the day-dreams of normal boys of his age. This point will be returned to later.

Paranoia, as has been shown by Freud and, after him, by Ferenczi and others,[1] takes its origin in the homosexual component of the sexual impulse. The homosexual wish-fantasy to love the man is or becomes unduly strong either (1) because of unhappy social relations with the man, the homosexual libido thus pent up inducing a psychical tension which calls for relief; (2) because of disappointment in the woman, the libido which should have flowed along heterosexual channels being diverted in a homosexual direction; or (3) because of an individual powerfulness of the libido all round. No matter which of these factors is operative, and provided the homosexual libido is afforded no adequate outlet, paranoia in its various forms represents all the possible ways of denying the homosexual need as expressed by the statement, "I (a man) love him (a man)." (*a*) "I do not *love* him, I *hate* him." To the paranoic this hate is consciously unacceptable, is repressed and projected into the mind of the former loved one—"he hates (persecutes) me"; in this way arises the delusion of persecution. (*b*) "I do not love *him*, I love *her*." This denial, which in adults leads to heterosexual love affairs, is not of present interest. (*c*) "*I* do not love him, *she* (? he, in the case of children) loves him." Herein originates the delusion of jealousy. (*d*) "I love no one but myself." The libido is now directed to the ego, an over-valuation of self results and from this develops the delusion of grandeur.

In Claude's case we recognize the paranoic state as yet not wholly developed and still showing at times a conscious recognition of the original homosexual wish, "I love him (my father)." Except in his earliest years he received little or no attention from his father: the normal outlet for a boy's homosexual affection was closed to him.[1] Yet this need was made all the greater in his case by the withdrawal of his mother's love, from which arose (as will be more apparent from his own story given below) a bitter disappointment in the woman of all women. The consequences of this heterosexual denial will be discussed later; at present it is enough to recognize in it the second of Freud's factors strengthening the homosexual libido. Thus placed, Claude evinced his paranoic tendency by becoming troublesome and unmanageable in relation to his father, and in course of time showed plainly his belief that his father disliked him. "I don't expect anyone to love me," "Everybody hates me," "He'll only be nasty to me as he was before."

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[1] *Jahrb. f. Psychoanalytische u. Psychopathologische Forschungen*, 1911, iii, pt. 1.

He was strikingly ill at ease with him, and never mentioned him during several months when he was away from home. Yet the direct "I hate him" never came to expression in so many words (it is significant that I found very much greater difficulty in getting him to speak of his father than of his mother), but was openly applied to the world in general—i.e., it was repressed and projected; and, I need hardly add, his antagonism to myself was always present and at times intense—more than once he would have struck me had he dared.

Similarly his jealousy where his father was concerned was a matter of general comment; however, so far as I could elicit, he was not jealous of his father liking his mother (his father, however, was not a demonstrative husband), but of his attention to other boys, and especially to the club boys—"I hate them. Why shouldn't I kill them." And, of course, the occasion of this outburst wrung from him also the first direct admission of his real want, "He is to love only me." It is right to assume, however, especially in view of his expressed unwillingness to own to liking anyone, that this wish for his father's love was still often in his mind, consciously and alternating with its repressed, projected form "he hates me"

IV.

We have already noted the very serious injury done to him in his second year when his mother, who until then gave him her love, withdrew this and transferred it to Maisie. The fuller effects of this emotional trauma can best be seen in the boy's own story as elicited by analysis. He recalled how his mother often kissed him until he was 2 years old, " when I began to be naughty," and she unkind. Thereafter she gave up kissing him except sometimes at night, and often he saw how he was the only one of her children she omitted to kiss—" But what is the use of a kiss ? " he added bitterly. She never took him on her lap, though the other three were favoured in this way, and he recalled how once when she came to the nursery and sat down, Maisie ran to her from one side, climbed on her knee and was kissed, but when he tried to do the same from the other side his mother pushed him away ; this made him very unhappy. Then again, his mother refused to kiss him when his eye was bandaged—" because I was ugly " ; and he cried all that evening. He could not remember her ever saying that she cared for him (I had occasion later to mention this to his mother and she did not deny it), and he readily recalled the many times when she had hit, kicked or bit him, though, as he admitted, only as a punishment when he had done the same to his smaller sister or brothers. Similarly he kept a lively memory for the innumerable occasions when his various nurses had slapped or pinched him. Once he listened outside the dining-room door and heard himself discussed ; he thought he heard his mother say she did not like him as much as the other children.

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[1] The father's belief that people had called him mad as he passed in the street may or may not have been well-founded. If imagined, it would point to a paranoic tendency, and himself as the subject of repressed homosexuality. He would thereby be peculiarly unable to meet his boy's demand for affection. His interest in boys' clubs may also be recalled in this connexion.

He remembered only three or four occasions when she had been kind and taken him for outings. He was sure she did not care for him, and this was made the more unbearable because he had heard her at times tell all the others that she liked them. He had been jealous of them as long as he could remember—and he added in a sudden burst, " I tell you what it is, I am jealous of the whole lot. . . . I want mother to like me." When I interposed that this did not seem to me altogether impossible he demanded sharply, " How can you make her like me ? I want people to like me, but not to say so—it's babyish," and tears trickled down his face.

Two things are plain in this account. Unhappiness (disappointment, jealousy, hate) first came into his life before he was 2 years : disappointment in the mother who showed herself false in her affection, jealousy and hate of his sister—and hate in children does not stop short of death-wishes. In his own eyes Claude must have thought himself justified in his jealousy ; but to the mother this was naughtiness to be checked by punishments—i.e., Claude was himself responsible for his behaviour, and was blameworthy ; but this again would be to Claude the innocent found guilty—and his sense of injustice would henceforth be sharpened to a degree. Moreover the outflow of his love in the direction of his mother being checked, it needs must have sought some other channel—either by transference to another person, or by regression to an earlier, lower level of attachment, or by both. As luck would have it, his nurse at the time (as did all his nurses subsequently) saw him through his mother's glasses as a difficult, tiresome child. Only the father remained. An increased homosexual need was felt, with results which have already been recognized—further disappointment, jealousy, hate and a heightened sensitiveness to injustice. In point of fact, however, one part of the heterosexual need was undoubtedly dealt with by regression, though the evidence for this must be reserved till later. That it should have been utilized in this way permits us to assume that the weak link in Claude's psychical development, the predisposition to the psychosis, lay deeper and earlier than his heterosexuality. This point will be taken up later in the light of further details of his history.

The second thing that is apparent in his account is a fixation of his affection to the period preceding Maisie's birth. This shows itself at many points in his behaviour. Thus, it is babyish for people to say they like you—this indeed was an experience limited to his babyhood. Again, it is babyish to change one's ways (i.e., in order to please another)—this he did only in the days when his mother loved him. Moreover he was still babyish in some of his ways. Also he was constantly afraid of being left behind when out of doors or in strange houses (a common fear of little children of being parted from their mothers). Most significant of all, to confine him to bed on account of slight illness was to convert him into a happy contented child again and his nervous tricks ceased: this repeated the situation in which, in his cot, his loving mother tended him, and him only. Contrast this with the little incident when, unprovoked, he suddenly struck his mother as she fed her pet birds. Is it impossible here to catch the echo of a day when his mother had called him *her* "little bird"? And his angry shout, "What do you mean by knocking me over?" is not so inexplicable when we remember that it was indeed his mother who first knocked him over—"off his perch."

The jealousy "of the whole lot" which he came openly to avow, though becoming in course of time directed over a wide field, continued most intense in the direction in which it was first aroused, namely, of Maisie; and, as we shall now see (again following his own account), his jealousy of her was at the root of the striking aggravation of his trouble subsequent to her death. When he was brought home after her funeral and neither dared to ask nor was told what had happened, he fell back on his own speculations. He soon came to the conclusion that she had died, or rather, had been killed. Who could have killed her? Presumably someone who knew her, someone who visited at the house. Whoever he was his mother would certainly have slain him in revenge; he would therefore watch to see which of their friends had fallen out of the home circle. But no one dropped out, and before long the boy was driven to another explanation which, likely enough, had been at the back of his mind all the time. It was this. He remembered that many a time when his jealousy had been roused against Maisie, he had wished she might meet with an accident which would remove her from his path; and he mentioned a fantasy which had occasionally come to him—of his mother attending to the nursery fire, dropping the poker on Maisie's head and killing her.[1] Sometimes, when he had been particularly jealous, his wish that Maisie might die had been intense. And when at last she died, the memory of this oft-indulged wish recurred to him, and with it the reflection that if a thing is wished hard enough it comes true. It was his wishing that had killed her. He felt secretly guilty of her death. It was only another step to project this belief into the minds of others.

Nevertheless another train of thought shaped itself—that although he had entertained this wish, he never did intend and never would have intended that it should be carried into effect. He felt he had been taken, as it were, at his word, when he had never expected this much. He had been tricked, deceived, put in the wrong; and really he was innocent. But all this is precisely what had befallen him years before, after Maisie was born. Then it was only his mother and father who blamed him; now it was the whole world. And even the death-wish for which the world held him guilty could be laid at the door of those who, by first making him jealous, were responsible for the wish.

It was this association between present and past, this return of the old situation in the new, that lent to Maisie's death so powerful an effect as to precipitate the outbreak of Claude's psychosis. With no one with whom he could discuss his trouble, he took to brooding, and became depressed and miserable. Always disputing in his mind his guiltiness of Maisie's death, he developed the habit of arguing everything that was said to him. At the same time it is impossible that the child could have ever been in sorer need of the comfort and love which his parents might have given. Homosexually and heterosexually his needs were intensified, and in either case repression was the course followed. On the homosexual side there inevitably resulted an intensification of hate which was projected, not on to the father alone, but on to the whole world, who, knowing his guilty secret, could only hate him. Hence followed the delusion of persecution; and this became the excuse for the outbursts of anger and the open expression of hate in the street. Simultaneously, the heightened but unsatisfied heterosexual need contributed to the enhanced jealousy which was now displayed (this was the period when he struck his mother for feeding the birds). It may be surmised, however, that the fate of this component of the libido is to be traced, partly in the nervous tricks which developed, and partly in those other happy moods into which he withdrew and from which he resented to be disturbed. The key to the whole illness seemed to lie with the fantasies associated with these moods. They must therefore claim our next attention.

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[1] From this fantasy is evidently derived the charge he launched against Miss P. of wanting to bang him over the head with a poker.

V.

It will be recalled that in these moods, which were distinct from his moods of depression, Claude became abstracted, stared into vacancy, talked to himself, smiled often and appeared happy and contented. Miss P. had many times tried to get him to say what he was thinking about, but he always refused to tell; nevertheless she was satisfied they were happy thoughts because he seemed to enjoy them. If interrupted in them he would exclaim angrily, "Now you have spoilt my nice thoughts." On one occasion, when asked what he had been thinking about, he rejoined, "I shan't tell you, but they were bad, horrid thoughts"; and once he explained, "I am always thinking bad, wicked things. I like thinking them; they are lovely."

It was to these fantasies that I necessarily wished to gain admission, but for some months he would tell me not a word about them, though when at last his resistance was overcome he talked freely enough. It then appeared that in these pleasant moods he was in the habit of reviving memories of real events which had happened during the previous few years, and in which a part had been played by a child rather older than himself, called Leo, who lived in a house with a large garden, part of which was a wilderness remote from prying adult eyes.

I remembered Claude mentioning this boy's name twice a long time before, and that on each occasion he blushed deeply and turned to another topic. He now gave many reminiscences of his association with Leo and others, which may be summarized as follows :—

He had sometimes slept with Leo, who taught him self-abuse, he in his turn instructing Maisie. Leo also taught him to pull at his underlip and at the lobes of his ears; to eat nasal mucus, ear-wax, toe-nail clippings; to stuff nasal mucus into his ear, and ear-wax into his nose. They amused themselves at different times by putting under their finger-nails ear-wax, nasal mucus, phlegm, dirt from between their toes, fæces and snow. In bed together they would "play at animals," biting, kicking and smelling each other, and each other's excretions; and, after locking the door, "milking" each other's genitals.

With other children, including Maisie and another little girl, they would, when in Leo's garden, expose themselves to each other, comparing the genitals of the boys and girls, and examining their navels. They watched each other urinate, and made contact between their genitals, at the same time urinating over each other. They played many games associated with urination—e.g., all helping to fill an empty can with urine, which they then used to wash down a summer-house; or urinating together to make "a pretty pattern like fireworks"; or running round the garden urinating, the winner being the one who went farthest before the stream failed; or frightening cats and birds by syringing them with urine.

Similarly, Claude and his companions contrived many games with fæces. By withholding a motion when they were sent to the lavatory, and later defæcating in the wilderness, they could always ensure a supply of excrement, and with it, mixed or not with water or urine, they painted doors, palings, a toy engine, &c., buried it and dug it up again, or used it as mortar on the garden wall. They would shape it as fireworks—Catherine-wheels and rockets especially—and once they moulded a pudding on which, after setting it on a cardboard plate with a sprig of holly as decoration, they urinated.

Indeed, Claude's interest in fæces surpassed all his other interests. He said that fæces, like lavatories, had a "lovely smell"; he had eaten fæces, drunk it mixed with urine, smeared his thighs with it, extracted it with his finger *per rectum*, and thrust penholders into his own and Leo's bowels. In a fantasy he urinated into the bowel of another child and watched the urine being evacuated. He asserted—though this may have been fancy also—that he had inserted his own fæces into other children. Often speculating how a motion of the bowel came about, he constructed a fantasy of two little men in his stomach, of whom the one in the upper part was furnished with a cricket-bat or a spoon to stir up the fæces and shovel it on to the other lower down, who worked with a hooked stick to drag it towards the anus. From time to time the lower manikin would throw a lump of fæces to his fellow, who ate it as his ration. Into this fantasy Claude also introduced his explanation of the rôle of the umbilicus, the true function of which was unknown to him. He supposed that the navel was the head of a nail driven into the body, and was intended to mark the upper boundary of the lower man's territory, and prevent him getting his hook too high.

Not only did Claude recall these and many other similar memories with evident pleasure—he brightened up, laughed and talked with animation—but he made it clear that in his abstracted, happy moods these were the thoughts, at once "horrid" and "lovely," filling his imagination, and that he re-lived these scenes with real enjoyment. His onanistic fantasies were as a rule of the same kind, and it is only probable that his dreams were built up of identical material; and though he brought very few and allowed none to be analysed, one of them was about *fireworks* (including Catherine-wheels and rockets), and *peacocks*, and another was the following: "With Leo in his garden. We were carrying milk-cans, one full of urine, the other of fæces.[1] In the cherry-orchard we were stoning cherries, filling them with urine and plugging the holes with fæces. Then we held them to our mouths, squeezing them, and the urine came bubbling out into our mouths." His laconic comment on the dream was "I *did* enjoy that dream." It should be added that cherries were his favourite fruit.

VI.

These many details of Claude's spontaneous behaviour among children of his own age, when free from observation by adults, and of his fantasies, prove his interest to have been strongly centred in his excretory functions, which still afforded him keen pleasure; still afforded, it must be said, because these, together with the function of ingestion, are the outstanding interests, as Freud was the first to recognize, of young infants; and to this early stage of psychical development, in which pleasurable sensations are derived directly from the infant's own body, he has applied the now well-known name of auto-erotism. Except, perhaps, at the very outset of life, auto-erotic sensations are produced over a wider area than bowel and bladder alone, and are provoked by stimulation of any part of the skin, especially the more sensitive regions, and particularly the genitals. Claude's strong auto-erotism is proved not only by the interest in the excretory functions (parenthetically, he was a hearty eater), but by masturbation and a whole series of nervous tricks played on different parts of his body. Here, too, falls into its place that incident when terror, once only in several months, seized him as he saw the water from the bath-taps threatening to flood the room. The bladder-erotism thus significantly disclosed may of course be recognized commonly enough in very young children by their interest in taps, &c.[1] But Claude was nearly 8 years old, and still absorbed in auto-erotic pleasures as a child of 2 or 3 years. This needs accounting for.

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[1] He had his own names for both excretions, but they are not of present interest.

It is commonly held that when psychical development is on normal lines, auto-erotism proceeds through narcissism (love of self) to the stage of "love of an object." It is impossible, however, to study young infants without recognizing object-love (i.e., of the mother) at a period when auto-erotism is as yet not fully developed and narcissism has still to make its appearance. Object-love, therefore, may stand directly and not mediately in relation to auto-erotism, and I have attempted elsewhere[2] to trace how the mouth, one of the auto-erotic areas, becomes, under the stimulus of nipple-breast-mother, the first and chief channel in transforming auto-erotic love into object-love; and further that the mouth zone on the one hand and the anal and urethral zones on the other are in this sense contending influences, the former leading to object-love, extroversion and affectionateness, the others to introversion and the "shut-in" personality, increasing degrees of which are to be seen in paranoia, dementia paranoides and dementia præcox.

It was stated earlier that Claude's case included no delusion of grandeur and that narcissitic traits were notably slight or absent from his character. It would appear, therefore, that in his earliest life, next after auto-erotism came heterosexual love of his mother, but this, when he was two years old and his mother withdrew her love of him after

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[1] An uncontrollable fascination exercised on a child of 2 or 3 years by bath-taps, waste-pipes and closet-plugs may be one of the first indications of dementia præcox.

[2] "The Rudiments of Character" (read before the British Psycho-analytical Society, May, 1919).

Maisie was born, suffered injury.[1] Thereupon, regression took place to auto-erotism. In this regression the heterosexual component of the libido returned to a lower level, and the anal and urethral zones acquired an undue importance which was still retained in the eighth year.[2] And though doubtless it is necessary, in order to account for the whole history, to look to some special hereditary (i.e., structural and functional) factors as affecting the various zones, the more direct origin of the psychosis was this regression to auto-erotism. From that period the tendency had been to develop along the lines of a "shut-in" personality; or, more particularly, the heterosexual regression to auto-erotism diverted the course of development in the direction of dementia præcox, the homosexual repression tended to paranoia. But the immediate cause of the outbreak of definite psychotic symptoms was the death of the younger sister, the situation then reproducing in important respects the double trauma of earlier life,[3] a flood of libido being forced back to burst its limits at both the weak points—auto-erotism and homosexuality.

VII.

Opinion is not likely to differ as to the diagnosis of the case given in the title of this paper. But it might have been interesting to speculate on the course of the case had it been left untreated. Happily, however, there is no occasion for this. For a week or ten days before the boy came to the story of Leo, he showed that his defensive armour had been penetrated. "I'm not going to think people are unkind to me any more," he said, "It makes me unhappy, and then I am naughty." He began to make himself useful in little ways, tried hard not to be rude, and gave up flinching and talking to himself. And when at last he had unburdened himself of his auto-erotic fantasies he became almost friendly. He hoped that Miss P. would continue her daily notes which had roused his suspiciousness, as they made it easier for him when he came to me. He went a half-day excursion with his mother, and, the report said, behaved quite nicely. A week later, and for the first time in history, he asked to be kissed at bedtime.

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[1] This transference of affection from the boy to the girl points to the mother as being herself a repressed homosexual. In that case both Claude's parents were of this type—with fateful results to the child.

[2] It seems desirable to distinguish this *secondary* auto-erotism from *primary* auto-erotism, which is the normal state of very young children.

[3] I recognize the vagueness in which I have left the history of the earlier relations between Claude and his father, and though the evidence given is conclusive that a repression of homosexual feeling occurred at some early period, the time and occasion of this remain uncertain. Whether it developed as rapidly and completely as in the case of the heterosexual repression, or was the gradual result of disappointment in the father spread over a long time, I could not elicit. But the boy's inveterate reluctance to speak of his father, while accounting for my failure, would seem to point to a deep wound. Those, however, who have experienced the difficulties in analysing a child of this inverted type will not be reprehensive of this gap in the history.

Of more lasting importance were my interviews with his parents separately. The father saw readily enough the mistake he had made and agreed to leave other boys to be looked after by other fathers, while he interested himself in his own children. But the mother was more obdurate. It was necessary to let her know the part she had contributed to her child's illness; but she did not see the matter that way—Claude was an exceptionally unpleasant, troublesome boy who would try the patience of Job, and it was her misfortune to be his mother; and she left me abruptly without disguising her indignation. However, my judgment of her had not been wrong, and in due course Claude was restored to his heritage in her affections; I heard that what she found hardest to forgive was his examining his sister's genitals.

But all these events are now six years passed. Claude returned to his parents and, in the changed atmosphere which they created to the best of their ability, began slowly to mend. A year or so later he was well enough to be sent to a boarding-school where he still remains. School life has suited him, he plays football and other organized games not badly, and has made "very steady progress" temperamentally. He is still moody at times, with gusts of temper; and he is shy with strangers, though "a delightful companion with those he knows well." His mental powers keep high above the average, and he recently won an open scholarship at a public school.

These facts come to me second-hand, for I have never been permitted to see my little friend again—from which it might, perhaps, appear that some forgiveness is owing to me somewhere—and I cannot speak more fully of his present condition. Time alone can show his future: such nervous traits as persist are, though slight, enough to enjoin caution in prognosis. On the other hand it would be less than just not to say that the analytical treatment which found him a troublesome, even dangerous paranoid dement, has been the means of restoring him to social life.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Section of Surgery.

President—Sir JOHN BLAND-SUTTON, F.R.C.S.

DISCUSSION ON SECONDARY DEPOSITS IN BONE, MISTAKEN FOR PRIMARY TUMOURS.[1]

The PRESIDENT.

WHEN secondary carcinoma grows in an organ not easily accessible to putrefactive micro-organisms the deposit may become big before attracting attention. This phenomenon has been particularly observed in connexion with the ovary. Large solid and semisolid ovarian tumours have been removed under the impression that they were primary. The histology of such tumours has proved that the primary tumours arose in the breast, gall-bladder, or gastro-intestinal tract, and the subsequent history of such patients has confirmed the histological findings. This is equally true of bones. Portions of long bones have been removed, and limbs have been amputated for tumours regarded as primary, but the tumour-tissue has contained epithelial elements. It is established that epithelial tumours do not arise primarily in bone.

Forty years ago the attention of surgeons was drawn to the occurrence of tumours in bone, regarded as primary, but which contained epithelium, in shape and arrangement resembling the lining of the vesicles of the thyroid gland. Many examples have been recorded in which such tumours have been discovered and removed, or recorded, in the belief that they were primary, but after a minute examination of the structure of the tumour, the surgeon has re-examined the patient and found an enlarged thyroid body. In recent years some remarkable bone tumours have been observed (and removed) in bones which, when examined, have been found to resemble the zona fasciculata of the adrenal. Subsequently a tumour of similar structure has been found in the kidney or in the adrenal. This matter is of clinical and pathological importance and well worthy the consideration of surgeons. It has been thought by the Council desirable to invite surgeons to bring to this meeting specimens and case-reports illustrating the difficult problem raised by such occurrences, so as to afford an opportunity of collecting the evidence. My contribution is a case described in full last year. Its leading features may be briefly summarized:—

---

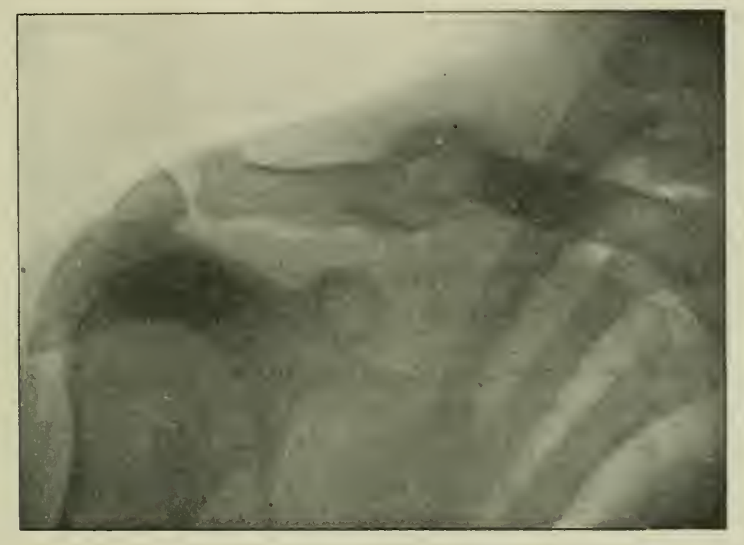
[1] At a meeting of the Section, held November 5, 1919.

A doctor, aged 45, had a swelling in the lower part of the right humerus. Clinically it furnished the usual signs of a myeloma. It was excised under this impression. Two years later it recurred in the stump of the bone; this was excised with 2 in. of the humeral shaft. He remained well for five years and died suddenly in the Middlesex Hospital. After death a tumour was found in each adrenal and in the right kidney. The tumours in the humerus and in the adrenals and in the kidney were so destroyed by extravasation of blood that the structure of the tumour could not be satisfactorily determined, but a solid secondary nodule in the heart, in shape and size like a nut, showed the characteristic structure of the zona fasciculata of the adrenal. On these facts I came to the conclusion that we had here an example of the dissemination of an adrenal tumour, but Professor Shaw Dunn who examined the specimens believes that the primary tumour arose in the kidney and was a hypernephroma.

The chief lesson to be drawn from the narrative is this: A man had a tumour, in the right kidney, that ran such a chronic course that no one suspected its existence, although a secondary deposit in the right humerus became large enough to be clinically obtrusive and mimic a myeloma. After removal of the secondary tumour the patient lived in comfort and usefulness for five years. It also illustrates what I have often tried to emphasize, that cancer *per se* is an extremely chronic disease until it becomes septic: then misery attends until life is extinguished by a terminal infection.

Mr. V. Warren Low.

H. P., aged 55, was admitted to hospital on December 11, 1913, for a lump on his right clavicle. About eight weeks before admission he "ricked" his shoulder while practising physical exercises. In order to ease the slight pain which followed he rubbed his shoulder with some liniment, and it was while doing this, about a fortnight after the accident, that he first noticed the lump. The latter was not painful and caused him little inconvenience. There was a hard, somewhat lobulated swelling on the right clavicle at the junction of the outer and middle third. The swelling was not attached to skin and appeared to embrace the shaft of the bone. An X-ray photograph showed that there had been a fracture and that the broken ends of the bone, which slightly overlapped, were enveloped in a mass of "growth." Although a Wassermann reaction was negative, antisyphilitic treatment was adopted for a short time with what appeared at first to be some benefit. It was soon apparent that there was no real diminution of the swelling and it was decided to explore.



Bony swelling of the clavicle.

With Mr. Fitzwilliams' assistance, on January 6, 1914, I exposed the tumour, and found it to be a definitely bony swelling of the clavicle. There was no evidence of anything like growth on the surface. I gouged through the compact tissue, which was certainly not thinned, and exposed a cavity containing some soft yellowish material not at all unlike what one would expect to find in a gumma. This was contained in a smooth-walled cavity in the bone, from which, however, the bleeding was profuse and could only be stopped by filling the entire cavity with wax. Some of this soft material was immediately cut and examined by Dr. Spilsbury, who pronounced it to be a carcinoma. We had no definite evidence of any primary growth and I therefore, with the concurrence of Dr. Spilsbury and Mr. Fitzwilliams, decided to remove the clavicle. This I did and his recovery was uneventful. There was no evidence of carcinoma either in his prostate, intestines or mammæ. He had no swelling of his thyroid, and we were driven to the guess that this was one of those rare bone tumours secondary to an adrenal growth. I may say that we had no direct evidence of this, as both kidney regions appeared normal.

Some three or four years ago, while I was away out of the country, the Ward Sister wrote to this man and received a letter from his wife saying that he had died but giving no particulars.

Microscopical Report by Dr. Spilsbury.—Growth of clavicle: H. P. Operation on January 6, 1914. Sections show a malignant growth having the characters of a carcinoma and resembling a hypernephroma. The cells of the tumour are large and in places they are columnar in shape and have a regular glandular arrangement. The cell protoplasm is finely vacuolated. There are numerous thin-walled blood-vessels but no other stroma. Blood is present in some of the tumour acini.

MR. NITCH.

I have had two cases, which are examples of errors in diagnosis, resulting from absence of any symptoms which would have led to a recognition of the primary growth.

The first occurred in a man, aged 52, with a swelling of two years' duration below the right elbow. This was causing great pain, and increased rapidly in size during the last three months. Immediately below the olecranon process there was a soft, fluctuating, fusiform swelling, 3 in. long and 2 in. wide, involving the upper third of the ulna, and invading the elbow-joint. A skiagram showed destruction of the coronoid process, and practically the whole of the upper third of the ulna, only a thin shell of bone being left over the back of the tumour. Owing to the long history and the severe pain the lesion was thought to be due to tubercle. On cutting into the swelling it was found to be a soft, highly vascular, plum-coloured growth, studded with sulphur-coloured nodules. Above it was encapsuled, but below it was infiltrating the muscles. It was widely excised. Dr. L. S. Dudgeon made a microscopical examination, and described it as typical of carcinoma of the adrenal cortex. The abdomen was then examined, and a nodular tumour was found in the right loin. Shortly after the operation a soft pulsating nodule was discovered on the ninth rib. The growth in the arm recurred, and is now as large as at the time of operation, six months ago. Until three weeks ago, when he had a severe attack of hæmaturia, there have been no symptoms suggestive of the existence of the primary growth.

The second case was that of a female, with a large globular swelling of the upper end of the right humerus, and another in the body of the left scapula. These were thought to be sarcoma, and their real nature was not recognized until after a microscopical examination had been made of a portion of tumour removed for the purpose. It was then discovered that the growths were secondary to an adrenal carcinoma, which up to that time had given no signs of its existence.

Dr. NICHOLSON.

The case I describe was under the care of Mr. E. C. Hughes. The patient, a girl, aged 19, was operated on for a soft central tumour of the lower end of the tibia. On microscopic examination it presented the typical structure of an embryonic tumour of the kidney. No abdominal neoplasm could be palpated. Two years later she was re-admitted for a recurrence in the tibia. A tumour was now palpable in the left kidney region. Mr. Dunn, who was asked to see the patient, agreed that it was a renal growth. No further operation was performed, and the girl was lost sight of.

Sir HUMPHRY ROLLESTON.

A squamous-celled carcinoma rarely produces metastases at a distance. The following two cases of curious metastases are worthy of mention: A man found dead in the street had a nodule of squamous-celled carcinoma in the apex of the heart; there was no other growth in the body, but the penis had been amputated in the past—presumably for a squamous-celled carcinoma.[1] In a man whose right arm had been amputated for recurrent squamous-celled carcinoma both layers of the pleura on the right side were infiltrated with secondary growth and enclosed a cavity containing blood-stained fluid. What is the relative tendency of malignant growths of the adrenals themselves on the one hand and of the so-called renal hypernephromas (" Grawitz tumours "), the adrenal origin of which is now much in doubt (Wilson and Willis), on the other, to produce bony metastases? It is noteworthy that the so-called renal hypernephromas are not associated with the abnormal sex characters seen in cases of tumours arising from the adrenal cortex (Glynn),[1] and it would be interesting to know if there is any corresponding difference as regards bony metastases.

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[1] *Trans. Path. Soc., Lond.*, 1892, xliii, 30.

Mr. CECIL ROWNTREE.

The following is a case of medico-legal interest: A boy was knocked down by a motor car, and his head was injured. Shortly afterwards a tumour of the frontal bone developed, and the boy died. The autopsy revealed the presence of a malignant growth in the sigmoid with numerous secondary deposits, including a deposit in the frontal bone, all of identical histological character. The Coroner found that the mass on the head was in fact secondary to a primary growth in the sigmoid, and returned a verdict of death from natural causes.

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[1] Glynn, *Quart. Journ. Med.*, Oxford, 1911-12, v, 180.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Case of Coxa Vara after Reduction of Congenital Dislocation of Hip.[1]

By E. Laming Evans, F.R.C.S.

The patient, H. B., is aged 12. In March, 1913, I reduced by manipulation, a left-sided congenital dislocation of the hip. The reduction presented no difficulty, and an X-ray examination after reduction showed no evidence of trauma, either at the epiphyseal line or in the neck of the femur. Retention was effected by a series of short unilateral plaster spicas in 90° of abduction and 50° to 70° of flexion for eighteen months; 50° to 70° of flexion was necessitated by a tendency to anterior transposition. During the last twelve months of plaster retention the child walked on a 5 in. patten on the left, or affected leg. After the plaster was removed, a 1½ in. patten was used on the sound side, to maintain abduction of the affected side. When the plaster was removed, the affected leg measured 20¾ in. and the sound leg 21 in.

The child has been kept under observation and a series of twelve X-rays taken during the six and half years of treatment. Six years after reduction, the leg measurements were, left 25¾ in., right 26 in. Six months later the difference had increased to ½ in. and the X-ray showed a varal deformity of 100°, occurring at the junction of the neck and great trochanter. The epiphysis is seen correctly placed upon the neck, and situated correctly in the acetabulum underlying a well formed roof.

The occurrence of coxa vara after reduction of congenital dislocation of the hip has an important bearing upon the functional result of a successful reduction. It produces a limp from shortening of the leg and from gluteal insufficiency, which, though of different origin

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[1] At a meeting of the Sub-section, held October 7, 1919.

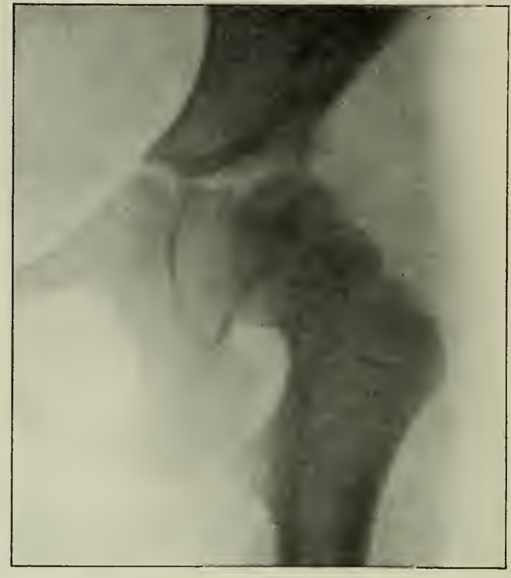


FIG. 1.

X-ray taken one year and four months after reduction.

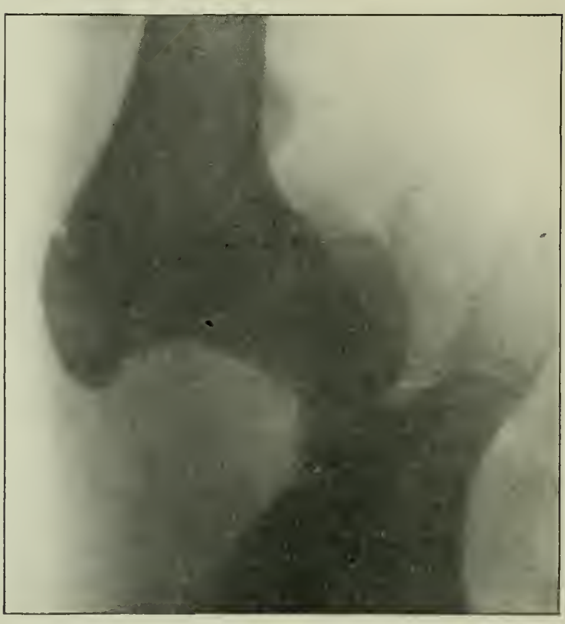


FIG. 2.

X-ray taken six years and six months after reduction.

and of lesser severity than that of unreduced dislocation, is, nevertheless, regrettable and, if possible, to be avoided. The recognized causes have been classified under five heads:—

(1) Trauma inflicted at the time of reduction, either upon the epiphysis or on the femoral neck.

(2) Prolonged weight-bearing, either before or after reduction.

(3) Absence of ossification in the head of the femur.

(4) Absorption of the ossific centre, which has appeared.

(5) Acute traumatic reflex atrophy.

That coxa vara is a not uncommon complication of successful reduction of congenital dislocation has been shown by Joachimstal, Horvath and others.

The points of interest in the present case are the remote period, i.e., six years after reduction, and the site of bending, i.e., at the junction of the neck and great trochanter. The functional disability with 100° of varal deformity is slight, but it necessitates further observation and, if progressive, treatment by a perineal crutch.

DISCUSSION.

Mr. H. A. T. FAIRBANK: X-ray examination seems to suggest the occurrence of an injury to the neck of the femur close to the epiphyseal line which may have influenced the later development of a coxa vara deformity. With regard to treatment the indication is to prevent the transmission of weight through the neck of the femur.

Sir C. GORDON WATSON: The deformity consists in a bending of the femoral neck at its junction with the shaft as in the rickety type of coxa vara, and not in a displacement of the epiphysis, as would be expected if injury were the cause.

Two Cases of Secondary Scoliosis.

By PAUL BERNARD ROTH, F.R.C.S.

CASE I.—SCOLIOSIS DUE TO PARALYSIS OF ERECTOR SPINÆ.

K. G., FEMALE, aged 8; first seen in December, 1918, with the following history. Was a strong healthy child till October, 1916, when after a few days' drowsiness and loss of appetite, ending up with feverishness and vomiting, she was found to be completely paralysed in both legs, and was taken to University College Hospital. There she was treated with massage for the legs: and after a few months by a jacket, as spinal curvature developed, and then, as this was not satisfactory, by plaster of Paris, which was applied twice. Patient was under treatment there for over two years, and during this time she became much deformed.

On examination, there was severe paralysis of the right erector spinæ muscle, with Ɔ-shaped scoliosis, there being "severe" deformity of the right ribs posteriorly; while the upper part of the trunk overhung the lower part to the right.

A tracing was taken of the amount of rib deformity, and daily treatment by "posture and exercise" begun. She was not allowed to walk about, but spent the day lying in a spinal carriage, either supine,—or prone with her body supported on her elbows with a cushion under her chest, whichever she preferred. She was allowed to sit up to the table on a chair for meals. In the summer she was taught to swim, and can now swim several times across the bath.

At the present time she is obviously very much stronger in every way than she was in 1918 when first seen, and what is so important, there has not been the slightest increase in the amount of structural deformity of the ribs.

Case II.—Scoliosis due to Congenital Malformation of the Spine.

J. W., female, aged 5; first seen on December 12, 1918. On examination, there was found **S**-shaped scoliosis, with "severe" deformity of the left ribs posteriorly, and with "moderate" projection backwards of the right erector spinæ in the lumbar region. She could be placed in a very much improved position, and could voluntarily retain it.

An X-ray showed that an extra half vertebra, carrying a rib, was interposed on the left side between the eleventh and twelfth dorsal vertebræ, and that only the right half of the ninth dorsal vertebra was present, it being fused with the body of the tenth: four ribs, two on each side, sprang from the fused mass. Other abnormalities were also present.

A tracing having been taken of the amount of deformity, treatment by "posture and exercise" was begun on January 9, 1919. She now holds herself in excellent position. The rib deformity has not increased, as is shown by comparing a tracing taken now with that made on December 12, 1918.

These two cases are shown to demonstrate that the progress of deformity may be arrested without resorting to mechanical support.

DISCUSSION.

Mr. A. S. Blundell Bankart: In such cases as these, a time must come when the treatment must be stopped. It will be impossible to continue treatment by exercises for an indefinite period of years. When this treatment ceases some degree of permanent scoliosis will persist and a support will then probably be required.

Mr. Laming Evans: In cases of paralytic scoliosis the same principle should be applied as in the treatment of paralysis of the lower limbs—that is, the support of the paralysed muscles in such a position as will prevent their being overstretched. This can only be carried out by the use of a mechanical support.

Mr. H. A. T. Fairbank: In congenital scoliosis a distinction should be drawn between the primary curve due to the original malformation and secondary curves, which may develop at any age. The latter as a rule are not marked in young children; they develop and become worse as the child grows. A careful watch should be kept upon these cases to prevent progress of this secondary deformity. The treatment, so far as the secondary deformities are concerned, should be on the same lines as for any other variety of scoliosis.

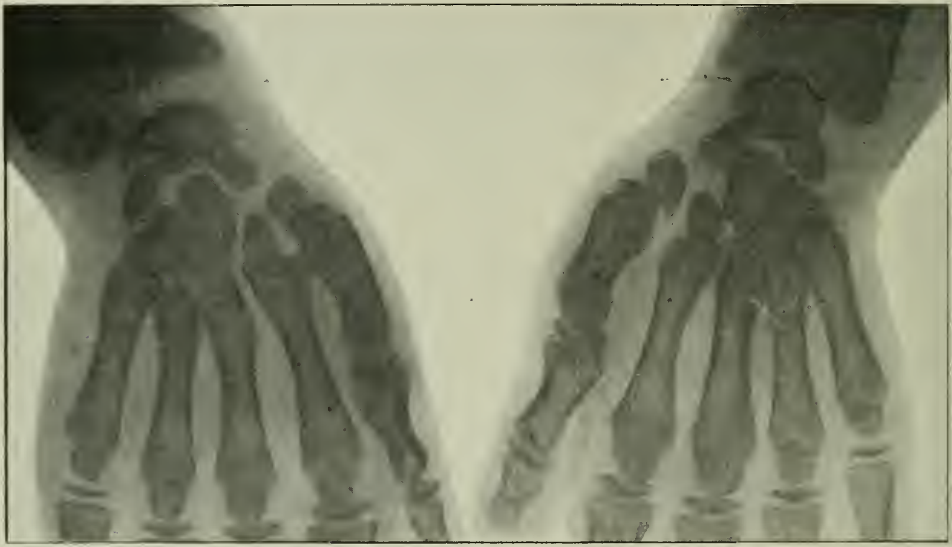
Mr. Elmslie: In the treatment of the paralytic case two mechanical factors have been neglected. The right lower limb is shorter than the left and this has not been compensated. Also, the whole thorax is tilted to the right to such an extent that a vertical line drawn through its centre of gravity falls much to the right of the centre of the pelvis. A severe strain is thus placed upon the weakened muscles and a mechanical factor left which must constantly tend to increase the deformity. I should first apply a support that will bring the centre of gravity of the body over the centre of the pelvis and then carry out muscular treatment.

Mr. Roth (in reply): According to the mother the child has already been treated on the lines suggested by Mr. Elmslie. I agree with Mr. Evans that the usual principles applied in the treatment of infantile paralysis should be observed, but after some years something more active should be done. If the deformity gets worse instead of better I shall use a support. I do not carry on treatment indefinitely; after a time observation, with periods of treatment by exercises, is all that is necessary.

Case of Infantilism.

By R. C. Elmslie, F.R.C.S.

The patient, a girl aged 15 years and 10 months, is said to have been healthy as a baby. At 1 year and 9 months she had diphtheria and after this suffered from gastro-intestinal disturbances with recurrent attacks of diarrhœa until the age of 13. Since that time her general health has been much improved, and is now excellent. Her height is only 3 ft. The head measures 20 in. in circumference, the forehead being rather high and square. The spine is scoliotic, with right dorsal and left lumbar convexities. There is coxa vara deformity of both hips with limitation of abduction, a slight degree of genu valgum and anterior curvature of both tibiæ. X-rays of the hips show coxa vara of the rickety type, the whole neck of the femur being bent without alteration in position of the epiphysis. X-rays of the wrists show cupping of the ends of the diaphysis with widening of the epiphyseal cartilages as in rickets; those of the tibiæ showed a general lightness of the bone with a very thin compact layer. Sexual development is absent, the breasts undeveloped, pubic hair absent and menstruation has not yet commenced. The urine contains a trace of albumin. So far there has been no opportunity of investigating the child's general metabolism.



Skiagram of the epiphyseal regions at the wrists in Mr. Elmslie's case of infantilism.

Two conditions are present, each of which is found associated with infantilism—namely, albuminuria and the chronic intestinal trouble.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. J. P. Lockhart-Mummery, F.R.C.S.

Case of Intestinal Coccidiosis (?)[1]

By P. Lockhart-Mummery, F.R.C.S., and W. B. Gabriel, F.R.C.S.

The patient, a man, aged 43, was admitted to St. Mark's Hospital with intractable diarrhœa and multiple adenomata of the colon. He had been ill about eight months.

The specimen shows that the mucous and submucous layers are much thickened with numerous small sessile projections. The whole rectum, colon, and part of the small gut were involved, also the appendix.

Microscopical sections taken from ileum, appendix and colon show innumerable definite cystic bodies of varying sizes which stain readily with hæmatoxylin. The majority are near the surface of the epithelium and many of the cysts are seen rupturing on to the surface.

Dr. Manson-Bahr: I do not think the structures and the section exhibited are coccidia at all: the appearance of cystic structures is simply a question of the way in which the tissues have been fixed. The surrounding epithelial nuclei are not well stained, indicating that the specimen has not properly been fixed, presumably in formalin, instead of in corrosive alcohol. If one goes into the question of human coccidiosis, it will be found that nearly all the cases of supposed human infections with this protozoon have an explanation similar to this one. I think there are only two recorded cases in Dobell's recent paper on the subject which will bear minute investigation at all. I have been finding these coccidia (*Isospora hominis*) occasionally in the stools of Turks and other Eastern natives: that is to say, one finds the cysts, under the microscope, but this does not necessarily indicate that they are derived from an ulceration of the bowel. What the explanation is of these human carriers of coccidia we are not at present able to say. But I may say that among all the post-mortem examinations I have made on Turks and natives of other Eastern countries, I have never come across an authentic case of human coccidiosis, though I have often seen sections in which the cystic appearances caused by the goblet cells resembled that seen in this case.

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[1] At a meeting of the Sub-section, held November 12, 1919.

Case of Multiple Adenomata of the Colon with Secondary Carcinoma.

By P. LOCKHART-MUMMERY, F.R.C.S.

THE rectum was excised by the abdomino-perineal route and the patient is doing well. The specimen shows numerous adenomata, and in one place near the lower end of the sigmoid secondary carcinomatous change has caused a stricture and passed through the walls of the bowel and become adherent to the peritoneum.

The PRESIDENT: I removed this a month ago from a woman, by the abdomino-perineal incision, and she is now all right. The specimen shows that the polypi have become malignant in one place.

Case of Sarcoma of the Rectum.

By P. LOCKHART-MUMMERY, F.R.C.S., and W. B. GABRIEL, F.R.C.S.

THE specimen was removed by operation from a man aged 47. It shows the lumen of the bowel encroached upon by an irregular mass situated beneath the mucous membrane.

Sections show a firm white fasciculated tumour in the submucous layer of the rectal wall.

Microscopically, masses of malignant cells are seen, many of them exhibiting central degeneration and hæmorrhages.

DISCUSSION.

The PRESIDENT: At the time of operation I regarded the case as one of carcinoma, but when we got it out we found something peculiar about it. There was no ulceration on the mucous membrane, and there was a very hard elastic layer outside. We have now cut sections of it, and it appears to be a round-celled sarcoma.

Mr. W. SAMPSON HANDLEY: It is difficult to express an opinion on a microscopical specimen in two minutes, but I think this is a sarcoma. There are large areas of connective tissue in the middle of the masses of malignant cells, an appearance not generally seen in carcinoma. What were the clinical features? Was there induration in the ischio-rectal fossa? One or two of the cases I have seen presented themselves as gigantic single piles, followed by ischio-rectal induration and infiltration of the ischio-rectal skin.

Mr. W. B. GABRIEL: The history dated from fourteen days previous to admission. The patient stated that he had had difficulty in defæcation, with slight pain: there had been no bleeding nor discharge from the rectum. On examination, a hard irregular mass was felt low down in the rectum, which bled on examination: it seemed to be slightly fixed anteriorly, but it was not found to be so at the operation. There was no induration in the ischio-rectal fossæ.

The PRESIDENT (in reply): It was a very early case. There was no broken surface on the epithelium at all; it was quite smooth over its whole extent. [Mr. SAMPSON HANDLEY: That has been my experience of sarcoma.] If it is sarcoma it is remarkable in the fact that it is so early. I have not previously seen sarcoma of the rectum except it was in the nature of an extensive growth beyond the hope of successful removal.

Dr. MANSON-BAHR: Does not the microscopical structure of this section militate against the idea of sarcoma? I suggest that the cells are too regularly arranged in rows, and that the arrangement of the blood supply is not what one would expect in sarcoma. More serial sections of different portions of the tissue should be made before a definite opinion can be expressed.

Sir GORDON WATSON: If a transverse section of that growth were made across the bowel, so that its whole surface could be examined, I think some point of ulceration would be found. I agree with Dr. Manson-Bahr that the structure is more like that of carcinoma than of sarcoma, though the section shows normal mucous membrane on the top of it. I think search would reveal a site at which there is a down-growth.

(On the suggestion of Mr. SAMPSON HANDLEY, it was agreed that Professor Shattock be asked to examine and report on the specimen.)

Specimen from Case of Anal Warts.

By J. P. Lockhart-Mummery, F.R.C.S.

This is the biggest case of anal warts I have seen. When fresh they were as large as a soup plate. The case was sent from the Army as one of epithelioma. The anus was normal. We burnt it off in portions with nitric acid. X-rays were found to be useless.

Case of Carcinoma of the Colon after Operation.

By Percival P. Cole, F.R.C.S.

R. E., female, aged 45. Admitted for increasing constipation, colic pains and borborygmi. Bismuth enema shows stricture in pelvic colon.

Operation, May 20, 1919: A muscle-splitting left-sided incision revealed a malignant whipcord stricture, about the middle of the pelvic colon, the bowel above being dilated and thickened, and the bowel below collapsed. The loop was brought out and two Paul's tubes put in, and immediate resection was done. She was left with two separate colostomy openings. Four or five weeks after—a rubber drainage tube by the Mitchell Banks method being used meantime—an area of skin was excised and the two ends of the bowel freed, the peritoneal cavity not being opened. I did a direct end-to-end suture, and brought the muscles over. It was a muscle-splitting operation, and there is a submuscular anastomosis.

DISCUSSION.

Mr. Swinford Edwards: Why did Mr. Cole do a preliminary colotomy here? The sloughing was due to the fact that such a large slice of mesentery was removed. Is that really necessary to such a large amount? I have operated on two or three similar cases, doing an end-to-end union, and have not removed any appreciable amount of mesentery. One case I can call to mind: that of a lady on whom I performed colotomy fifteen years ago. I still see her from time to time, and she is perfectly well. Now and then she has some hæmorrhage from the rectum, and some difficulty with her motions, but I have examined her with a large calibre sigmoidoscope, especially the area of operation, and except for a few granulations, all seems to be normal. I have not been able to get very far above it, so that possibly the bowel is in a granular condition higher up. Her condition was undoubtedly one of carcinoma, and she has lived without recurrence for fifteen years. She came to me after having been under treatment by the late Dr. Lewis Jones for rodent ulcer of the face; this he had cured by radium.

Mr. W. ERNEST MILES: The case is very interesting from the point of view of spread of cancer. I have done several resections of the pelvic colon, and formerly I removed a segment of bowel and effected an end-to-end anastomosis: I performed seven such operations in three or four years. Subsequently, as a result of intestinal obstruction, resulting from the adhesion of a loop of small intestine to a plaque of growth, it was possible to see the existing condition, which was a metastatic deposit in the pelvic mesocolon itself. I think, therefore, it is very important to remove a considerable area of the pelvic mesocolon in these cases. The lymphatic arrangements are as follows: Along the attached border there are the paracolic glands, and from these the lymphatics pass across the mesocolon along its parietally attached border and thence to the lumbar glands. I have repeatedly seen small plaques scattered about in the mesocolon, along the parietally attached border, in the middle of it, and also close to the attachment of the colon. In deciding the operability of a given case, you must ascertain whether there is actual visible spread of the disease in this highly vulnerable tissue. We have also to remember the possibility of the existence of microscopical spread: it is quite impossible to say whether a detached carcinoma cell may not have been carried some distance from the primary growth in the lymphatic stream, which may result in a metastasis a month or two after the operation. I agree with Mr. Swinford Edwards that, by freely removing the mesocolon, one may endanger the vitality of the distal portion of the colon, but if one remembers the anatomical blood supply and does not divide all of the three sigmoid branches of the inferior mesenteric, I think there will probably be very good union. I am very keen on the operation being fashioned to include the whole area of the possible microscopical spread of the disease.

The PRESIDENT: I agree with what Mr. Miles has just said. I think that where surgeons get into difficulty in these cases is, that there are no means of finding out whether one is dealing with a case in which there is considerable microscopical spread, or with one in which there is none. At St. Mark's I had a case in which I cut out a small carcinoma of the sigmoid, the smallest that I had ever seen removed: it was no larger than the tip of my little finger. It was detected with the sigmoidoscope. I resected 4 in. of gut, and a large amount of mesentery. Apparently even its pedicle was not malignant. Yet that woman came back eight years later with an extensive recurrence all over the mesentery and in the liver. Therefore a growth must have spread to an immense distance even so early. On the other hand, the surgeon meets with cases such as the one Mr. Edwards quoted, it which one cuts down close to the growth and removes no lymphatic area because one could not very well do otherwise, and yet in fifteen or twenty years there is no recurrence. Our real trouble, therefore, lies in not being able to know one type of case from the other. With regard to preserving the blood supply, the difficulty is when the patient is a very fat person. In a thin patient one can tell when one leaves the sigmoid arteries intact. But if it is a stout person with the mesentery full of fat, I find nothing more difficult than to find out where the blood supply has got to, and to resect a large wedge and get the patient through is almost an impossibility.

Mr. IVOR BACK: I understand that the question Mr. Cole has provoked in this case is: "Is it safer to resect a large portion of bowel with its mesentery by this method of exteriorization, than it is to carry out the ideal method of resection and end-to-end anastomosis?" One can remove as much mesentery by one method as by the other. I think the point at issue would be solved if the members of this Sub-section would all give an honest opinion as to what is their mortality from resection and end-to-end anastomosis by the ideal or intraperitoneal method in the colon, as compared with this exterior method in which the peritoneal cavity is not opened. If Mr. Cole, or any of us, can prove that there is a definite mortality from doing it intraperitoneally, and that this mortality is greater than it is from the exteriorization method, a great point will have been established in its favour. My own experience is, that there is a definite mortality from end-to-end anastomosis intraperitoneally in the case of the pelvic colon, or in any part of the colon, because of the risk of soiling the peritoneal cavity at the operation, however perfect we think our technique is. I see Mr. Cole's point, that the danger of soiling the peritoneal cavity is eliminated by this operation, or nearly so. My experience and observation lead me to say that the mortality from end-to-end anastomosis intraperitoneally is certainly above 10 per cent., probably nearer 15 per cent., giving just a casual idea based upon past cases, whereas in this method it should be almost *nil*. If so, I think much has been established in its favour.

Sir GORDON WATSON: I think the method adopted by Mr. Cole is the right one when there is obstruction. I think it is usually taught, and I have taught students that they should adopt it in cases of obstruction when it is possible to bring the growth outside. In many cases it is not possible to bring it outside: short mesenteries are met with as often as long ones; and if there is a growth, the mesentery is more often shortened than not, and adhesions are quite common.

Mr. COLE (in reply): Five weeks elapsed between the first and the second operation. They had not united, because I stitched the two ends of the bowel together laterally, so that that should not occur. It is really Paul's original method. The patient came with colicky pain, and she had visible peristalsis, with chronic obstruction. There was some dilatation of the bowel above the stricture. It seems to me that the origin of sepsis in these cases where there is obstruction is the increased virulence of the micro-organisms due to the obstruction. Even if there is not clinical obstruction, but obstruction which is evidenced by dilatation of the bowel when the abdomen is opened, it is better to keep to the two-stage operation, for it is more conservative of the patient's life than the ideal operation of resection and end-to-end anastomosis, which, one gathers from observation and conversation with others, has a definite mortality. And the scope of this operation is equal to that of the other. I have treated a number of cases in the same way, and I do not think there is danger of the lower end sloughing. I do not claim that there is anything new in this method: it is simply the one which Paul advocated many years ago; the point is as to the relative safety of the two procedures. If it conflicted with the radical nature of the operation one would be justified in arguing whether safety should be placed first. But that does not apply in this case, for as much mesentery can be excised or resected by exteriorization as by the primary and finished operation. Hence it becomes absolutely a question of relative mortalities.

Case of Piles of Unusual Size and Duration.

By H. Graeme Anderson, F.R.C.S.

W. S., AGED 48, stick carver. A case showing very well marked large internal piles of forty years' duration. The constant prolapse has rendered the sphincters atonic; although there has been a fairly constant discharge of mucus there has never been any pruritus. In operating on such a case the sphincters should not be stretched.

DISCUSSION.

Mr. W. SAMPSON HANDLEY: Although those piles have been extruded so long, there is no sign of conversion of the columnar epithelium into the squamous variety, a change which often occurs when piles have been exposed for a long time.

Mr. W. ERNEST MILES: My chief interest in this case is, that I do not agree with the diagnosis: I do not regard it as one of internal piles. My understanding of internal piles is, that there is an altered condition of the veins in the lower part of the rectum: they have become dilated and varicose, and the walls are considerably thickened by fibrous tissue. There is a certain definite arrangement of internal piles in relation to the orifice of the anus, which is determined by the blood supply. It is well known that the blood supply of the rectum is derived from the superior hæmorrhoidal arteries, of which there are two. The right one divides, soon after perforating the muscular wall of the bowel, into two branches: an anterior, which courses down the right anterior quadrant, and a posterior, which supplies the right posterior quadrant. But on the left side the vessel remains single and passes down the rectum on the left side. Therefore the piles which originate from these three main vessels, viz., the right anterior, the right posterior, and the left, are what I have described as primary piles. The main branch in the right posterior quadrant gives off two secondary branches: an anterior, which develops into a pile at the right point of the anal orifice, and a posterior, which develops into a pile in the middle line posteriorly. The left main vessel also gives off two branches: a left posterior and a left anterior, which give rise to piles in these positions. These four piles I call secondary piles. Consequently, there are seven piles which can develop and these preserve a constant position in regard to the anal orifice, which is characteristic. In this case you will see there is a very definite fold on the right side, extending practically the whole length of the right side of the anus. There is also one on the left, and there are two smaller ones in the middle line, an arrangement which is quite distinct from that shown by internal piles. I believe this to be a case of prolapsus mucosæ recti, i.e., prolapsed folds of redundant mucous membrane thickened from protrusion, but there are no dilated veins, beyond the ordinary supply from the venous plexus. This idea is borne out by the length of time the condition has lasted: the patient began to develop the protrusion when he was eight years of age, and people seldom have piles at so early an age. I do not regard it as a case of procidentia. The differentiation of the condition from piles is important not so much from the point of view of treatment, but from a pathological basis.

Mr. SWINFORD EDWARDS: I am inclined to agree with Mr. Miles, but whether it is prolapse of mucous membrane or internal hæmorrhoids, the same operation will cure the patient—namely, operation by ligature. I questioned this patient, and he says he has never been able to afford the time to lie up for operative treatment. That being the case, I cannot see why this should not be treated by carbolic acid injections: it seems a very fitting case for it. It is true there is but little sphincter power, but I should keep him in bed for a day or two. If he is warned to replace the prolapsus at once should it recur, I conceive injections would cure him rapidly without his having to lie up.

Mr. GRAEME ANDERSON (in reply): I thank Mr. Swinford Edwards for his suggestion in regard to treating this case by injection, and I think it is well well worth while to try it. There is a strong family history of hæmorrhoids. In these long standing cases the prolapsing hæmorrhoids gradually weaken the sphincters, and then become fused together and form a prolapsus mucosæ. Although hæmorrhoids take up as a rule definite positions, it must be remembered that abnormalities in the blood supply of the ano-rectum occur frequently.

The Treatment of Procidentia by Injections.

By F. Swinford Edwards, F.R.C.S.

I thought you might be interested in hearing my experience of what I conceive to be a somewhat novel method of treating procidentia recti. During the past few years I have treated three cases of this condition by a combined method of injection and linear cauterization—the object being to produce adherence not only between the mucous and muscular coats but also between the muscular coat and the surrounding tissues by inducing a hyperplasia with subsequent fibrosis and consolidation.

The operative treatment of the minor degrees of prolapsus by linear cauterization is most successful, but I cannot say that this has been my experience in complete prolapse or procidentia—i.e., where the muscular coat descends as well as the mucous, carrying with it in front a pouch of peritoneum and protruding from the anus some 5 in. or 6 in., or even more. For this condition ventrifixation or sigmoidopexy and rectopexy, either by suturing or packing, and amputation have been and still are practised. More recently the injection of paraffin was tried, but I believe has now been abandoned.

I have performed both sigmoidopexy and rectopexy on several occasions, but I cannot say that complete success was the result, at least in every case. I call to mind one case of sigmoidopexy in which, although the operation had been quite successful in keeping up the prolapsed bowel, the patient was so worried with a dragging pain in the hypogastric region (the area of the operation) that he begged me to release the gut as he preferred the prolapsus to the abdominal pain. I may mention that although this patient had remained free from procidentia for over a year it recurred within a month after I cut it away from its anchorage to the abdominal wall.

Amputation I look upon as being too risky to be generally applicable. It should be limited to cases of sloughing or where the protrusion is non-reducible.

To take the place of these various operations I have been trying a method which I will now describe, and which I adopted after reading a paper in the *Practitioner*, 1909, by Dr. Inglis Parsons, in which he advocates the use of quinine injections for prolapsus uteri. This treatment of his was quite successful in a number of cases. The patient is anæsthetized and placed in the lithotomy position. The prolapsed bowel, if down, is replaced, and a duck-bill speculum is introduced. Linear cauterization is then carried out by means of a Paquelin's cautery, four eschars being made: one anteriorly, one posteriorly, and one on each side midway between the other two. The length of each eschar is from 3 in. to 4 in., the width about 1¼ in.; I endeavour to burn through the mucous coat. As the cautery knife approaches the anus it is sunk somewhat deeper, so as to involve the superficial fibres of the sphincter, in order to tighten up a patulous anus, for there is usually in these cases considerable loss of sphincter power. The speculum is withdrawn and some vaseline inserted. Two drachms of the following solution is now injected: Quinine sulph. 12 gr., acid sulph. dil. and water of each 30 minims. This is injected into the perirectal tissues —through a needle 3 in. long—by three punctures, two lateral and one posterior, entering the skin about an inch from the anus. When the needle is *in situ*, and before the fluid is injected, I pass my finger into the bowel to make sure that the point of the needle is in the right place. Before starting the operation the usual sterilization of anus and adjoining skin should be carried out.

After-treatment is of importance. As a rule, not much pain is complained of, but I give a morphia injection after the operation, and some preparation of opium in the evening to keep the bowels quiet. No action of the bowels should take place for a week, and then in the lateral decubitus—the nurse pulling the upper buttock to one side in order to give some support to the anus. This position indeed should be maintained at every movement for the first month, during which time I have kept my patients in bed. For the next two months the action of the bowels should be on a bed-pan in the dorsal position. When the usual position is resumed it is well so to regulate the bowels as to prevent any straining.

The following are the three cases in which I carried out this treatment:—

Case I.—Miss J., aged 26. Seen with Dr. Lowenthal. This patient had suffered with procidentia for years. It was a great inconvenience to her, always protruding at stool and on any exertion. There was but little sphincter power. The entire bowel protruded for about 5 in. The operation just described was performed: 24 gr. of quinine being injected, the bowels were opened in the lateral decubitus for a month. I heard some years afterwards that the patient married a few months after the operation and had since had two or three children. She has had no prolapse since the operation, ten years ago.

Case II.—Miss L., aged 50. Sent to me by Dr. Wallace, of Weston, for procidentia recti. She had suffered with protrusion at stool for many years, and recently both on micturition and walking. In this case precisely the same measures were employed as in the first case. Two months afterwards I received a letter saying that there had been no sign of prolapse since the operation, and again, after a further four months, the report was equally satisfactory.

Case III.—Miss J., aged 25. Sent by Dr. Gardiner, of Richmond. Her only trouble was protrusion of the bowel, both at stool and on walking. The prolapsus was of considerable size, protruding for 6 in. The same operation was performed, but in the right lateral instead of in the lithotomy position. In this case unfortunately the Paquelin's cautery let me down, refusing to work, as it will do at times. Luckily I had with me some cautery irons, which I used instead, but these are neither as efficient or convenient as is the Paquelin when in good order. In this case I injected 3 dr. of the solution instead of 2 dr., as the protrusion was so large. No prolapse occurred for three months, when there was a slight protrusion at stool. Seven months after the operation there was prolapse at stool, but not on walking, and it is half the size it was originally. I repeated the operation, this time placing the patient in the lithotomy position. Cauterization was more satisfactory, as the Paquelin worked well. On this occasion I used 4 dr. of the solution, or 48 gr. of quinine, and made the needle enter midway between the former punctures. I heard three months after that there had been no further prolapse.

This treatment seems free from risk of suppuration or cinchonism, and proved successful in all three cases. May I plead for a further trial of this method before resorting to either of the more serious operations?

Mr. W. ERNEST MILES: We should be very grateful to Mr. Swinford Edwards for having brought to our notice the treatment which he has found so satisfactory for procidentia of the rectum, because my experience is, that this condition is one of the bugbears of rectal surgery. One is confronted with cases of the condition which recur repeatedly, in spite of the treatment which has been carried out. I shall certainly give this method a trial in my future cases. In very pronounced cases—i.e., where the whole of the rectum has been protruded, the method I have hitherto been driven to employ has been ligaturing masses of mucous membrane, in order to constrict the orifice in the endeavour to keep up the procidentia. I also have adopted complete excision, known as Mikulicz's operation. Just before the war I employed this method in two cases, and the result has been extremely good It seems that the lower part of the peritoneum, that in proximity to the rectum, is more resistant to possible infection than the rest. I was able to pull down the sigmoid colon until it was taut with the descending colon, and then remove 6 or 8 in. of the lower part of the bowel, and finally suturing the proximal end to the skin, leaving the external sphincter intact. Both patients made excellent recovery, and were left with very good control. Mr. Cole has reported one or two cases he treated in a similar way.

Gunshot Wounds of the Large Intestine and Rectum, with Special Reference to Surgical Treatment.

By HAMILTON DRUMMOND, F.R.C.S.Ed.

THE interest aroused by the remarks of Dr. Arthur Keith and Sir Gordon Watson reported in the records of the last meeting has encouraged me to submit the following observations based upon a selection of my cases during the war.

Dr. Arthur Keith stated that a review of the reports of cases of gunshot wounds of the large bowel and examination of specimens had led him to the conclusion that retroperitoneal wounds of the colon or rectum and infection of the pelvic subperitoneal tissue were just as fatal as penetration of the peritoneum with intraperitoneal laceration of the bowel. Of the correctness of this opinion there cannot, I think, be any doubt, and, indeed, I would go further and venture to affirm that retroperitoneal infection causes a more serious lesion and one more frequently fatal than in the case of lesions limited to the peritoneum and laceration of the large bowel, and for the following reasons: First, because of the extraordinary power possessed by the omentum and peritoneum in dealing with septic organisms, especially the anaërobic varieties; and secondly, on account of the comparative ease with which wounds can be exposed when limited to the peritoneal portions of the gut. The retroperitoneal tissue on the other hand when infiltrated with blood in the presence of a damaged gut forms an ideal nidus for the growth of organisms, and the exposure to provide adequate drainage, especially in and around the pelvis, is most difficult.

From the accompanying table it will be seen that out of fifty-seven colon cases operated upon there were thirty-seven deaths and twenty recoveries; of the thirty-seven fatal cases nineteen died of shock and sixteen of retroperitoneal infection, two died of peritonitis, one of which was accompanied by retroperitoneal sepsis. From the table of gunshot wounds of the rectum there were fourteen deaths out of sixteen cases with two recoveries; seven died from shock, six from retroperitoneal sepsis and one from peritonitis with retroperitoneal sepsis. In the majority of these cases careful post-mortem examinations were made by competent pathologists, and I have to thank Dr. John Shaw Dunn and Major McNee for their assistance.

The extreme gravity of retroperitoneal infections accompanied by large bowel wounds was recognized and fully described by Sir Cuthbert Wallace[1] in his book on "War Surgery of the Abdomen," published during the last year of the war. John Fraser[2] also laid great stress on retroperitoneal sepsis, and applied the term "colon septicæmia" to a group of symptoms present in cases of colon wounds with a spread of infection to the retroperitoneal tissues. His description is as follows:—

> "In the third instance the features of an intensely rapid septicæmia appear. The signs may appear and develop with startling suddenness. Patients suffering from this condition have generally a grey pallid appearance, and it suggests that there has been an extensive loss of blood, but investigation of the history will show that this has not been the case. There is restlessness and great uneasiness. Signs of delirium appear and become established. The pulse is characteristic; from the normal rate it very rapidly increases, so that in the course of a few hours it may have reached a speed of 150 a minute. The respiration rate increases until it reaches 40 to 50 a minute.
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> "The temperature behaves variously; in the most intense cases it falls to subnormal and remains so, in less acute cases it rapidly rises to a considerable height (104° to 105°), and shortly before death it falls with a crisis. Vomiting is common, frequently in mouthfuls, ultimately resulting in acute dilatation of stomach. Before death the delirium passes into complete loss of consciousness, and the extreme general pallor is replaced by a slightly jaundiced appearance. The abdominal signs are primarily those of localized infection. Later the abdomen becomes markedly distended; there is not the same degree of tenderness or muscular rigidity as one associates with a generalized peritonitis."

The most common colon areas for the development of fatal retroperitoneal infections are along the ascending and descending portions of the bowel where the mesentery is absent. In the *British Journal of Surgery*[3] I recorded along with Dr. John Shaw Dunn six cases of gunshot wounds of the colon and rectum, with post-mortem examinations, where the missile passed close to the colon or rectum without actually touching it, giving rise to extensive ulceration of the colic or rectal mucous membrane, and I may say that subsequently we met with many more instances of the kind. The following is an extract from the paper referred to:—

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[1] "War Surgery of the Abdomen," 1918.

[2] *Brit. Med. Journ.*, March, 1917.

[3] *Brit. Journ. Surg.*, v, 1917.

> "In explanation of the lesions two main facts have been observed by dissection. First, there was always some laceration of the muscular coats, though this was usually very slight in the mucosa, and it was not associated with actual loss of muscular substance. In one instance the damage to the muscular layers had clearly been produced by a dragging effect transmitted through strands of fibrous tissue or blood-vessels; in this case there was also deep laceration in the cortex of the kidney, though the capsule was intact. In the other cases, the effects were most probably due to sudden impact on a gas-filled sac, resulting in bursting at weak points. The lacerations here occurred most commonly in the unsupported sacculi.
>
> "The other fact of interest was that many of the perforating vessels to the mucosa in the damaged areas were torn across at the points where they passed from the muscular into the submucous coats. This afforded an explanation of the great extent of loss of mucous membrane where the injury of muscle was comparatively trivial; the patch of mucosa had really been killed by loss of blood supply.
>
> "In all cases it was apparent that the damaged tissues were subjected to early infection from the lumen of the bowel, and readily became gangrenous; at later dates the loss of tissue from this cause would no doubt have been much greater."

Death invariably resulted in these cases from retroperitoneal sepsis.

TREATMENT.

It is now generally agreed that the abdomen should be opened in every case except in cases of low buttock wounds where we can be certain that the missile has not either directly or indirectly injured the peritoneum.

Intraperitoneal wounds of the rectum low down in the recto-vesical pouch are most difficult to treat by suture on account of the depth of the narrow male pelvis, and I think in these cases Sir Cuthbert Wallace's suggested obliteration of the recto-vesical pouch by sutures with the addition of a colostomy, a plan well worth a trial; or the introduction of a large tube into the anus to provide for very ample drainage. Again, these intraperitoneal rectal wounds are frequently accompanied by much laceration of the lowest coils of the ileum as it lies in the true pelvis, and also by injury of the bladder, and severe compound fracture of the pelvic bones. In six of the sixteen cases in this series the small bowel was perforated, and the bladder ruptured four times, whilst the sacrum and pelvic bones were fractured in eight cases.

In extraperitoneal wounds of the rectum the only hope of success lies in very free local drainage carried out at the earliest possible moment. With a view to establishing efficient drainage I removed in two cases the uninjured coccyx in addition to free drainage of the wounds of entry and exit, and found by stripping up the bowel that one was able to expose the wound in the rectum and was thus enabled to drain and pack off the surrounding parts and prevent further tracking by retroperitoneal hæmorrhage. Both patients succumbed from other complicating wounds, but I think in suitable cases the plan is a good one.

I was much interested in Mr. Gordon Taylor's enterprising suggestion which he carried out on two occasions, viz., removal of the rectum by an abdomino-perineal route in order to overcome the acute sepsis, for the results in these cases are so bad that desperate measures are well worth a trial.

Too much must not be expected from colostomy in subperitoneal wounds of the rectum especially in the type already described, where the impact from a blow on the sacrum produces a laceration of the underlying muscular tissue and rupture of the small vessels, resulting in very rapid gangrene of the mucous membrane of the gut. And likewise where the actual gangrenous patch occurring in the bowel is separated from the track of the missile by a layer of undamaged tissue as in the case of B., Case IV (p. 29). This ulceration and gangrene may occur as early as seven hours after the infliction of the wound.

In these cases where the infection to the retroperitoneal tissue is established almost directly, colostomy can be of little service as a primary measure as one might just as well perform an enterostomy in the hope of curing a case of acute gangrenous appendix; indeed the only rational line to adopt is to expose, and, if possible, excise the local gangrenous patch in the rectum. Moreover we know that the intestine remains motionless for a considerable time after injury, as has been proved clinically when the missile has taken a superficial course through the abdominal wall and wounded the small intestine in its immediate neighbourhood, the injured coils being found in the same place after a considerable interval.

In conjunction with Sir Cuthbert Wallace I performed the following simple experiment which bears on this point. A dog was given a bismuth meal and six hours later X-rays showed the stomach empty and the bismuth well into the small intestine. The experiment was repeated with the addition that the animal was anæsthetized for half an hour before the X-ray examination, when the bismuth as before was found to have passed into the small bowel, proving that the anæsthetic does not interfere with the motility of the hollow viscera. A third experiment was carried out when laparotomy was performed after a bismuth meal and a loop of small intestine removed with end-to-end anastomosis; at the end of six hours all the bismuth still remained in the stomach; we carried out other experiments on cats which also proved that injury of the large bowel resulted in a complete temporary stasis.

Colostomy may be of service at a later stage of treatment in subperitoneal rectal wounds especially in cases complicated by compound fractures of the sacrum, but the primary aim must lean towards very complete local drainage.

Rutherford Morison has suggested that in certain cases of gunshot wounds of the rectum with entry and exit wounds in the buttock, such as those passing through the great sacro-sciatic foramen, the rectum should be exposed by an incision down one side of the sacrum fully exposing the wound in the rectum and post-rectal tissue.

Case I.—E., aged 20. Admitted six hours after wound, very ill, pulse 125, extremities cold. Abdomen rigid down left side, most marked in iliac fossa. Catheter passed showed normal urine in bladder. Entry wound over right great trochanter of right femur the size of a bantam's egg (no exit wound). Severe wounds in both legs. Operation eleven hours after being wounded. Wound over left trochanter explored; it passed behind left hip-joint and into pelvis through great sacro-sciatic foramen. Wound laid open and a large piece of shrapnel removed from middle line behind rectum and in front of the sacrum. Large tube drain down to old site of missile. No sign of missile. The abdomen was opened in middle line below umbilicus. There was extensive retroperitoneal hæmorrhage over bladder and rectum but no intraperitoneal injury of gut. Abdomen closed in layers. The following day the temperature was 103° F. and pulse 142. Patient looked extremely ill and was restless. There was a foul discharge from buttock wound. Death twenty-four hours later. Postmortem showed extensive wound of muscular wall of rectum without any injury to the mucosa. (There was scanty blood in the peritoneum.) There was much retroperitoneal hæmorrhage in pelvis and over the left psoas muscle. Gas bacilli of *perfringens* type were found on film and culture from the retroperitoneal hæmorrhage.

Case II.—G., aged 40, was admitted to hospital in fairly good condition; temperature and pulse normal. There was an entry wound at a point just posterior to the great trochanter of the left femur the size of a two-shilling piece. The exit wound was over great sacro-sciatic foramen of the same size as entry wound. Operation one and a half hours after admission. Both exit and entry wounds were freely opened up. No sign of injury to the rectum was made out. Two days later wound smelt very foul and patient looked extremely ill. Temperature 102° F., pulse 140, and he vomited freely. An anæsthetic was given and entry and exit wounds were joined up by a fresh incision, and wound packed with eusol gauze. Patient gradually became worse and died five days later from acute toxæmia. Post-mortem showed retroperitoneal hæmorrhage over the pelvic peritoneum and meso-rectum and a gangrenous patch in rectum wall, 1 in. square, which had not yet separated.

Case III.—R., aged 24, was admitted six hours after injury very shocked from multiple wounds. There was a shell wound in right buttock close to coccyx. The gluteus maximus muscle was exposed and the wound extended into the rectum, and there was a hole in posterior rectal wall which admitted two fingers, and a perforated wound of the chest on right side at lower angle of scapula. Operation eight hours after admission. Wound cleaned up under a local anæsthetic and freely drained. On the following day a left colostomy was performed. He made a good recovery and left hospital eight days after admission. Temperature and pulse normal.

Case IV.—B., aged 24, was admitted six hours after being wounded, very shocked, and pulse 120. There was an entry wound the size of a two-shilling piece over the right sacro-sciatic foramen. Exit wound over sacro-iliac joint same side the size of a five-shilling piece. The right size of sacrum had been very severely damaged, leaving only the coccyx intact. Blood oozed freely from exit wound mixed with liquid fæces. Operation eleven hours after being wounded, when wounds were joined up and superficial part of gluteus maximus muscle divided. The rectum was then fully exposed as the sacrum in the line of wound had been blown away. There was a bruised wound on posterior rectum wall the size of a five-shilling piece. This was cleaned well with eusol, packed with eusol gauze, and large drainage tube inserted down to site of injury. A left colostomy was then rapidly performed. The patient died shortly afterwards. Post-mortem, by Dr. John Shaw Dunn, showed an entry wound, the size of a half-crown, present on the right buttock over the great sacro-sciatic notch; the exit wound, much larger, centred round the site of the right sacro-iliac joint. The greater part of the right side of the sacrum, and the posterior end of the iliac blade, had been carried away by the missile. The perirectal adipose tissue, which was exposed in the wound, was not actually lacerated, but exhibited much diffuse hæmorrhage over an area 2 in. in diameter. The appearance of the tissue indicated commencing gangrene. The rectum was removed, and opened from the front. A large and deep ulcer was found on its posterior wall corresponding in site to the hæmorrhage seen posteriorly. The lesion measured 2½ in. across and 1¾ in. in length, and had a hæmorrhagic, gangrenous base composed of perirectal adipose tissue. Two areas of superficial ulceration, associated with hæmorrhage, were present on the rectal mucosa at a lower level. The appearance of the large lesion was very suggestive of the actual loss of a large portion of the muscular and mucous coats, but in view of the fact that the injury took place only thirteen hours earlier, it is apparent that the gap must be explained by the retraction of torn edges of tissue. The tear was evidently not produced by immediate contact of the missile, for behind it the layer of adipose tissue was ⅜ in. in thickness. The superior hæmorrhoidal artery was traced downwards through this tissue to its finer branches. The wall of the vessel showed some hæmorrhagic staining, but no actual rupture. The smaller vessels entering the damaged muscle tissue were ruptured.

Case V.—D., aged 25, was admitted to hospital four hours after being wounded. General condition good, pulse 90. There was a shell wound of right buttock on level with hip joint and posterior to it. There was some hæmorrhage from the rectum. On examination of rectum a wound was felt on posterior wall about 2 in. in length about the level of the fourth sacral vertebra. On passing a catheter normal urine was drawn from the bladder. Operation an hour after admission. Spinal anæsthesia. Laparotomy. No intraperitoneal injury. Left colostomy performed. Pelvic colon was brought out divided across. Paul's tubes put into the proximal and distal ends. The coccyx was removed and a wound in rectum exposed. Tube inserted into rectal wound through coccyx incision. The original wound was also drained with a large tube. Before leaving table the distal end of colostomy was washed through with warm eusol and out through wound in rectum, fæcal matter escaping freely from tube in coccyx wound. Patient did well for two days, when his pulse rose to 120 and he commenced to vomit. In spite of persistent stomach lavage he vomited persistently and suffered from great abdominal distension and died four days after injury from retroperitoneal sepsis. Post-mortem showed there was no evidence of a general peritonitis but the retroperitoneal region was distinctly affected. The infection had reached up to the level of the kidneys. The missile, a piece of shell ¾ in. by ½ in. was found lying to the left of the rectum in a depression which proved to be an exit wound in the rectum itself. The shell fragment had passed through the rectum from right to left in a slightly upward direction below the level of the reflection of the peritoneum. The bladder was severely bruised but not torn. It showed some marked evidence of acute inflammation in the mucous membrane, which was intact and dark plum coloured.

Case VI.—S., aged 35, was admitted to hospital almost pulseless and died half an hour after admission. There was a rifle bullet wound 1½ in. above and behind great trochanter of the left femur from which much blood escaped. No exit wound. Post-mortem showed the lower abdomen and pelvis to contain much clotted blood. There was considerable hæmorrhage into the mesentery of the rectum and recto-vesical pouch; so much so that it filled the whole true pelvis. The whole of the pelvic colon and rectum were plum-coloured, due to extravasated blood both in the mesentery and under the peritoneal covering of the bowel. A rupture was found in the rectum opposite the third part of the sacrum. Dissection showed the superior hæmorrhoidal artery torn across. The bullet had crossed the pelvis and left it through a hole in the obturator foramen on the right side, lodging in the gluteus medius muscle.

Case VII.—L., aged 22, was admitted eight and a half hours after injury in poor condition, pulse 110. Rigidity of lower half of abdomen with tenderness. Small entry wound of rifle bullet over right side of sacrum opposite third part. Operation ten hours after injury. Spinal anæsthesia. Middle line incision. Marked extravasation of small gut contents in lower abdomen. Loop of ileum within 1 ft. from cæcum contained eight holes. Resection of damaged loop, lateral anastomosis. Intraperitoneal perforation of bladder on right posterior wall—sutured. A damaged rifle bullet lay in bladder and was removed. Perforation of rectum at reflexion of peritoneum; the tear was 2 in. in length and extended to the retroperitoneal portion of gut to sacrum. Left colostomy was performed, also drainage of bladder. Great difficulty was experienced in closure of hole in rectum. The entry wound on sacrum was enlarged, cleaned and drained. On following day his pulse was 140 and temperature 99·4° F. He vomited in small quantities frequently: 100 c.c. of intravenous eusol was given; vomiting continued, and third day after operation stomach lavage was commenced. He got gradually worse, and could only be kept quiet by repeated doses of morphia, and he died on fifth day. Post-mortem showed general peritonitis on the small intestine area to be healing up well, and there was no great distension of the bowel. Pelvis was fairly clean, and the wound in rectum looked like healing. There was a very well developed retroperitoneal cellulitis around the base of the bladder and the adjacent part of the rectum. The bullet had passed through the sacrum without much damage to the bone.

Case VIII.—G., aged 32, was admitted to hospital half an hour after injury. He looked very ill, and his pulse was 96. The abdomen was tender and rigid, the rigidity more marked on left side. There was an entry and exit wound over right and left buttocks at the level of the lower part of the sacrum: each wound the size of a shilling. The fæces escaped from the wound on left side. Operation an hour after injury. Both wounds freely opened up and the rectum exposed on left side. Free drainage with large tube. The abdomen was opened. Laparotomy. No injury to intestine or peritoneum was found, but a large retroperitoneal hæmorrhage over the whole of the rectum and bladder practically filling the true pelvis. Left colostomy was performed. The left testicle, which was severely lacerated, was removed. Death from shock and hæmorrhage four hours after being wounded. Post-mortem showed that the missile had passed into the great sacro-sciatic foramen at right side and out at the left. There was a large hole on the posterior wall of the ampulla of the rectum.

Case IX.—F., aged 26, was admitted to hospital one and a half hours after injury, suffering from considerable shock: pulse 100 and temperature 96·2 F., and deeply under the influence of morphia. There was an entry wound in the left adductor region the size of a five-shilling piece, while the exit wound in the region of the rectum was of the same size. The missile (a shell fragment) had tracked deeply through the adductor muscles, blowing away a large portion of the perineum and gluteal muscles from their attachments to the sacrum and coccyx. Operation two hours after injury. The adductor muscles, very much destroyed, were cleaned up and dead tissue removed. The missile had fractured the descending ramus of pubic bone of the left side and ruptured the urethra completely across in front of the prostate. Great destruction of the gluteus maximus muscle which was partially removed. The rectum and anus were completely detached from the surrounding structures and lacerated. The pelvis was packed with flavine gauze. The abdomen was opened and perforation found in the lower end of ileum close to cæcum. This was sutured. Colostomy was performed. Patient lived nineteen hours after injury and died from shock.

Case X.—R., aged 25, was admitted two hours after injury, pulse 120, suffering from marked shock. There was a small entry wound due to shell fragment the size of a sixpenny piece over the right upper aspect of the sacrum, from which fæces escaped. His condition did not permit of any operative treatment, and he died twenty hours after admission. Post-mortem showed the abdomen contained much blood. There was a perforation of the lower jejunum. The pelvic colon showed two large perforations in centre of loop, one extending into the mesentery. The rectum was wounded on right lateral wall. The missile, having passed through bowel, entered the recto-vesical pouch 1½ in. above the reflection of the peritoneum. It then passed in and out of bladder, passing the posterior and left lateral wall. The missile (a small fragment of shell) lay on the brim of the pelvis in the parietal peritoneum covering the psoas muscle of the left side.

Case XI.—T., aged 37, was admitted to hospital five hours after injury, very shocked, pulse 132. There was a small entry wound in the right rectus muscle, midway between the umbilicus and pubes. There was a large exit wound 3 in. by 2 in. over the sacrum on same side. His general condition was considered to be so poor that it did not permit of any surgical treatment further than drainage locally of the exit wound of sacrum. This was freely opened up and fæcal matter escaped from the rectum. Patient never picked up, and died on third day. Post-mortem showed general peritonitis. There was a perforation in the ileum 3 ft. from the cæcum, and an in-and-out wound of the bladder towards its base on the right side. There was also an in-and-out wound of the retroperitoneal portion of the rectum opposite the third sacral vertebra. The same portion of the sacrum was very much destroyed, the track of the missile being through the rectum notching the symphysis pubis, through the ileum, through the bladder and rectum out through the wound in the sacrum.

Case XII.—H., aged 23. Admitted seven hours after being wounded by a rifle bullet. He did not appear to be shocked to any great extent; pulse 88, blood pressure 120. He complained of great abdominal pain and lay on his left side. Abdomen retracted; it moved little and was generally rigid and tender. There was a small entry wound midway between umbilicus and pubis in the middle line. The exit wound, also small, was situated at the sacral margin on the right side opposite the third portion. Operation, three quarters of an hour after admission to hospital. The exit wound was first dealt with by enlarging the skin wound where there was a fracture of the sacrum which communicated with the rectum and peritoneal cavity. Tube drain down to peritoneal cavity. A mid-line incision was made below umbilicus. The belly cavity contained a quantity of blood, and the omentum was found in upper abdomen having shrunk away from the area of infection. The ileum was perforated in twelve places and, at a point 5 in. from its lower end, was completely divided across. The majority of perforations were large wounds in the gut. The pelvic colon was ruptured on its convex surface in the middle of the loop. All perforations in both large and small gut were sutured. The bullet had passed into pelvis, through the recto-vesical pouch, through the rectum, but without any injury to the bladder. The rectal wound could not be reached for treatment by suture. A tube was inserted into pelvis after washing out with saline, and the abdomen closed in separate through-and-through sutures. Patient died of shock twenty hours after being wounded. Post-mortem showed the wound in the rectum to be 1 in. above the floor of the recto-vesical pouch; the hole in the gut was $\frac{3}{4}$ in. from above downwards, and the right lateral wall of the gut had been opened. There was no retroperitoneal hæmorrhage, the blood having escaped either into the abdomen or through the exit wound in the sacrum. The fracture in the sacrum involved the third and fourth portions of bone.

Case XIII.—H., aged 19, was admitted to hospital three hours after injury by rifle bullet; pulse 124, temperature 103·6° F., blood-pressure 110. The abdomen was neither rigid nor tender. He complained of considerable pain in the inguinal region. There was a small entry wound over the right inguinal canal the size of a threepenny bit. The exit wound was the size of a two-shilling piece over the left sacro-sciatic foramen. There was a considerable amount of blood and fæces leaking from it. Operation under gas and oxygen; the exit wound dealt with by free incision through gluteal muscle. The wound, which lay through the sacro-sciatic foramen, was freely opened up into the rectum. It was washed well with flavine and packed lightly with flavine gauze. The abdomen was opened in the middle line but no peritoneal injury was found. Patient's recovery was satisfactory until the third day when he had a very severe secondary hæmorrhage from within the pelvis. An anæsthetic was given and the wound in pelvis explored through buttock. The hæmorrhage had ceased and its origin could not be ascertained with certainty. The rectum was packed lightly with gauze and removed twenty-four hours later. Six days after injury he had another violent hæmorrhage and died in a few minutes, blood coming from rectum and the wound through the great sacro-sciatic foramen. Post-mortem showed an extensive retroperitoneal sepsis and a large necrotic perforation in rectum. The extensive retroperitoneal sepsis which involved the whole of the true pelvis made it impossible to find the origin of the hæmorrhage.

Case XIV.—G., aged 32, was admitted to hospital one and a half hours after being wounded. Death six hours after injury. Pulse 104, respirations 22, temperature 96° F., blood-pressure 40 systolic. There was an entry wound over the upper part of the right buttock the size of a threepenny bit. The exit wound was the size of a shilling, and was situated on the left side of the umbilicus on the rectus muscle. Through this wound the pelvic colon prolapsed. Post-mortem showed the abdomen was full of blood, due to a perforation of the mesentery in the pelvic colon and the mesentery of the small intestine. There were seven perforations in the ileum, which was completely divided in two different sections. The pelvic colon itself had a large tear on the antemesenteric border. There was a perforation of the rectum just below the reflection of the peritoneum on the right side of the gut wall.

Case XV.—H., age not stated, was admitted to hospital six hours after injury with pulse 120. He suffered from shock and was very cold. There were multiple wounds of back, thighs and both buttocks. The abdomen was rigid and tender over the right side but not so on the left. There was a distinct improvement four hours after admission, when he was operated upon. The abdomen was opened in the middle line and the small bowel 6 in. from the cæcum. Perforations sutured. No other gut injuries. There were two wounds, one in each buttock. The missile, a fragment of shell, had passed through the left sacro-sciatic foramen, pierced the rectum and passed out of the pelvis through the right sacro-sciatic foramen. The wounds were thoroughly opened up, the rectum being drained with large tubes, one from each side. Patient died from shock and hæmorrhage shortly afterwards.

Case XVI.—G., aged 26, was admitted to hospital four and a half hours after injury. General condition good, pulse 92. Abdomen normal with retention of urine. Catheterization of bladder showed 18 oz. of normal urine. There was a wound over the left aspect of the coccyx that easily admitted two fingers. The exit wound, over the third part of the sacrum was 2 in. long and ½ in. broad. Fæces and blood leaked from this wound, on removal of first field dressing. Operation half an hour after admission. Spinal anæsthesia. Left colostomy done by muscle-cutting incision, pelvic colon divided across and Paul's tube inserted into proximal and distal ends. Patient turned on the left side and the skin between the in-and-out wounds was completely laid open. It was then found that the whole of the posterior wall of the rectum, in the whole length of the wound was torn across (from coccyx to third sacral vertebra). The wound was washed clean with cusol and packed lightly with cusol gauze. All hæmorrhage stopped and fæcal matter removed. Patient did well and went to base a fortnight later feeling well.

Sir GORDON WATSON: Mr. Drummond's communication will form a most valuable record for our *Proceedings* because it is an account of his own work: a series of cases most carefully recorded during the stress of war work at a busy casualty clearing station. I was personally in touch with some of the work Mr. Drummond did at a casualty clearing station, and I saw some of his cases of ulceration of the bowel following concussion wounds, at the time they were investigated.

Section of Surgery.

President—Sir JOHN BLAND-SUTTON, F.R.C.S.

DISCUSSION ON THE SURGERY OF THE PITUITARY GLAND.[1]

Mr. PERCY SARGENT, C.M.G., D.S.O., F.R.C.S.

IT is only during the past fifteen years or so that the pituitary gland has been brought within the reach of surgery, or that much solid knowledge of its functions and its diseases has been gained. It is not my intention to speak of its physiology nor of the inter-relation of its activities with those of other ductless glands, but to suggest for discussion a certain number of points connected with the surgical aspects of the diseased gland. Few individual surgeons can have had a sufficiently large personal experience to enable them to form more than a provisional opinion upon the many questions which are offered by a new field of surgery such as this. My own experience is merely large enough to make me realize how much has yet to be learnt before the operative treatment of pituitary lesions can be established upon a firm basis.

If the arbitrary system which apportions the various organs and their afflictions amongst different specialists was formerly unable to find a sponsor, other than the psychologist, for the pituitary body when its functions were totally unknown, it is scarcely less at a loss at the present time, seeing that disease of the gland may manifest itself in so many different ways. A patient may be brought in the first instance to the ophthalmic surgeon for disturbances of vision, to the physician for adiposity or glycosuria, to the pediatrist for infantilism, to the neurologist for epilepsy, or to the gynæcologist for amenorrhœa; the ever-increasing activities of the surgeon naturally include the gland within their orbit; and the rhinologist puts in his claim on the dual ground that the pars anterior is developed as an outgrowth from the stomodæum, and that one of the paths which lead to it is his right of way.

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[1] At a meeting of the Section, held December 4, 1919.

Lesions of or implicating the pituitary gland give rise to symptoms which fall into three groups:—

(I) Those dependent upon disordered function, whether in the direction of excessive or of deficient activity, or of a combination of both. (For our present purpose there is no need to take into account the dual constitution of the gland.)

(II) Those which result from an increase of the general intracranial pressure.

(III) Those due to pressure upon neighbouring structures, particularly the optic chiasma.

These groups are not, of course, sharply marked off from one another, and in most cases the clinical picture is made up of all three in varying proportions.

Tumours which cause pituitary symptoms do not necessarily arise in the gland; interpeduncular tumours, frontal lobe tumours, and growths involving the base of the skull may cause pressure upon it and the surrounding structures, whilst symptoms due to disordered pituitary function are also met with in hydrocephalus. It is important to bear in mind such cases, since the glandular symptoms may be sufficiently prominent to cause errors of diagnosis, and to lead to misdirected surgical procedures.

I shall endeavour to deal first with the question as to what effects upon the symptoms of pituitary disease are to be expected from operative treatment; and, secondly, what is the best method of surgical attack.

(I) GLANDULAR SYMPTOMS.

It is now commonly believed that gigantism and acromegaly are manifestations of excessive activity of the pituitary gland, while infantilism, adiposity, and impotence result from deficient activity, and that there is thus an analogy with the corresponding disorders of function of the thyroid gland. In both cases the symptoms of excessive or deficient functional activity may merge into one another. Further, both thyroid and pituitary tumours cause symptoms due to pressure upon neighbouring structures. If we pursue the analogy, we should expect that as the symptoms of Graves's disease can be modified by the removal of the greater part of the diseased thyroid, so partial excision of the hypophysis might be expected to benefit a progressive acromegaly. Whether or not an operation for glandular symptoms alone would be justifiable is a point upon which I am unable to express any opinion. So far as I am aware Cushing is the only surgeon who has operated for dyspituitarism unaccompanied by any other symptoms except headache. He relates two cases of this kind. Both were adult male acromegalics, with histories extending over eight and fifteen years respectively. In one of them the partial removal of the pars anterior by the nasal route was followed by immediate cessation of the headaches, and three months later there was a marked change in the patient's appearance. In the other the sella was but slightly enlarged. A subtemporal decompression afforded no relief of headache, but later some improvement was noted after a course of whole-gland extract to the extent of 100 gr. a day.

In a third case of dyspituitarism uncomplicated by pressure symptoms other than headache, the predominating signs were those of deficient glandular activity. The patient was a fat amenorrhœic, aged 24. Treatment by glandular feeding was carried out for a time with some slight improvement but later the headaches became more severe and the vision began to fail. A sellar decompression was then done, with prompt cessation of the headache. This patient resumed work a week after the operation, but no further information as to her subsequent history is given. This cannot, however, be regarded as an operation for glandular symptoms alone, as it was not performed until, whilst the patient was under observation, visual deterioration began.

I have no experience of operating for glandular symptoms alone, and I should hesitate greatly before attempting the experiment in a patient with little or no enlargement of the sella, in whom neither headache of any severity nor neighbourhood symptoms existed. In the opposite class, however, with a large sella easily accessible through the nose, and with a progressive Frölich syndrome, the operation might be more readily undertaken. For it is possible that the relief of pressure upon any remaining normal gland tissue, afforded by the emptying of a cyst, the removal of a mass of adenomatous tissue, or by merely incising the dural capsule, might be beneficial.

(II) SYMPTOMS OF INCREASED INTRACRANIAL PRESSURE.

This group need not detain us, for the onset of general pressure symptoms marks the beginning of the terminal stage. A simple decompressive operation will abolish the headache due to general intracranial pressure, but could not be reckoned upon to relieve the "bursting" frontal pain due to sellar distension. In those cases which show papillœdema, subsidence of the swelling is to be expected after decompression. It is probable that a simple decompressive operation will prove to be a useful, if not an essential, preliminary measure before making a frontal attack upon a pituitary tumour.

(III) NEIGHBOURHOOD SYMPTOMS.

It is chiefly for visual disturbances that the pituitary region has hitherto been subjected to surgical interference. In Cope's admirable paper on "The Pituitary Fossa and the Methods of Surgical Approach thereto,"[1] the following extremely important observation occurs, which is completely in accordance with my own experience. Cope says that after a thorough search of the literature of pituitary operations, he had not come across a single case with optic symptoms, in which verification at operation or autopsy was permitted, which did not show a large intracranial extension; and concludes that all pituitary tumours which come to operation for symptoms other than acromegaly have long before burst the bounds of the fossa. This fact should be given due consideration when the merits of the different methods of approach are being weighed.

NATURE OF PITUITARY TUMOURS.

The majority of the tumours which arise in or primarily involve the pituitary gland appear to be of an adenomatous character, and are called by Cushing "chromophobe struma," that is to say, an adenomatous hyperplasia characterized by deficiency or absence of the eosinophilic granules, which seem to be associated with functional activity. Of Cushing's own cases twenty out of twenty-six were of this nature; in Hirsch's, seventeen out of twenty-two; and of the seventy-four cases collected from various sources by Frazier, there were no fewer than thirty-nine. Taking these three series together, 62 per cent. of the tumours subjected to operation were of an adenomatous character.

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[1] *Brit. Journ. Surg.*, 1916-17, iv, pp. 107-144.

Frazier's seventy-four collected cases contain eleven instances of cyst or cystic tumour. It is possible that this proportion (nearly 15 per cent.) is too high, seeing that cysts are the most easy form of pituitary enlargement to deal with, and, further, as they give the best immediate results, are the most likely cases to be reported. There are, at least, ten instances of malignant tumour in this series, or about 14 per cent.

Another kind of tumour, not arising in, but secondarily affecting the gland, is the suprapituitary endothelioma.

I think that we may at once put aside the possibility of extirpating a solid pituitary tumour in the sense in which an adenoma can be removed from the breast, or a simple meningeal tumour from the brain. Hitherto, so far as I am aware, any removal that has been accomplished has been only partial. Nevertheless a sufficiently large part of such a tumour can be removed to afford great relief, at any rate for a time.

METHODS OF APPROACH.

A large number of operative procedures for approaching the pituitary fossa have been proposed and practised. They fall into two groups, the *extradural*, including transpalatal, nasal, and paranasal operations, and the *intradural*, comprising the temporal and the frontal. Special mention must be made of the temporal route, because it was so strongly advocated by Victor Horsley, the pioneer of pituitary surgery, as of cerebral surgery in general. In 1906 Horsley had already operated upon nine cases, and at that date he wrote that the "prehistoric way of raising the frontal lobe was not the proper way to remove the tumour; it should be done by raising the temporal lobe." The only recorded cases of his which I have been able to find are mentioned in Tooth's exhaustive analysis of 265 cases of brain tumour operated upon between 1902 and 1911 at the National Hospital, Queen Square. These cases are four in number; in all the temporal lobe was raised, the diaphragma sellæ incised, and tumour partly removed. One patient died six hours after operation; one lived nine months, another three years, and another eight years. Ophthalmoplegia followed the operation in two of them, and aphasia and hemiparesis in two.

At present the two methods of approach which hold the field are the nasal and the frontal. Of the former the trans-sphenoidal operation of Cushing is probably the best; of the latter the orbito-frontal operation of Frazier may be taken as the type. For the sake of brevity, I shall speak of the nasal and the frontal route. To some extent these are rival methods, but I think that experience will show that neither should be altogether rejected. In time it may be possible to make a sufficiently accurate diagnosis to enable one or other to be selected for any individual case. I do not propose describing the operations in detail, but merely to give a brief general outline.

(1) *Cushing's Nasal Operation.*—A transverse incision an inch in length is made through the frænum of the upper lip, and through this incision the mucous membrane is separated from both sides of the septum. When this can be accomplished without button-holing the mucous membrane, the entire operation is conducted without invading the nasal cavities. A long bivalve speculum is introduced, one blade on either side of the septum between it and the mucous membrane. Part of the septum is now removed, including portions of the vomer, septal cartilage, and median plate of the ethmoid. The anterior and inferior walls of the sphenoidal sinus are removed, when the floor of the pituitary fossa is brought into view. This, which is sometimes represented merely by a thin flake of bone, having been removed, the tumour, enclosed in its dural capsule, comes into view. A crucial or stellate incision is then made through the dura. The operator may be fortunate enough to have opened into a cyst, but when, as is more often the case, he finds himself confronted with a solid mass of tissue, the next step presents difficulties. It may be that the compressed gland, capable of functioning once the pressure has been relieved, lies between him and the tumour. Should there be any doubt as to this a fragment should be removed for microscopic examination, and further interference postponed. A fragment of an adenomatous tumour can generally be recognized by the naked eye. It is pale and comparatively bloodless, so that its removal by means of a curette is accompanied by but little bleeding. The extent to which such removal may be carried out can only be a matter for experience to decide. With a very large fossa and a bulging tumour, there is no great difficulty in carrying out this operation, but when the sella is small, one may, by directing the line of approach too high, enter the cranial cavity through the ethmoid in front of the pituitary fossa. This accident is at once evidenced by the escape of cerebro-spinal fluid, and by it the danger of meningitis is enormously increased.

(2) *The Frontal Route.*—Frazier's operation commences with the reflection of an osteoplastic flap consisting of a large part of the frontal bone, hinged in the temporal fossa. The supra-orbital margin is resected and removed, to be replaced at the termination of the operation,

and the greater part of the orbital roof is cut away. The orbital contents are now depressed by one retractor whilst the frontal lobe, enclosed in its dural covering, is elevated by means of a second retractor until the optic foramen comes into view. The dura is next opened, and the retractor introduced through the incision so as to elevate the frontal lobe and expose the optic chiasma and the tumour, which can then be partially removed by means of a curette. The operation, thus briefly outlined, is I think capable of a good deal of modification in detail. If, for example, the osteoplastic flap is made a good deal larger than Frazier makes it, the orbital roof need not be interfered with.

Before proceeding to discuss the comparative merits of these two methods of approaching a pituitary tumour, I should like very shortly to relate my personal experience. It would be absurd to speak in percentages of such a small number of cases as I have operated upon —fourteen in all. I do not intend, on the other hand, to weary you with a recital of the details of all these cases, but merely to pick out the salient points with special reference to the difficulties and misfortunes which I have myself encountered.

GROUP I.—SIX CASES OPERATED UPON BY THE NASAL ROUTE.

Two of these were not pituitary cases, but I must refer to them because they illustrate errors of diagnosis leading to misdirected surgical procedures. One was a man with frontal headache and symptoms of chiasmal pressure; the X-ray showed a somewhat enlarged pituitary fossa. The operation of sellar decompression was performed; no improvement followed, and the man died a few days later. Post mortem, an infiltrating frontal lobe tumour was found. The second of these two non-pituitary cases was that of a girl, aged 12, with a three months' history of failing vision, polyuria, increasing adiposity, and somnolence. There was a visual defect in the upper part of the temporal fields. The sella, radiographically, was somewhat enlarged. In the belief that we were dealing with a pituitary tumour, I opened the sella by the nasal route. On curetting the supposed tumour a sudden gush of cerebro-spinal fluid occurred. I placed a small piece of silver wire in the fossa for purposes of radiographic orientation, but the child died twenty hours after the operation. Post-mortem, the silver wire was found lying partly in the substance of the pituitary gland, and partly projecting through the diaphragma sellæ, exactly in the sagittal plane. The third and lateral ventricles were greatly dilated, and their lining membrane showed an intense, hæmorrhagic, purulent inflammation. A small glioma involving the corpora quadrigemina completely blocked the aqueduct of Sylvius.

The other four cases operated upon by the nasal route were typical instances of dyspituitarism with visual defects and gross enlargement of the sella turcica.

(1) One was an undeveloped boy, aged 20, somewhat adipose, and of feminine type. He suffered from severe frontal headaches, and his vision was so far gone that he could only see, and that very defectively, in the upper part of the temporal field of one eye and in the nasal field of the other. A sellar decompression was done, and some of the enlarged gland was removed. This tissue proved to be a hyperplasia of the pars anterior. Severe facial neuralgia and considerable febrile disturbance followed this operation, but these symptoms subsided after the escape of a quantity of pus into the nasopharynx. Headache was relieved, and some little improvement of vision resulted. The boy died of pneumonia about fifteen months later.

(2) Another case in this group is an acromegalic upon whom I operated only a few weeks ago. A cyst containing chocolate-coloured fluid was emptied by the nasal route. The pituitary headache has gone, but as yet there is no improvement of vision.

(3) The third case died of meningitis, the cranial cavity having been opened into through the posterior part of the cribriform plate at the same time that the pituitary fossa was entered. Post mortem there was found a large intracranial extension of the growth.

(4) The fourth case was an undersized, effeminate boy, aged 21, with symptoms of hypopituitarism and frontal headache of two years' duration. One eye was completely blind, and in the other the temporal field was gradually being lost from above downwards. The immediate effect of the sellar decompression was very striking, and three months later the size of the visual field had been almost completely restored.

GROUP II.—SEVEN CASES OPERATED UPON BY THE FRONTAL ROUTE.

Three of these I pass over, because a thorough exposure of the interpeduncular region failed to reveal any tumour. There are some extremely interesting points about these cases which have little bearing upon the present question. I only mention them, first, to illustrate the fact that conditions other than tumour in this region may occasion symptoms upon which a diagnosis of pituitary tumour may be based, and, secondly, to show that a frontal exploration of the interpeduncular space can be performed with very little risk. All three patients made an uninterrupted recovery from the operation, and two have shown a remarkable improvement in their symptoms. The other four cases had pituitary tumour with large intracranial extension. In one I removed, piecemeal, the greater part of the tumour. The patient was up and about in ten days, and exhibited very marked improvement of vision. Unfortunately the war interfered with the following up of this case, and since my return I have been unable to trace the patient.

The three fatal cases in this group well illustrate the misfortunes which may be encountered:—

(1) A man with well marked hypopituitary symptoms, a considerably enlarged sella, and progressive failure of vision, had twice previously been operated upon by the nasal route, and each time the operation had been followed by some temporary visual improvement. I operated by the frontal route, and evacuated a large cyst containing chocolate-coloured fluid. From this fluid a *Staphylococcus aureus* was subsequently grown, so that had he survived the operation he might have succumbed to meningitis. Towards the end of the operation, however, which up to this point had been free from any difficulty, the elevated frontal lobe suddenly began to swell and to protrude. A hæmorrhage had clearly taken place into it. The patient succumbed a few hours later.

(2) A man, aged 23, an acromegalic, had suffered for three years from facial neuralgia, failing vision, and a periodic discharge from the nose. Two months previously some growth had been removed, by the nasal route, from the enlarged sella. No relief had been afforded, and since the operation he had suffered from unquenchable thirst and polyuria. I approached the pituitary region by the frontal route. Immediately the dura was opened, and the brain elevated, the optic nerve came into view, flattened and stretched over a soft dark lobulated tumour. As it seemed possible to remove enough of the tumour to relieve pressure upon the chiasma and the other optic nerve, and as the eye on the side of the operation was practically blind, it was decided to divide the nerve. This was done, and a beautiful exposure of the tumour and sella was obtained. Directly, however, I began removing the tumour piecemeal a severe arterial hæmorrhage occurred, which could only be controlled by plugging and suturing the flap over the now protruding frontal lobe. Death occurred six hours later. Post mortem

a very large tumour was found occupying an enormously enlarged interpeduncular space. The circle of Willis was greatly stretched out over the tumour, and it was from one of its branches that the hæmorrhage had taken place.

(3) The third fatal case was that of a man, aged 35, with symptoms of hypopituitarism and bitemporal hemianopia. The frontal operation presented no difficulties and a large part of the tumour was removed piecemeal from the interpeduncular space. There seemed to be no reason why this operation should not have been successful, but the patient never recovered consciousness and died on the second day. Post mortem nothing was found to account for the fatal result.

The last of my fourteen cases was one of a large suprapituitary endothelioma. The only symptom for years had been a progressive failure of vision. When I was asked to operate the patient was practically blind, and comatose. A bilateral subtemporal decompression proved unavailing. It is of course ludicrous to include a case like this in any statistics of pituitary surgery.

In attempting to make a fair comparison between the two methods of operating upon pituitary lesions, we ought to bear in mind not only the route of approach, but also what can be accomplished when the objective has been reached. The nasal route presents several disadvantages. It is narrow and unclean, and the difficulties may be much increased by acromegalic deformity of the bones. Even in the most favourable circumstances very little room can be obtained for dealing with a solid tumour, and when (as is always the case when symptoms of chiasmal pressure are present) there is an intracranial extension, it is only the smallest part of the tumour which can be attacked. The relief afforded by removing this part of the mass, or even by evacuating the contents of a cyst, is usually small and transient. On the other hand the frontal operation is free from unavoidable risks of meningeal infection; and allows the intracranial part of the tumour to be inspected and dealt with. Its chief dangers are illustrated by my own failures. One is that the frontal lobe may be injured. Another, that of serious and even fatal hæmorrhage, depends upon the fact that the circle of Willis is stretched out around the tumour, and so may easily be damaged. But if these dangers can be avoided (and increased experience should diminish the risks) a much more radical and efficient operation can be accomplished.

I have referred to the results obtained by the nasal operation as inadequate and transient. This opinion has been reached not only as the result of my own small experience, but from a careful review of the cases so fully recorded by Cushing in his book on "The Pituitary Body and its Disorders." There are thirteen cases described, having both glandular and neighbourhood symptoms; in seven the glandular, and in six the neighbourhood symptoms predominated. All had visual defects, for which particularly the operation was performed. The results which I quote refer only to the effect of the operation upon vision.

Case 1.—Much improvement noted six months later.

Case 2.—Died next day.

Case 6.—Some improvement noted sixteen months later.

Case 7.—No improvement. Subtemporal decompression two months later followed by improvement.

Case 10.—Marked improvement noted three months later.

Case 11.—No improvement. Died six months later.

Case 12.—Some improvement after three months; vision deteriorating again after fifteen months.

Case 17.—Slight improvement; relapse after twelve months; second operation; death.

Case 19.—Great improvement; relapse after twelve months; second operation; improvement.

Case 20.—Progressive improvement for eight months; thereafter condition stationary.

Case 21.—Slight improvement for four months; relapse; second operation advised.

Case 22.—Considerable improvement after two months; thereafter stationary.

Case 23.—No improvement when discharged from hospital.

To sum up, there are thirteen cases, fifteen operations, two deaths, four greatly improved, five somewhat improved, three not improved and four relapsed after temporary improvement.

It would appear therefore that, from the information at present available, the beneficial effects upon vision of partial removal of a pituitary tumour through the nose, or of merely allowing it to bulge downwards into the sphenoidal sinus, are uncertain, inadequate, and transitory.

The effect upon the severe headache due to sellar distension is more uniform and may be very striking, but recurrence tends to occur.

Of the effect upon glandular symptoms it is less easy to form an estimate, particularly as practically all the cases were treated at the same time with large doses of pituitary gland extract.

It may be that in the future increased knowledge of the functions and inter-relations of the ductless glands may be productive of some medicinal form of treatment capable of arresting the growth of the commonest variety of pituitary enlargement, the adenomatous hyperplastic tumour. In this direction glandular medication has hitherto given no very striking results, though the prospects are not devoid of encouraging signs. It is therefore to surgical treatment that we have to look, at the present time, for the relief of symptoms due to tumour of or in the neighbourhood of the pituitary gland. I would urge that if surgical treatment is to be given a fair field it should be applied at an early stage, and not merely as a last resort in cases rapidly advancing towards a fatal termination. The existence of a high degree of intracranial pressure due to the invasion of the cranial cavity undoubtedly increases the risks of a surgical attack upon the pituitary tumour, just as it does in all other cases of cerebral tumour. I look forward to the time when headache, vomiting and papillœdema will no longer be regarded as the symptoms of cerebral tumour, to be expected and awaited before operation is proposed, but as signs of impending disaster in neglected cases. The work of the neurologist and physiologist has in the past few years enabled many cerebral tumours to be localized accurately at an early stage and dealt with before serious symptoms of intracranial pressure have appeared. There is no reason why pituitary lesions should not be diagnosed at an even earlier stage, for with them we have so many sources of information in radiography, examination of the visual fields and optic disks, and all the symptoms which may be due to abnormalities of glandular function. I believe that, whatever advance in pituitary surgery may take place in the future, it is to be looked for in three main directions: first, earlier and more accurate diagnosis; secondly, a better understanding of the objective to be aimed at in every individual case, so that the most suitable surgical procedure may be selected; and thirdly, improvement in the technique of the intracranial operation.

Dr. Tooth, C.B., C.M.G.

The remarks which I have to make on this subject are somewhat belated. I have had, since the war, only one, perhaps two, cases of pituitary gland disease which may be said to have come within the range of surgery, and, of course, there has been a long hiatus in my work owing to war activities. Therefore my remarks are based on what is already ancient history in a way, namely, the group of cases of pituitary tumour included in a report on all cases of cerebral tumour which came to operation during ten years, 1902-11, communicated to the International Congress.[1] This group numbered fourteen only. And, considering that the symptomatology of pituitary lesions is so distinctive, this seems a small number to have occurred in ten years among a gross total of 500 cases of intracranial tumour, and we may assume that this form of growth is not very common. Of these, four were brought to operation under Sir Victor Horsley, who was, at that period, at his greatest activity. Mr. Sargent has referred to them.

I would like, as a physician, to draw attention to the other side of the picture. We have heard the surgical side, and even Mr. Sargent cannot say very much for it in the present state of our knowledge. If left alone, what is the expectation of life of a patient with pituitary tumour? There were ten cases over and above the four I mentioned as having come to operation. Seven of these I was able to trace; they were alive at the time of the inquiry, which was in 1912. Two I was unable to trace, and one, I found, died two years after leaving the hospital. Therefore I will now only concern myself with the seven cases I could trace, and I propose to give you a very few words about each of them. Two were women, five men. I received answers about all of them. The replies were received in April, 1912.

(1) A man, aged 52, wrote that he was able to work as a light porter. He was almost blind in the right eye. From his first symptom up to the date of my receiving the reply was sixteen years.

(2) A man, aged 25, replied that he was totally blind, and that he suffered from recurring headaches. In his case the survival was three years after the first symptom. I may say that in practically all the cases the first symptom was something connected with the eyes, e.g., hemianopsia or amblyopia.

(3) A woman, aged 31, wrote that she was blind in the right eye, and had very defective vision in the outer field of the left eye. She cannot walk straight, and tends to fall to the left. Duration to the date of inquiry four and a half years.

(4) Girl, aged 13, wrote that she was blind in the left eye, and that the right eye remained in the condition it was in when she left the hospital, namely, with $\frac{6}{18}$ vision. She was quite helpless, but mentally bright. This was four and a half years after the first symptom.

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[1] *Trans. XVIIth Internat. Med. Congr.*, Lond. (1913), Sect. XI, Neuropathology, 1913, part I, pp. 161-257.

(5) Man, aged 35, wrote that he was blind but could get about. He was mentally sound. All that he suffered from, besides his blindness, was occasional slight headache. It was six years after the first symptom.

(6) A man, aged 29. The reply came from the Principal of the Royal School for the Blind, Leatherhead. He wrote that this man was able to work and was making articles for sale, and was doing very well at it, and able to get about. He had no headaches, and was in good health. Survival from first symptom eleven and a half years.

(7) A man, aged 42, wrote that he was able to work as a clerk, and was able to walk about a great deal and enjoyed it, so that he had nothing in the way of paralysis. His left eye was nearly blind. He suffered from headaches occasionally, but only after worry. The period from the first symptom to date was six and a half years.

The longest period was sixteen years, the shortest three years. Averaging them out, we get 7·5 years of life to the time of the inquiry from the first symptom. In the four operation cases referred to by Mr. Sargent, the average duration of life was 5·3 years. Of those four cases, one lived eight years after the operation, but so few cases do not lend themselves to statistical consideration; indeed the seven I have quoted barely do so. Still, it seems that if the patient with this condition is left alone, it takes a long time for him to die; whereas, apparently, if treated by surgery, life may be shortened. And these people all write, as blind people always do, in a cheerful vein.

How we are to get nearer to cure without these severe operations and their high mortality I do not know. Cerebral surgery is still going through the stage that abdominal surgery did, and we have yet to learn our limitations; the road to that knowledge is beset with difficulties and disappointments. But if these pituitary cases are left severely alone, they do not seem to do so very badly, and if I were one of them I think I would prefer to accept blindness and enjoy what life was left to me.

But there is one symptom in these cases which does call for treatment. Mr. Sargent hinted that when a patient has got optic atrophy and the eye symptoms are well established, there is not much that operation can do for him in that way, and with that I agree. But a symptom which calls for surgical treatment is the headache, which is severe and frequently recurring. I recently had, in St. Bartholomew's Hospital, a young man with very terrible headaches; they would come on at any time, and last an hour or more. There was no vomiting, nor anything suggestive of it being due to cerebral trouble.

He also had marked polyuria. There was no alteration in his sugar tolerance. The sella turcica was somewhat enlarged. My surgical colleague, Mr. Rawling, was not inclined to do anything in the way of extirpation, but suggested decompression in order to relieve the headache. He did a bitemporal decompression, and the headaches have now ceased. I have lost sight of the patient now, but he was certainly very much happier for that operation.

Mr. V. Z. Cope.

I do not think I can add anything of interest to what I said four years ago, because I have during that time been abroad, and have had no opportunity of following the subject up. But since my return I have had a case which pointed out one of the mistakes in diagnosis which can be made. This case was handed over to me with the diagnosis that it was probably a pituitary tumour. Erosion of the sella turcica was clear from the radiogram, and the symptoms were very suggestive of pituitary derangement. It was such a large erosion that I did not feel justified in approaching it by the frontal route—which I usually advocate—and therefore I did a temporal decompression. There was, however, a lethal result. There was found to be a large inoperable tumour growing down from the lateral ventricle and pressing on the pituitary gland. If I had known during life what was the actual condition found I doubt if I would have operated: but the suggestion was that a temporal decompression would relieve the headache and the pressure symptoms.

My experience is too limited to enable me to give a generalization, but I would say the surgical outlook in these cases is not so hopeless as Dr. Tooth's remarks indicate, because much can be done by operation even to relieve the blindness. In one case I recorded the lad was practically blind: he could not see the clock on the wall. But after the decompression and the opening of the cyst in the sella turcica, he saw the time well. He was a dancer, and after the operation he was able to return to his occupation. In selected cases of this condition I think there is a hopeful opportunity for surgery if the technique, which has now been carefully laid down, is followed. For the general surgeon, the frontal route is my preference.

Mr. J. Herbert Fisher.

I have taken considerable interest in the pituitary body and the ophthalmic symptoms in connexion with it, and I have seen cases both before and after operation by both the routes which have been described this evening.

With regard to the possibility of recovery of vision, that is an established point, and it would be of very great importance to the branch of the profession in which I am interested if we could, by ophthalmoscopic observations, determine what optic nerve was capable of recovery, and what was not. But I am afraid we have not yet reached that stage. Even in ordinary cases of optic atrophy it is impossible to tell by ophthalmoscopic examination how far vision may have deteriorated, and that is certainly true of the cases of pituitary body disturbance of the visual pathways. There is known to be, however, a well-established resistance to disintegration of the axis cylinder processes in the optic nerves, and although there may be appearances of advanced atrophy and, corresponding with those appearances, a very marked diminution in the visual function, still these axis cylinder processes are not incapable of still transmitting the impulses to which the retina gives rise. I have had occasion to lament the unfortunate results of operative procedures in many of these pituitary cases, and by watching cases of pituitary body disease I can confirm what has been said as to the long duration of life in many cases. When operated upon by the transnasal route, they are certainly relieved of the more distressful symptoms of headache and pressure, and are able to lead comfortable lives after recovering from the operation. How far those results will be permanent is another question altogether. Many have relapsed, and further operations by one or other route have been necessary.

I would emphasize—though possibly it is hardly right to do so at a meeting of the Surgical Section—the very great improvement which is capable of being obtained in the visual and general symptoms by means of organo-therapy. One of the earliest cases of the kind which came under my observation was that of a man who apparently had advanced optic atrophy in both eyes. He had been treated by the usual methods on the suspicion of a syphilitic origin of the trouble, but without benefit. One eye was completely blind, but he still preserved the nasal half of the field in the other: his direct vision was reduced to about $\frac{6}{36}$. Under the administration of thyroid—at that date very few cases had been given pituitary extract—the field in which he had sight recovered to the full, and vision was $\frac{6}{9}$ and even partially $\frac{6}{6}$. He resumed his work as a labourer on the Medway, and was capable of again wheeling a barrow along planks. I kept him under observation, and continued the thyroid administration over a number of years. Ultimately his vision began to deteriorate by contraction of the temporal field, and later I lost sight of him. He had the general symptoms, such as impotence, of a case of hypopituitarism.

I have had more recently under treatment a case which was sent to me from South Wales, of a man blind in one eye, and vision much impaired in the other: he was also suffering severely from headache. The skiagram showed a pituitary body tumour. He was treated with thyroid and pituitary extracts, and he lost his headache, and the visual field, which in the temporal part had been almost obliterated, expanded again satisfactorily while direct vision distinctly improved. He has now returned to his home, and is probably again working in the mines.

The cases of cyst of the pituitary are those which the surgeons are anxious to encounter, but I think only few are pure cysts, though here and there will occur a case with cystic formations in connexion with adenomatous growths. The relief of pressure produced by evacuation of the fluid will obviously be a very desirable result for the surgeon to aim at. But even in these cases, I take it, there will be a solid growth, which will go on progressing. This would, I think, be established by the results of certain cases one knows of, in which there has been spontaneous rupture of the fluid through the sphenoidal air-cell and into the nose.

I do not think I can add anything very useful from the surgical aspect, and if I were to digress in an ophthalmic direction, my remarks might not be very useful to this meeting.

Dr. F. B. R. Atkinson.

I wish to speak purely from the medical side, chiefly in regard to the pituitary gland in relation to acromegaly. I suppose the majority agree that acromegaly is due to a tumour of that gland: usually an adenoma of the anterior lobe. I have examined the records of about 400 cases of acromegaly, and find that very few have been operated upon, and that patients without operation have lived for a long time: one case to 72 years of age. That seems to show that surgical intervention is not needed in these cases. I think Eiselsberg should have the credit for priority in operating on them. The cases have not done very well afterwards; most of the cases died shortly after the operation, and it improved very few, except as regards the headache and vision: the enlargement of the extremities was not affected. Organo-therapy has been frequently tried for acromegaly, but in most cases unsuccessfully; neither, I think, do most people expect benefit from this line of treatment. In acromegaly, vision is very frequently affected, and diminution of the visual fields quite common. Yet later, for some reason, the eyesight may improve without any treatment at all. This has been reported over and over again.

Mr. W. G. HOWARTH.

I would like to make a few remarks on this subject from the rhinologist's point of view. I have had the opportunity of operating on six pituitary cases, and, naturally, have always adopted the trans-sphenoidal route. I think there is something to be said for this method of approach, as it is a very simple one, and gives a little wider view than Mr. Sargent would have us believe. I do not myself adopt Cushing's incision into the mouth, because I think the purely intranasal operation is less likely to lead to septic infection, though one cannot deduce any general conclusions from such a small number of cases as six. Still, I have had no septic infections so far.

Another advantage of the trans-sphenoidal route seems to be that if one is dealing with a malignant tumour, it makes it possible to insert radium afterwards. I have done that in two cases: in one very successfully, i.e., the patient was much improved both in vision and as regards the headache after the use of the radium. Of course, this route really only admits of a decompression operation, and I do not think it is possible to attempt to extirpate the tumour. If cases could be seen earlier, I think the trans-sphenoidal route would have something definitely in its favour, certainly as a preliminary to the more serious procedure. In my cases I do not think I have improved the vision at all, except temporarily, because all the cases have been very advanced when I have first seen them, and the tumour has probably become extrasellar before one has had the opportunity of operating. [The PRESIDENT: It would add to the interest if you could tell us the nature of the tumour to which you applied radium with improvement.] The first tumour was reported to be a perithelioma, which is, I believe, a form of endothelioma, and the improvement was in the vision and in the cessation of the headache.

Mr. E. D. D. Davis.

I would like to give my experience as a rhinologist. I much regret I cannot speak of the operation by the nasal route as favourably as Mr. Howarth has done. In the two cases upon which I operated for headache and in others which I have seen, there was only transitory and temporary improvement. One of my cases has been mentioned by Mr. Sargent. As a decompression operation the nasal operation does not appear to me to be satisfactory. The hole in the sphenoid cannot be made much larger than a sixpenny piece, otherwise the cavernous sinus would be injured, and the hole becomes plugged by the tumour. These tumours and cysts, as Mr. Sargent has already shown, tend to grow forward between the optic nerves into the anterior fossa of the skull and become intracranial, where they cannot be reached by the nasal route.

The President.

I cannot myself speak of the surgery of this gland from personal experience, but I have always been deeply interested in its pathology, and therefore the facts which have been given in this discussion have been of great interest to me.

I agree with much of what has been said concerning these operations, but all the big operations of surgery have passed through a groping stage, and it is a satisfaction to many of us that operations on the pituitary body are in the hands of a few competent and dexterous surgeons—men who will not be afraid to publish their results, whether they be good or bad. We shall continue to watch these cases with very great care and interest. When Dr. Howard Tooth was speaking as to the rarity of these tumours, I reflected what a lucky thing it is that enlarged hypophyses are not as common as fatty tumours.

I am sure we are deeply grateful to Mr. Percy Sargent for bringing this subject before us in such a clear, incisive and practical way.

Mr. Percy Sargent (in reply).

I think the President has hit the nail on the head in pointing out that all surgical operations which have been put upon a firm footing have gone through a groping stage. That is the stage at which we now are in regard to pituitary surgery. If the question of vision, or even of the glandular symptoms, were the only questions which had to be considered, then the case of the person who survived with pituitary tumour so long as Dr. Tooth has indicated that they do might not be so very unenviable. What Dr. Tooth said might possibly have suggested that the condition of such a patient was no worse than that of a person blind from any other reason. But, at least in some of the cases, the headaches which occur are of an extraordinarily distressing nature, and many a patient with this form of headache would much rather be dead, or at least would prefer to accept such risks as are inherent in the operation.

With regard to vision, if the ophthalmic surgeons could tell us, from the ophthalmoscopic appearances, what the prospects of recovery of vision would be in any given case if decompression were done, we should have valuable ground to go upon, especially in regard to undertaking a procedure of such severity as intracranial operation. The nasal operation is not of great magnitude: the mere opening into the sphenoidal sinus should present little difficulty. Where the sella turcica is very much enlarged it is easy to incise the dural envelope, and scoop out fragments of growth. But when the sella is very small, it is questionable whether the lesion ought to be approached by this route at all.

What Mr. Cope said about the stage of development of the tumour is, of course, one of the important things. We are now asked to operate on cases at a very late stage, and I think that whatever success we may have in future will largely depend upon getting them at a much earlier stage.

With regard to acromegaly, to which Dr. Atkinson referred, if we regard acromegalic and gigantic symptoms as due to excessive activity of the gland, it is futile to expect improvement by adding more gland substance to the patient's metabolism. The cases of pituitary lesions which have improved at all under treatment by gland extract have been those of hypopituitarism.

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Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. J. P. Lockhart-Mummery, F.R.C.S.

DISCUSSION ON DIVERTICULITIS.[1]

Dr. W. H. Maxwell Telling (Leeds).

I am very sensible of the honour you have done me in asking me to open a discussion on diverticulitis, a subject in which the Sub-section of Proctology has naturally a very special interest, but of which a full practical knowledge should be possessed by every abdominal, gynæcological and genito-urinary surgeon, as well as by physicians and morbid anatomists. In the short space of time which introductory remarks should occupy I will concern myself with the broad lines of the subject and mainly in its clinical aspects.

In 1917, when last reviewing the subject, I wrote[2] : "Diverticulitis is a condition which has now passed out of the realm of doubt and uncertainty into that of proved and accepted fact. It has an important place in medical literature, and in the experience of every operating surgeon of large practice. This last statement is not as yet true of all clinicians, some of whom are still apparently unaware of the condition, and not a few of its frequency and clinical importance. Not until a morbid condition is described in all the ordinary student's text-books[3] of medicine and surgery can it be said to have attained complete recognition, and this is not yet the case with diverticulitis." This is practically the situation to-day.

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[1] At a meeting of the Section, held January 7, 1920.

[2] *Brit. Journ. Surg.*, 1917, iv, pp. 468-530. To this article reference may be made for a detailed study with analysis of cases.

[3] A small paragraph first appeared in the 1910 edition of "Osler's Medicine."

Diverticulitis means any inflammatory change in any diverticulum or series of diverticula. In practice, however, usage has restricted the term to the inflammatory changes and secondary pathologic processes generally occurring in or in connexion with a *certain type of diverticulum.* This type is the secondary, acquired, multiple, false diverticulum of the large bowel, particularly and nearly always found in the sigmoid flexure. The diverticula occur mainly in the lower part of this structure, in small or great numbers, but may be found elsewhere, as in the descending colon, the other flexures, the cæcum, the rectum, or throughout the entire large bowel. They may be limited to these special positions, though when occurring therein are more usually associated with sigmoid lesions as well; if limited to them they may give rise to puzzling and anomalous symptoms. The cæcal cases, especially, are of considerable interest, and merit much more attention and investigation than they have already received. Occasionally there is a widespread tendency to diverticulum-formation throughout the whole intestine. For convenience, however, I shall confine my remarks to the typical lesion as it occurs in the sigmoid flexure.

Historically, diverticulitis may fairly be said to be a new subject. Odd specimens had been described from time to time during the last century, but such descriptions were "buried" and isolated, and the clinical aspects of the lesion were for all practical purposes unknown.

In 1898 Graser[1] described examples associated with fibrous hyperplasia, causing stenosing tumour formation—*peri*-diverticulitis: similar cases were then described in America, and Patel,[2] in France, drew particular attention to the more acute inflammatory lesions—sigmoiditis and peritonitis. I observed my first case in 1899, and finding nothing about it in the literature, or even anyone who was familiar with the condition, I thence onwards made it a practice to examine almost every sigmoid flexure personally at the autopsies I made. The result was the accumulation of a considerable amount of material. In 1907 Moynihan[3] published the first case in England of the Graser-tumour type, and this induced me to analyse and classify my cases. This I did in 1908[1] and this was the first time the subject had been treated as a whole and the various pathological findings and clinical symptoms correlated, or that any complete arrangement and classification had been attempted. That classification has been, in essentials, accepted by almost all subsequent writers and observers, and writing now, twelve years later, when a very considerable number of cases has been observed and recorded, I have no important modification to offer.

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[1] *Centralbl. f. Chir.*, 1898, xxv (*Bericht. ü. d. Verhandl. Deutsch. Gesellsch. f. Chir.*), p. 140.

[2] *Lyon Chir.*, 1911, v, p. 121.

[3] *Edin. Med. Journ.*, 1907, xxi, p. 228.

[1] *Lancet*, 1908, i, p. 843.

The reason that this early "stabilization" of the subject was possible is this: Given the occurrence of a tendency to the formation of multiple hernial out-pushings of the mucosa in the sigmoid flexure, every secondary process that does in fact occur may be logically deduced *a priori* by general pathological comparison and study. I am not proposing to deal with the vexed question of *why* these diverticula occur in the first place except that there is no doubt that they are largely, if not entirely, due to increased pressure within the bowel. Constipation, therefore, plays an important part, but I feel convinced that flatulent distension, often co-existing, of course, has a rôle that cannot be overlooked and needs more study.

A knowledge of the anatomy of these diverticula is of the first importance in understanding the secondary pathologic processes and also in detecting them at all, either in the deadhouse or at operation. At this point the inquiry is pertinent as to how it comes about that the condition has for so long escaped practical recognition, having regard to the large number of cases recorded—not a tithe of those observed—since 1898. The answer is, I think, to be found in three facts. Firstly, the majority of demonstrators of morbid anatomy fail to devote special and personal attention to the sigmoid flexure and perhaps to those surgical autopsies in which, from their point of view, the surgeon has "spoiled" the abdomen generally and a specimen in particular by his manipulations: particularly when the structures operated on have been more or less inflamed, adherent and "mixed-up." Secondly, the diverticula themselves are "adepts" at concealment. to this I will return later. Thirdly, the secondary pathologic processes are liable to be so extensive and disproportionate to the comparatively insignificant diverticula as to hide the true nature of the case from any but patient and skilled observers. This must be the general experience of all who have studied a series of specimens *in situ*.

The second of these points, the "self-concealment," turns upon the anatomy of the diverticula. Firstly, they are small, varying from minuteness up to the size of the last joint of the little finger; more commonly they are one-third to one-fifth this size. Their apertures are often minute, from $\frac{1}{16}$ in. to $\frac{1}{3}$ in. in diameter, and these apertures usually occur in two rows, mostly opposite the appendices epiploicæ, and often completely concealed by the rugosa mucous membrane. On the outside of the gut the diverticula are seldom observed except by the trained eye, because (1) they mainly enter the appendices epiploicæ as stated, and (2) the gut in which they occur is very frequently fat-laden, necessitating careful dissection to discover them at all. The occurrence of adhesion to other structures and of peridiverticular hyperplasia tends still further to conceal and almost to obliterate their presence. Even so, the fact that isolated observations on them for over a century should have failed to lead to complete recognition is a little remarkable but none the less a fact.

I am stressing these anatomical points rather carefully, as they must be fully appreciated by every operating surgeon if he hopes to recognize the condition at the time of the operation.

Once the diverticula are "in being" the whole series of possible consequences depends upon the following three facts: (1) being formed by pressure from within they tend to enlarge; (2) they tend to harbour fæcal or other harmful contents; and (3) as a consequence they tend to undergo various secondary pathologic processes. In their enlargement they become flask-shaped, with a bottle-neck; this bottle-neck may greatly elongate, its lumen remaining very narrow. This elongation is brought about by *peridiverticular hyperplasia*, which often pushes the "flask" to its circumference, where it sits on the surface of the tumour and works special mischief of other kinds, such as adhesion, perforation and fistulous communication.

The narrow mouths tend to promote the *retention of fæcal contents*, soft or forming actual concretions. Here analogy with appendicitis will largely help us to prophesy results, except in regard to the remarkable hyperplastic peridiverticulitis. The fæcal contents form a bacterial nidus, a reservoir of toxins, and a source of mechanical irritation. As a result secondary changes supervene; if not, the condition remains a harmless pathological curiosity and gives rise to no symptoms or trouble. Perhaps the large majority of instances are largely or entirely innocuous.

The main secondary processes which do occur, separately or variously combined, are:—

(1) Ulceration of the mucosa of the diverticula.

(2) Perforation as a consequence.

(3) Adhesion to other structures, which, when combined with (2), leads to fistulous communication.

(4) Peridiverticular fibrous hyperplasia leading to tumour formation and, as a sequential result, stenosis of the bowel.

Confining our attention to these main and usual secondary processes, which, as has been stated, may be variously combined, a few moments' consideration will show how protean and puzzling the resulting clinical manifestations may be. In the first place mucosal ulceration will weaken the wall of the sac. This will facilitate perforation, and a concretion will be specially liable to be driven through at any time of increased pressure or violent effort, causing a local or general peritonitis. In quite a number of cases this has actually happened.

The mucosal ulceration also sets up inflammation on the outer surface of the diverticulum, leading to adhesions. These *may* produce no important results, but *may* produce vesical or pelvic syndromes or an acute intestinal obstruction by band or kink. Adhesions *plus* perforation lead either to local abscess, with its varied symptoms, signs and sequential risks and possibilities, or to fistulous communication with other viscera, of which that with the bladder is the commonest, most interesting, and surgically the most important instance.

The culminating interest is reached, however, in the production of the fourth type of lesion, the *peridiverticular hyperplasia.* This may exist with mucosal ulcerations of the diverticulum but not infrequently occurs without it, and is a common and characteristic result, the one which first led to a systematic investigation. It is undoubtedly due to a chronic leakage of toxins or bacteria, or both, through the sac walls, producing a hyperplastic fibrous-tissue formation somewhat similar to that found in a hyperplastic tuberculosis of the cæcum, or more closely resembling a scirrhous cancer. Because of the hyperplasia, which may amount to an inch or more in thickness on section, a tumour is produced; because it is fibrous the tumour contracts and stenoses the bowel, tending towards chronic intestinal obstruction. This thickening and stenosis may be so local as to make an annular stricture and it will be realized how close is then the mimicry of carcinoma. In this way doubtless many of the cases of so-called "cured" cancer in this region may be explained, as well as the placing on museum shelves examples of peridiverticulitis labelled cancer. Others besides myself have found them.

There is, however, a very special relationship of the condition to true carcinoma. A simple peridiverticular stenosis (or possibly other diverticular lesion) may develop carcinoma secondarily, as in the case of a chronic gastric ulcer. A small number of cases have now been put on record, and since my last records were published in 1917 I have had a further case. The patient was riding and felt a sudden pain in the left iliac fossa followed by temperature and signs of inflammation. I made a diagnosis of perforative diverticulitis and Sir Berkeley Moynihan operated and confirmed, finding multiple diverticula—one of which had perforated and set up general peritonitis. There was also some thickening and stenosis but no naked-eye evidence of malignancy. A year later a second surgical inspection was made and malignant disease was then obvious and inoperable.

It is not too much to say, therefore, that no surgeon ought to decide about or operate on a supposed carcinoma of the sigmoid flexure without an accurate knowledge of what may be termed "diverticular possibilities," nor ought any such tumour removed by operation to lack a complete and *skilled* examination for the presence of diverticula. It was only by this means that Moynihan detected and recorded the first of such cases in the English literature.

We can usefully divide the secondary processes into two main groups: (*a*) Mechanical (mainly), and (*b*) inflammatory (mainly). Both processes may go on in the same case, just as there may be present together varying types of inflammatory reaction.

(*a*) *Mechanical.*—(1) Formation of fæcal concretions in the diverticula. (2) Torsion of the diverticulum. (3) Lodgment of foreign bodies within the diverticular sacs. (4) Perforation.

(*b*) *Inflammatory.*—(1) Diverticulitis: (i) gangrenous; (ii) acute; (iii) sub-acute; (iv) chronic; (v) latent. (2) Passage of organisms without perforation. (3) Peridiverticulitis—chronic proliferative inflammation with tendency to stenosis of the bowel. (4) Perforation of the diverticula, giving rise in particular to: (i) general peritonitis; (ii) local abscess; (iii) fistula, especially into the bladder; (iv) suppuration in a hernial sac. (5) The formation of adhesions, especially to: (i) the small intestine; (ii) the bladder; (iii) the female genitalia. (6) Chronic peritonitis, local. (7) Chronic mesenteritis of the sigmoid loop. (8) Metastatic suppuration. (9) The secondary development of carcinoma.

By the aid of this pathological series it is not difficult to forecast the main clinical symptoms and also to surmise that they will be liable to be variable, complex and puzzling. This is so in fact, as a study of case records abundantly proves. At the same time the majority of cases conform more or less to the following types: (1) Inflammatory trouble or tumour in the left lower quadrant of the abdomen; (2) general peritonitis; (3) vesicocolic fistula; (4) pelvic syndromes; (5) intestinal obstruction; (6) mimicry of carcinoma.

The frequency of tumour or abscess formation in association with any or all of these is considerable (about 30 per cent.) and one may say that in any case from the age of 30 onwards, presenting these symptoms, calls for mental reservation as to the possibility of a diverticular origin, and in some instances the diagnosis can be and has been made with certainty and accuracy.

As general clinical points it must be remembered that cases occur mostly at or after middle age (though a few cases in early life have been noted), in males more frequently than females; usually with a history of constipation and very seldom causing blood in the stools. A few comments are called for on some of the foregoing clinical types of case.

Inflammatory Trouble, more or less acute, in the Left Lower Quadrant of the Abdomen.—Cases of this type constitute by far the largest group. Erdmann has gone so far as to say that the occurrence of a left-sided pain, with mass or not, but with tenderness and rigidity, is practically pathognomonic of diverticulitis. This is the extreme view, favoured by the supporters of the almost exclusive rôle of diverticula in inflammatory troubles in this quarter. Fitz, in 1912, said that the cases comprised in this "inflammatory" group made a fairly complete picture of what must be recognized as a new disease of the lower abdomen—diverticulitis of the left quadrant, analogous to appendicitis of the right quadrant. Bladder and pelvic symptoms are not uncommon. Various sub-types of the inflammatory group may be distinguished and a useful clinical classification is as follows: (i) Acute and fulminant (gangrenous); symptoms practically identical with acute right-sided appendicitis. (ii) Recurrent; acute or sub-acute attacks over a long period. (iii) Cases ending in spontaneous recovery. (iv) Protracted cases—those which continue till the clinical picture is changed by one or other of the following developments: (*a*) simulation of carcinoma; (*b*) intestinal obstruction (acute or chronic); (*c*) bladder perforation; (*d*) general peritonitis; (*e*) incidence of pelvic symptoms.

Trauma.—The mode of onset of many of the acute cases is of interest as showing the great part which trauma of various kinds may play. It has a definite diagnostic value when diverticulitis is under suspicion and acts as an exciting cause of, at any rate, the acute manifestations. These have occurred while straining at stool, during the administration of an enema, following a dose of castor oil, jolting in a motor car, following a heavy meal, while lifting a weight, jumping or hunting, and during an abdominal operation. In this group pain of one kind or another (often recurrent and colicky) and tenderness are very frequent. Tumour-formation has been noted in 30 per cent. of the cases, and the tumour is not infrequently and then characteristically variable. Abscess-formation is very frequent, consequently fever and leucocytosis are often present. Bladder and pelvic symptoms are not uncommon. Apart from actual fistulous communication with the bladder, which constitutes a separate clinical group, there are frequently symptoms of bladder irritability, probably due to the nearness of inflammatory foci or to actual adhesion. Pelvic symptoms are of some frequency, and sometimes make the clinical picture.

Vesico-colic Fistula and Bladder Symptoms generally.—In a recent review[1] Bryan has stated that "diverticulitis" is the most frequent cause of sigmo-vesical fistula. Moynihan was the first observer in England to emphasize this fact. He wrote in 1907: "The formation of a vesico-intestinal fistula seems to be one of the tendencies of a perforated false diverticulum; a search through the literature has shown us that it is far more common than was supposed. In cases where a hard growth in the intestine is accompanied by the passage of flatus and fæces by the urethra, a diagnosis of carcinoma seems irresistible, yet the *probability* is that the growth would be simple, and that the cause of the fistula would be a false diverticulum which had burrowed its way through all the coats of the bowels, and thence through the wall of the bladder which had become adherent." Experience has abundantly confirmed the truth of this statement, and its recognition has been followed by some brilliantly successful cases, cured by operation after long periods of suffering.

Pelvic Syndromes.—Much has been written in recent years about the close pathologic relationship between the sigmoid flexure and the other pelvic viscera. Much of it has been without any or a very extensive knowledge of the rôle of the diverticulum. It is not surprising to find diverticulitis increasingly frequent in the practice of gynæcological surgeons. Pelvic symptoms of one kind or another existed in 7 per cent. of my collected cases, and a number of examples have been placed on record in which the symptoms were entirely pelvic, bowel symptoms being absent. There is no doubt that Shoemaker[2] strikes the right note in saying that, in any suspected case of pelvic or utero-ovarian disease, a routine and thorough examination of the recto-sigmoidal bowel and its functions is just as important as a vaginal and uterine examination.

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[1] *Ann. Surg.*, 1916, lxiii, p. 353.

[2] *Ann. Surg.*, 1914, lix, p. 422.

Diagnosis.—This can only be made after a careful consideration of all the facts of the case. Inflammatory lesion in the left lower quadrant in a person of middle or advanced age carries with it a strong *prima facie* possibility of a diverticulitis being the cause, but, beyond this, exact diagnosis is often difficult, and all special aids to diagnosis may have to be requisitioned. These include: (1) The sigmoidoscope, which has been disappointing in the main in this condition, though perhaps some members may have had more fortunate experience; (2) the cystoscope, which has been of particular value in a small number of cases; and (3) examination by X-rays. This last procedure holds out most promise and has been greatly developed during the last few years. As may be expected, the technique is not of the easiest, and there are many fallacies in the appearances noted, but there is no doubt of the general diagnostic value. The outstanding condition calling for differential diagnosis is carcinoma. It may be and often is impossible, not only before operation but at operation. The following summary of the chief points differentiating diverticulitis from carcinoma may be of use:—

(1) The absence of the "shadows of malignancy" from the general picture.

(2) Tendency to obesity, and maintenance of good nutrition generally.

(3) Long history of attacks of abdominal pain in the left lower quadrant.

(4) History of tumour formation with subsequent disappearance.

(5) Absence of blood (visible to naked eye) in stools, over a prolonged period.

(6) Presence of a vesical fistula, in which malignancy can be excluded by cystoscopy.

(7) Negative sigmoidoscopy as regards malignant disease.

(8) X-ray demonstration of diverticula.

(9) Pyrexial attacks.

(10) Examination of blood: the presence of neutrophilic leucocytosis and the absence of the specific nuclear changes characteristic of cancer.

In carcinoma of the sigmoid, loss of flesh is early, pain and tenderness are late, and are often preceded by tumour. There are certain other conditions which have to be considered in arriving at a differential diagnosis. They are: (*a*) sigmoiditis, (*b*) hyperplastic tuberculosis, (*c*) actinomycosis, (*d*) syphilis, (*e*) pelvic conditions generally.

Treatment.—This is comprised in a single word—surgery—unless operative interference is specially contra-indicated. As a physician I shall not venture to discuss particular surgical procedure, but confine myself to a few general points. In view of the potentiality for mischief of diverticula, all diverticulum-bearing gut should be removed. At operation search should be made whenever possible for further diverticula. Recurrence after operation has occurred, presumably from neglect of this point. There seems to be a special liability to post-operative peritonitis, possibly due to the chronic infection of the inflamed tissues in some cases. Care should be taken in handling the gut, as at least one instance has occurred of rupture of a diverticulum by surgical traumatism. The good results already obtained in certain bladder fistulæ should stimulate surgeons to deal with such cases.

Lastly, no case of supposed carcinoma of the lower bowel must in future be regarded as inoperable, either before or at laparotomy, unless diverticulitis has been remembered, fully considered, and systematically investigated.

Sir JOHN BLAND-SUTTON.

Thirty years ago colic sacculi were mainly regarded as pathological curiosities, and our knowledge of their potentialities for evil is a direct result of surgical enterprise. My interest in them was aroused when trying to find an explanation of the phenomenon known as "spontaneous disappearance of abdominal tumours." This matter was discussed in the Royal Medical and Chirurgical Society in 1894 by Mr. Greig Smith,[1] and increasing experience in abdominal surgery enabled me to learn the part played by colic sacculi in producing localized swellings in the belly, which sometimes disappear by expectant treatment. The phrase "spontaneous disappearance of abdominal tumours" disappeared from surgical phraseology when surgeons learned that acute and chronic diverticulitis, or sacculitis (names as ugly and cacophanous as appendicitis) mimic many important lesions, especially acute and chronic appendicitis, cholecystitis, tumours of the stomach, subdiaphragmatic abscess, perigastric abscess, splenic abscess, tubal infections, ovarian abscess, cancer of the uterus, and infected uterine fibroids.

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[1] *Med. Chir. Trans.*, 1894, lxxvii, p. 139.

The extraordinary feature of sacculitis (or pericolitis) is its mimicry of cancer of the colon. In 1901 I excised the sigmoid flexure of a spinster, aged 60, under the impression that it was cancerous. After the operation critical examination of the tumour proved that the supposed cancerous mass was a pericolic mass due to infection of a sacculus caused by a piece of straw. The patient recovered. This, with a case of epiploic abscess, was described before the Medico-Chirurgical Society of Nottingham in 1903, and full details are published in the *Lancet*, 1903, ii, p. 1148. Since that date I have seen and operated on many similar cases, and it is clear that in the past patients have submitted to colotomy and colostomy under the belief that they were suffering from colic cancer; subsequently the lump disappeared and the patients survived the operation many years, and at death there has been no evidence of tumour. This admits of no doubt. Also cases are not uncommon in which a swelling is detected in connexion with the large gut; a diagnosis of cancer is made and the patient is advised to submit to an operation, but he obstinately refuses, and in due course the swelling disappears in spite of the gloomiest prognostications of physicians and surgeons of great experience and repute, and the patient lives to mock at his advisers. The disappearance of the mass is due to pus or the exudate finding a channel of escape.

During the last ten years acute diverticulitis, especially in the lower abdomen, is recognized with the same certainty as acute and chronic appendicitis. The disquieting feature of these new things in abdominal surgery is the fact that as we grow older colic sacculi tend to increase in number, and as many as 200 have been counted on a colon. Thus diverticulitis is a newly discovered "bane of elders."

As prevention is better than cure, it will be the duty of the family medical attendant to encourage men and women after middle life to have efficient sets of teeth, and warn them against gobbling indigestible food or bolting hot unpalatable morsels.

Dr. E. I. SPRIGGS (Banff).

The knowledge I have on this subject of diverticulitis—and this probably applies to many of us—I have derived from Dr. Telling's papers, and most of it from the valuable monograph which Dr. Telling and Dr. Gruner published two years ago. We have learned that diverticula occurring in the colon are small and multiple, that they are commonest in the sigmoid flexure, that they generally occur in people over 40 years of age, and that the majority of them occur in men. Since 1917, an American writer, D. Roberts, has published a series of twenty-four cases, and had made the diagnosis with X-rays in twenty-two. He expressed the opinion that these diverticula of the colon are exceedingly common. Another American writer, J. W. Keefe, has also used X-rays in detecting diverticula.

Our experience at Duff House is as follows: We have X-ray photographs of between twelve and thirteen hundred alimentary canals, taken consecutively. I cross out the first two to three hundred, because we had not learned then the importance of taking frequent photographs to observe detail in any part of the alimentary canal. In the last 1,000 cases we have seen diverticula of the colon six times. I do not mean there have been diverticula of the colon only six times in that number of cases, because there are cases in which attention is not directed to the colon, and in which, at the various screenings, the sigmoid is not well seen. But certainly in the last few hundred cases steps have been taken to get a good view of the sigmoid by repeating the opaque meal if necessary or giving a barium enema. So I do not think diverticula, as observed by X-rays, are very common. Of course some may not be seen because they do not happen to be in profile, or because the diverticula are filled with fæces, which not having been emptied out by the preliminary purging, prevent opaque substance from entering. But they are not so numerous as some believe, or more would get on to the plate in the examination of a large series. If the diverticula retain fæcal material, it is easier still to recognize them, provided the barium enters some of them, because after the rest of the bowel is emptied, the diverticular contents remain and give the characteristic picture of a chain of opaque bodies.

I will now show, by means of the epidiascope, photographs taken by Mr. Marxer from these cases. I shall not weary you with clinical histories, but will give a summary of them when I have shown the slides.

All the patients were males, and their ages ranged from 53 to 71. One patient had a single diverticulum only, in the middle of the transverse colon. Five had multiple diverticula. Of these five, two also had diverticula in the terminal ileum, and one of them had a diverticulum in the third part of the duodenum, a large single one.

It is believed that diverticula of the colon are due, as Dr. Telling has said, to extrusions at weak places, and I think that the pictures support that view, in so far as the diverticula are seen to occur where muscular contraction may be expected to be specially vigorous and pressure inside the lumen of the bowel to be specially high, namely, towards the end of the small gut and towards the end of the large gut.

With regard to the rôle of constipation, one of these patients had had severe constipation for many years. Four had had only moderate or intermittent constipation, and one had not had any. Constipation from the X-ray point of view—delay in the rate of passage of the opaque meal through the intestine—was present in two out of the six patients—i.e., the material did not pass through in seventy-two hours. One of the patients showed hypertonus of the colon, and one of pelvic loop only.

This is a very small series, but I show it because, first, I believe that diverticula, with adequate examination, can be easily recognized by X-rays. Secondly, because it is an early series. In none of the cases was there any symptom which could be clearly attributed to the diverticula, though I must qualify that by mentioning particularly two patients. In one we thought there was probably an adhesion of part of the sigmoid: this is the man who showed a single small diverticulum in the middle of the colon. He had a number of other symptoms, and was suffering from the neurasthenia, which is observed sometimes after severe venereal infection. So there were other causes to account for his symptoms, and there was nothing which, from Dr. Telling's description, could be called diverticulitis. Another case was that of an elderly man with arteriosclerosis, who had vague abdominal symptoms. We could see that his appendix was retrocæcal. He had had two or three attacks of what was regarded as appendicitis, as I believe they were. He was not, on other grounds, suitable for operation, and is now well. Of the other four cases, one is under treatment. He has symptoms typical of duodenal ulcer, and there is a deformity of the first part of the duodenum; but there are no bowel symptoms. The other three suffered from achylia, or poverty of gastric juice, chronic appendicitis, confirmed by operation, and duodenal ulcer. All are well, and are doing their work. We are, of course, watching them, but, so far, no complications have developed.

Mr. McAdam Eccles.

It is probable that "diverticulitis" is more common than is usually thought. The two forms are acute and chronic. The large intestine is the most frequent portion of the bowel to be involved. Acquired diverticula are generally multiple. It is by no means rare to find the diverticula running out into masses of fat, sometimes into actual appendices epiploicæ. The acute variety when occurring in the pelvic colon may cause suppuration around the loop, and give rise to a condition termed erroneously "left-sided appendicitis." I have on several occasions opened abscesses in the left iliac fossa, which I have little doubt were due to perforation of these diverticula. The chronic form in this region is of great interest because of its similarity to carcinoma in the same part. The lesion occurs more frequently in men, and in men after middle life. It gives rise to troublesome constipation, with much flatulency, and considerable distress from frequent desire to defæcate. A tumour forms, and then with some emaciation, nearly every sign and symptom of malignant disease, except hæmorrhage, is present. Even when the abdomen is opened it is difficult in many cases to be sure whether a simple or a malignant condition is present. The specimen shown is a typical one which I removed from a gentleman aged 74, in November, 1913. I had a letter from him two days ago to say he was now 80 years, and that he was in "rude health and very active," and had no return whatever of any of his troublesome symptoms. It will be seen that the lumen of the pelvic colon has been very greatly narrowed, that there are numerous diverticula. In some instances a fairly certain diagnosis can be made by the appearance presented in a skiagram taken after the rectal injection of barium under a vertical pressure of 3 ft. Several of the diverticula will be seen radiating from the lumen of the gut. Probably excision of the narrowed portion of the bowel is the most appropriate treatment.

Mr. G. Grey Turner (Newcastle-on-Tyne).

My interest in this subject was first aroused in the year 1903, when, as Surgical Registrar, I had an opportunity of seeing Professor Morison remove what was supposed to be a malignant growth of the sigmoid flexure in a man aged 60. On examining the specimen I found it did not present the ordinary features of malignant disease, and careful scrutiny showed that it was an example of diverticulitis. The patient died, and at a later stage we found the diverticula extended from the middle of the transverse colon to the point at which the sigmoid had been divided, but none were present in the bowel below. It is rather curious that we should have overlooked the importance of these conditions at that time, because a paper by Schreiber was epitomized in the *Medical Review* of about that period.[1] Since then I have had the opportunity of seeing many cases which have illustrated practically all the clinical features mentioned by Dr. Maxwell Telling in his very complete opening paper.

I will speak of three particular instances. One was that of a lady, aged 34, upon whom I operated in 1904, under the impression that she was suffering from appendicitis. There was a typical attack, followed by the development of a lump in the right iliac fossa. At the operation, however, the appendix was found to be normal, and, on further examination, a hard mass was found in the cæcum, with great œdema of the retro-peritoneal tissue and a small diverticulum at the caput. The cæcum and ascending colon were excised and an anastomosis made. Subsequent investigation of the specimen showed that the mass was entirely inflammatory around a diverticulum, and that the one at the apex of the cæcum showed no pathological change. That patient recovered, and is at present alive and well and without any symptoms suggesting the presence of further diverticula; none were noticed when the anastomosis was made.

The second case I want to mention is probably one of the curiosities of pathology. It was that of a man aged 60, who was admitted to the hospital in 1902 because of retention of urine. He said the condition had come on gradually, and that as it had been found impossible to relieve him by catheters an attempt had been made to empty the bladder by suprapubic tapping. Afterwards the patient became very ill, and was admitted with obvious peritonitis, from which he soon died. I made a post-mortem examination, and found extensive septic peritonitis, with fæces all over the abdominal cavity. Low in the sigmoid there was a mass which I took to be cancer, and above that a large hole in the wall of the bowel. Further examination showed a large gall-stone free in the pouch of Douglas, and this gall-stone just passed the hole in the colon, and had evidently been extruded from the intestine above the supposed growth. There was a communication between the gall-bladder and the duodenum and the hepatic flexure, and a calculus projected from the viscus through the hole in the large bowel. The specimen was put aside for further investigation, but no use was made of it until 1908, when I was particularly interested in the subject of gall-stone ileus, and it was then that I discovered that the supposed growth was not malignant, but was a typical example of diverticulitis.

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[1] *Med. Rev.*, 1902, v, p. 737.

The third case was that of a man on whom I operated because of pelvic peritonitis of undetermined origin. It turned out to be due to the perforation of a diverticulum in the sigmoid. Unfortunately the patient died of pulmonary embolism. That afforded us an opportunity of making a complete examination, and, although a most careful search was made, we could only discover three diverticula in the affected bowel, and it was one of these which was ruptured. They were the smallest diverticula I have seen.

With regard to the ætiology of the condition, I very much regret that Professor Keith is not here, as I know he holds strong views on this matter, views opposite to those I have formed. I think this condition is essentially congenital in origin. By that I do not mean that the fully-developed disease is present at birth, but that the factors which allow it to develop are there, and are due to the anatomical arrangement of the parts. I do not know whether that is true, but, if it is, it would bring the explanation of this condition into line with the general surgical principles which we recognize in connexion with the other diverticula, which are so much more common in the small intestine. Even if present at birth, these conditions may only develop gradually. Recently I operated upon a lady aged 60 for what turned out to be a diverticulum of the duodenum, and even at that age it had not been completely distended, for there were large masses of mucous membrane thrown into rugæ at the apex.

Dr. Maxwell Telling mentioned the case of a man who had a diverticulum in the bladder associated with diverticulitis of the colon. I had a similar case in which I found two large so-called saccules in connexion with the left ureter, in a man who died from perforation of a diverticulum in the colon. I think extended experience has led one to believe that most of the saccules in the bladder are not dependent upon obstruction, but are of congenital origin.

With regard to diagnosis, I have been painfully struck with the wonderful stories which these patients tell of long-continued trouble when we go carefully into their histories, stimulated by the discoveries of the operation table! In one of my patients—I show the specimen here—the history is of ten years' duration.

In reference to the use of the sigmoidoscope, I think we owe much to that instrument simply on negative findings. We may think we have a case of malignant growth in the lower bowel, but there is nothing to be seen. While Dr. Spriggs was speaking it occurred to me that it might be a good thing in these cases not to be content with a bismuth meal, but to give a series of such meals at varying intervals, so that the bismuth or barium would have time to worm itself into the small diverticula.

So far as treatment is concerned, I would like to mention two cases. One was that of a lady, aged 55, who came under my observation nearly seven years ago. She presented symptoms which, we were led to believe, depended upon an inflammatory condition in connexion with an old ectopic gestation in the left side of the pelvis. On opening the abdomen I found this pelvic condition was really a mass of bowel, and, on separating it, I saw that there were diverticula above it. It was in such a position that it would have been difficult to excise it and make an anastomosis, and I did not care to cut it away and make a permanent colotomy, as one would have been glad to do if it had been malignant. Therefore I made a colotomy alone. The patient remained well eighteen months, and at the end of that time she developed intestinal obstruction, and I had to reopen the colotomy wound. Afterwards she took care of the lower segment of the bowel by washing it through with saline, and kept well for five years. At the end of that time she developed another inflammatory mass, terminating in an abscess, which burst at the lower end of the abdominal incision. She got over that, with the persistence of a small mucous fistula, and is now in fair condition.

When surgeons get to know this condition better, and talk about it as we are talking to-night, it will gradually filter down to become common knowledge among those who see people in earlier stages of disease, and it is certain that cases will come to us for advice and treatment. And then our difficulties as surgeons will, I think, commence. It is all very well to say surgery is the only remedy, but it is a formidable undertaking to excise a colon simply because it harbours multiple diverticula which have not caused serious symptoms! It has been my province to operate on two such cases. In one the man had suffered from symptoms which justified exploration. In that case it would have been dangerous to remove the whole bowel, and I had to be content with inverting several of the more prominent diverticula and stitching over the peritoneal pocket thus left. Other doubtful areas were covered with the neighbouring appendices epiploicæ; in that way more serious consequences would be staved off until something could be done. The patient was told of the necessity for exercising care with the bowel. That is nearly eighteen months ago, and the patient remains in good health. In another case the diverticula were too rigid, and I could not treat them in that way. The patient was an old lady, and they were covered with fat. Therefore I left them alone and closed the abdomen.

Mr. G. Grey Turner also showed the following specimens:—

(1) *Diverticulitis of the Sigmoid excised under the Impression that the Condition was Malignant.*—The operation was performed by Professor Rutherford Morison in August of 1903. The condition was recognized as being due to diverticulitis, and was correctly described in the notes, but the case was not published until 1912.

(2) *Diverticulitis of Ascending Colon.*—The cæcum, with part of the ascending colon and ileum, excised from a female, aged 34. The specimen shows two diverticula. One was inflamed, and during life formed a hard mass associated with retroperitoneal œdema. The condition was diagnosed as appendicitis. No other diverticula were noticed during the course of the operation, and the patient is alive and well fourteen years after the operation.

(3) *An Example of Diverticulitis of the Sigmoid which simulated Malignant Disease and in which Obstruction was precipitated by the Impaction of a Large Gall-stone.*—The latter ulcerated through the bowel, and was found free in the peritoneal cavity, death occurring from general peritonitis. The patient was a man, aged 57, seen in 1902. The true nature of the condition was not appreciated until the specimen was re-examined in 1908.

(4) *Serial Sections of a Specimen of Diverticulitis of the Sigmoid, excised under the Impression that it was malignant.*—The patient was a man, aged 69, who had suffered for nearly ten years from attacks of abdominal pain with tenderness in the lower abdomen. The bowels were usually lax, and he never passed blood or mucus. An impending attack was often warded off by a dose of physic. After an especially bad seizure a "lump" was discovered, and he began to lose weight. The operation disclosed a large hard mass adherent to the bladder and apparently undoubtedly malignant. It was excised and the bowel anastomosed. The patient recovered with a tiny fæcal fistula, and lived in good health for six years afterwards.

(5) *Carcinoma of the Sigmoid; Multiple Diverticula; Rupture of Diverticulum with Fatal Peritonitis.*—Specimen obtained from a man, aged 70, admitted to hospital with peritonitis, which proved fatal eighteen hours after a sudden onset.

Mr. RUTHERFORD MORISON (Newcastle-on-Tyne).

For centuries certain deformities of the blood-vessels and urinary bladder have been recognized and described as sacculi—sacculated aneurysms, sacculi of the veins, and sacculated bladders have been for all this time acknowledged conditions, so that they have the hall-mark of tradition. It is now known that all of the hollow viscera may be similarly affected, but confusion has arisen because these protrusions have been described, chiefly by Americans, as diverticula, and Dr. W. H. Maxwell Telling has followed their example. His excuse—I cannot think of it as a reason—is that sacculation of the colon has been described by the anatomists as its normal condition. This difficulty can be got over by regarding ordinary sacculation as normal, and acquired sacculation as pathological, and this is easier when it is remembered that that part of the colon, the sigmoid loop, most generally affected by pathological sacculi is at times almost devoid of the normal sacculation.

The name in this matter is of great fundamental importance, because it involves a principle on which the progress of surgery depends. If each small part of the body is to be considered as the possessor of diseases peculiar and special to itself, then all real advance is impossible. The fact is, and all educated specialists recognize it, that the same pathology is applicable to all parts of the body, and that in similarly constituted parts of it even the smaller details of disease are, in essential qualifications, alike. The signs of resistance of the tissues to their enemies (pathology) are relatively simple in their manifestations when compared with anatomical and physiological problems.

Another reason for my objection to the term "diverticulitis" is that since Meckel's diverticulum was known this name has had a special significance. This anomaly is of congenital origin; all coats of the intestine enter into its composition; it has a special vascular supply of its own; is single, and is found at any age. These qualifications are common to all the ordinary intestinal diverticula, such as the vermiform appendix, and those associated with small pancreatic outgrowths.

The sacculi are not of congenital origin, but are acquired; their walls are thin and not composed of all the coats of the intestine; they have no special vascular supply; they are multiple, and rounded in shape and almost limited to elderly adults; they have been found in all of the hollow viscera and are of an importance not yet fully realized. There is the same difference between a sacculus and a diverticulum as there is between an acquired and a congenital hernia.

The only excuse for confounding them with the diverticula is that the pathology of both is the same. Both may harbour fæcal concretions or foreign bodies, and inflammation in them leads to the same results—gangrene and perforation and general peritonitis; ulceration and sloughing with abscess formation; or inflammatory thickening and fibrosis, closely resembling a malignant growth of the bowel. The last condition is described, especially by American authors, as "peridiverticulitis," and it is a very important one, but *not* special to the colon. It may be found in a portion of intestine, reduced after strangulation; it has been found in the stomach (and called linitis plastica); it occurs in the skin (and is called keloid or pseudo-keloid); it occurs in the cæcum (and when due either to subacute sepsis or to tubercle is apt to be called hyperplastic tuberculosis). In all, it is difficult to distinguish clinically, and in some pathologically, from cancer; in all it may be due to subacute sepsis, to tubercle or to cancer.

If the Sub-section of Proctology is convinced by my reasoning, the name "diverticulitis" will be replaced by "sacculitis," and I think with advantage.

Mr. HAMILTON DRUMMOND (Newcastle-on-Tyne).

During a period of over eighteen months I have examined the intestines of all the bodies upon which post-mortem examination was made in the Royal Victoria Infirmary, Newcastle-upon-Tyne, with a view of investigating the ætiology of pathological sacculi of the large intestine—about 500 bodies. During this period I met with twenty-two specimens showing marked condition of pathological sacculi, and these were from the bodies of people who died from causes that had nothing to do with intestinal lesion, in many cases they were found in the pelvic colon only.

It was an interesting and somewhat remarkable fact that in five of the cases in which sacculi were found in the colon there were also saccules in other hollow viscera: for example, in four there were saccules in the small intestine, the opening appearing between the leaves of the mesentery; and in one case there were multiple sacculi in the bladder wall. This tendency to diffuse sacculation would seem to have an important bearing upon the causation of sacculi of the colon as it suggests a more or less general weakness (perhaps congenital) of the non-striated muscle tissue throughout the body.

Stress has been laid upon venous congestion as a predisposing cause in the production of pathological sacculi of the colon, and in this connexion I might mention that many of the bodies examined in the post-mortem room, when searching for specimens of sacculi, were cases of chronic heart disease with marked back pressure and in elderly people, and that in none of these were saccules found, a fact which does not support the venous congestion theory.

Intestinal obstruction is another cause put forward in attempting to explain the development of sacculi, but in only two out of the twenty-two cases referred to could obstruction be said to be a factor and these were typical ring cancers with well marked sacculi above the growth.

An examination of the specimens procured during the investigation demonstrated that the sacculi almost invariably took their origin between the mesocolic and one of the lateral bands, and frequently opened into the appendices epiploicæ. They were rarely found in the leaves of the mesentery or on the antemesenteric aspect of the gut.

The appearance of pathological sacculi at these definite points in the bowel wall was, I venture to suggest, accounted for by the anatomical distribution of the vessels, for numerous microscopical sections pointed to the great frequency with which these sacculi pushed their way along the course of vessels. This fact was pointed out many years ago by Klebs and Graser.

Mr. GARNETT WRIGHT (Manchester).

The following is a record of a *case of colicovesical fistula, resulting from diverticulitis of the sigmoid colon.* The patient, a man, aged 45, was seen by me on September 30, 1911. For years he had suffered from a urethral discharge, which had not given him much trouble until six weeks before. Then he began to have pain in the lower abdomen and some difficulty in passing urine. He was catheterized by his doctor, who taught him to pass a catheter for himself. For three weeks he had had pains all over, with occasional rigors, the temperature rising to 103° F. He also had diarrhœa and vomiting, and three days before I saw him he began to pass fæces in the urine. On examination, the pulse was 80, and the temperature 102° F. The abdomen was somewhat distended but not tender or rigid. There was a thick creamy discharge from the urethra. The prostate was rather hard, but no fistulous opening could be felt per rectum. Solid fæcal matter came away with the urine during the whole period of micturition, flatus coming at the conclusion. A diagnosis of colicovesical fistula was made, possibly resulting from gonorrhœal inflammation of the bladder. Treatment with bladder washes for a time was recommended, in the hope that the acute symptoms would subside, but they did not do so. Operation was undertaken on October 30, 1911. The abdomen was opened through a median suprapubic incision, and an appendix epiploica of the sigmoid colon was found to be adherent to the posterior aspect of the bladder, low down in the pelvis. The adhesion was divided, and the fistulous track between the bladder and the colon found in it. The openings, both in the colon and the bladder, were closed by a double row of sutures, the bladder opening being difficult to deal with. The patient had a stormy convalescence, the bladder sutures giving way and urine escaping from the wound. The fistula became re-established. When the wound had healed, a few weeks later, the abdomen was re-opened and the bladder and colon were again separated and sutured. As it was considered possible that the proximity of the repaired portions of colon and bladder might be responsible for the re-establishment of the fistula, the affected loop of sigmoid was, on this occasion, fixed up in the left iliac fossa by several sutures. The abdomen was closed, and the patient made a good recovery.

A curious feature of this case was the occurrence of uncontrollable hiccough after each operation, lasting about a fortnight. The patient left this country a few years later in good health.

Mr. R. P. Rowlands.

During the last ten years I have operated upon eight cases of diverticulitis of the colon and one of the duodenum. Three of these operations were for peritonitis, due to perforation of a diverticulum, and are reported in "The Operations of Surgery," ii, p. 437. The remaining five were for intestinal obstruction, and were of great interest to me. Two of these were published in the *Lancet* for 1910, i, p. 1194, and may be briefly mentioned here.

Case I was that of a very feeble, thin, anæmic lady, aged 60, who had suffered from constipation all her life, with mitral regurgitation, almost complete intestinal obstruction, with œdema of the legs and ascites. Carcinoma of the sigmoid colon was suspected. The colon from the splenic flexure almost to the rectum was rigid, very thick and inflamed and almost solid. She was too ill for resection. A short circuit was made with difficulty between the ileum and the lower part of the pelvic colon. This saved her life but did not cure her. For over a year she suffered from flatulent distension, and looseness of the bowels. Secondary resection was declined. Gradually the diarrhœa ceased. Ten years later Dr. Ryle writes that "she has gone on well on the whole, though living a more or less invalid life. She has suffered all along from great flatulent distension, and the bowels always require the assistance of cascara. She has had no serious or acute affection of the bowels since the operation."

In *Cases II and III*, men aged 49 and 40, resection of 6 to 8 in. of the pelvic colon was performed for what appeared to be a malignant disease. It was only after the operation in each case that an accurate diagnosis was made and the diverticula found after opening the bowel. Microscopic examination revealed the absence of carcinoma. In each of these there was stenosis with a definite tumour the size of a hen's egg. Both of these patients had rigors before operation. Both of them have remained quite well and worked very hard for nine years. In one of these, resection with immediate end-to-end union was adopted and proved most satisfactory. In the other, resection with fixation of Paul's tubes in the open ends of the colon had been adopted elsewhere. I closed the artificial anus, which had become intolerable, some three years later. This proved very difficult.

Case IV was that of a lady, aged 60, who had many attacks of pelvic peritonitis during twenty years. Appendicectomy had been performed elsewhere and a tumour then discovered in the pelvic colon. Three years ago several diverticula containing calculi were removed from the pelvic, iliac and descending colon, and the tumour, localized to the anterior wall of the pelvic colon, was incised and found to contain two diverticula full of pus. These were excised and the bowel closed. The patient was well for a year, but since then she has had several milder attacks, but these have not been severe enough to call for further operations. This case illustrates the well-known difficulty of finding and removing all the diverticula, and points to cylindrical resection of the diseased segment of bowel as the operation of choice.

Case V was that of a man, aged 40, who had suffered from many attacks of pelvic peritonitis, supposed to be due to appendicitis. Four years before I saw him he had developed a colicovesical fistula and was told he had inoperable cancer of the bowel. He was in a poor state and very miserable when he came to me. The fistula was closed by laparotomy: it was between the middle of the pelvic colon and the left side of the bladder. The openings in the bladder and colon were closed and inverted. There were extensive adhesions with pockets of pus in them. The patient died of pelvic peritonitis and pericarditis a week later. No diverticula were found at the operation, but several were found from within the lumen of the bowel after death. In such a late and difficult case it is probably wise to start with a preliminary colostomy. This immediately relieves the very troublesome cystitis and sepsis and makes the subsequent operations easier and safer.

In conclusion I would urge (1) the importance of keeping diverticulitis of the colon in mind, when operating for stricture of the colon.

(2) That early resection with end-to-end union is the operation of choice when a stricture has developed.

(3) That temporary colostomy is indicated in late or complicated cases, especially when distension cannot be overcome before operation, or a colicovesical fistula exists.

(4) That in some late cases a short circuit may be made, followed by subsequent resection, if necessary and possible. Preferably the transverse colon should be joined to the pelvic colon. The transverse colon is generally healthy and low in these obstructive cases. The subsequent resection is thus made easier and more satisfactory. Failing the transverse colon, the ileum or cæcum may be joined to the pelvic colon.

(5) When temporary colostomy is performed it appears wise to make the opening only a little above the stricture, so that it can be removed with the stricture at the secondary operation, and a third operation thus avoided.

(The Meeting was adjourned to January 14.)

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. J. P. Lockhart-Mummery, F.R.C.S.

DISCUSSION ON DIVERTICULITIS.[1]

The President.

When diverticulitis was first described it was looked upon as a pathological curiosity rather than as an important clinical discovery, but I think it is generally recognized now that diverticulitis is a most important clinical condition, and that it is nothing like so rare as was at first supposed. One of the first papers on the subject was written by Sir Humphry Rolleston, now President of the Royal Society of Medicine. His paper was called "Pericolitis Sinistra," and was published in the *Lancet* of April 1, 1905.[2]

Perhaps the greatest importance of diverticulitis lies in the fact that it is easily and frequently confused with cancer of the sigmoid or rectum. For this reason the definitive diagnosis between it and cancer is of great importance, and I hope we shall hear some interesting discussions upon this point. The importance of being able to distinguish between these conditions lies in the fact that if diverticulitis is mistaken for cancer it is always mistaken for inoperable cancer, as the tumour is large and fixed by adhesions, hence it usually follows that no operation is performed, and unless, which is rarely the case, complete obstruction is present, it thus may happen that a patient, who might have been saved by an operation, is left until serious septic complications have arisen, and it is too late to benefit him much by operation.

One of the most valuable means of diagnosing diverticulitis in its early stages is by means of the X-rays. The beautiful photographs shown by Dr. Spriggs demonstrate what can be done in this respect.

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[1] At a meeting of the Section, held January 14, 1920.

[2] *Lancet*, 1905, i, pp. 854, 860.

As regards the clinical points in diagnosis, the occurrence of acute inflammatory attacks with high temperature and rigors is a point very much in favour of diverticulitis as against cancer. The absence of blood in the stools is also of some importance. The fact that a large tumour has existed for some long period, and yet the patient shows no signs of secondary deposits, is also in favour of diverticulitis. It is quite possible for a surgeon to explore the abdomen and handle one of these tumours during an operation without being able to distinguish its nature, but a large tumour with very extensive adhesions should make one suspect it, and then a careful hunt for diverticula will possibly enable a diagnosis to be made. Perforation of one of the diverticula into the bladder is a not uncommon complication. It occurred in four of my twenty-four cases. In each of these cases there was an acute cystitis with rigors, and flatus and fæces were passed *per urethram*. In all four the perforation healed in time after a colostomy above the stricture had been performed. It would be interesting to know how many of the cases of perforation of the bladder from the sigmoid, reputed to be due to cancer as in the series reported by Mr. Harrison Cripps, were really cases of diverticulitis. I myself have never seen actual perforation of the bladder from cancer of the intestine in the sense that there was a passage between the two viscera.

In my series there are two cases of secondary carcinoma supervening upon an old diverticulitis. In both cases the patient had been operated upon previous to the onset of carcinoma, and there were no signs of malignancy at the time of the operation. In one case the carcinoma appeared to start in the walls of a sinus which had perforated the abdominal wall. In the other some part of the tumour had apparently become malignant. In both instances the patient was suffering severely from sepsis around the diverticula at the time of the operation, and this sepsis failed to clear up when the fæces were deflected, and had continued up to the time when obvious malignant disease started. I am inclined to attribute the malignant change to the irritation of chronic sepsis in and around the diverticula, and I think that for this reason colostomy should be performed as soon as possible if any septic symptoms are present, even if the obstruction is not serious enough by itself to warrant it.

The specimen shown by Mr. Grey Turner raises a most interesting point. In the lower part of the specimen there is a small carcinoma, and it looks as if the diverticula, which are in the bowel above it, had possibly developed as a result of the stricture caused by the growth.

If this is so, it means that we may have diverticulitis secondary to carcinoma in the bowel, as well as the reverse process.

Treatment.—As Dr. Maxwell Telling, who opened the discussion, said, the ideal treatment is to resect the mass, and rejoin the ends of the bowel, or leave the patient with a permanent colostomy opening. I think most surgeons will agree with me in saying that this is too optimistic a view. In practice it is seldom possible on account of the dense adhesions to surrounding structures. Nor is it advisable to attempt to separate these adhesions. They are not only very dense but intersected with septic foci, which, if disturbed, will very probably set up acute peritonitis. It must be comparatively seldom that the surgeon has the opportunity of performing a complete primary resection in these cases. In my series only four cases could be treated in this way. Three, however, who were so treated made good recoveries, but in one (Case 10, p. 82), resected by Sir Gordon Watson, the diverticulitis recurred in the descending colon, necessitating a subsequent short circuit. The patient has, however, done well, and this is the case the X-ray photographs of which Dr. Jordan is exhibiting. In the great majority of cases resection of the affected portion of the colon is not possible, and the alternative lies between short-circuiting the stricture or performing a colostomy above it. If a short circuit is done, it is important to be sure that only healthy bowel is used for the anastomosis, and that the tumour itself is not interfered with. In most cases colostomy is alone possible, and transverse colostomy will have to be performed in order to get well above the diseased area. Patients, not unnaturally, object to the performance of colostomy, but I would strongly urge that, in view of the very serious septic complications which tend to occur, operation should be performed early, and that we should not wait until it becomes urgent. The results of colostomy in these cases are extraordinarily satisfactory. If the disease has not progressed too far before operation, it becomes completely arrested after colostomy, and the patient regains normal health. After a time, when all signs and symptoms of active trouble in the diseased section of bowel have disappeared, the question of resection and closing the colostomy can be considered, and I am assured that this is a far safer plan than attempting to do a primary resection in a doubtful case. I would most strongly urge that the operation of choice in diverticulitis is colostomy above the obstruction, or short-circuiting it, and that only in exceptional circumstances should an attempt be made to do primary resection of these highly septic tumours. In cases accompanied by perforation of the bladder, colostomy is indicated even if resection can be done later.

As will be seen from the printed table of cases, my series show an average age of 60, which corresponds very closely with that of other observers.

TABLE OF CASES OF DIVERTICULITIS.

	Sex	Age	Diagnosis confirmed by operation	Region	Main symptoms	Length of history	Treatment and results
1	F.	63	Yes	Sigmoid	Chronic obstruction; pain	—	Patient died from another surgeon attempting to separate adhesions; general peritonitis
2	M.	68	Yes	Sigmoid	Pain and chronic constipation	Three years' history of pain; tumour felt iliac region	Patient operated on for supposed cancer; the abdomen was reclosed and patient treated with petroleum
3	M.	65	Yes	Sigmoid, lower end	Pain and chronic obstruction; painful meso-iliac fossa	Several years	Diagnosis by sigmoidoscope and X-rays previous to operation; ileo-sigmoidostomy; patient well six years later
4	M.	65	Yes	Sigmoid	Acute obstruction relieved with difficulty; tumour in iliac fossa	Two months	Complete resection successfully performed by the Mayo brothers
5	M.	64	No	Sigmoid	Chronic obstruction	Fifteen years	—
6	F.	48	No	Sigmoid	Pain in abdomen; bleeding from bowel; diarrhœa and constipation: tumour felt	—	—
7	F.	55	Yes	Sigmoid	Chronic obstruction and abdominal pain; tumour felt	Two years	Exploratory laparotomy, nothing done; patient well six years later
8	F.	62	Yes	Sigmoid	Severe constipation; acute obstruction; colostomy performed four years previously	Seven years	Colostomy; fixed tumour with no ulcerating surface; patient well
9	M.	57	Yes	Sigmoid	Tumour; pain; obstruction	Two months	Colostomy
10	M.	46	Yes	Sigmoid and descending colon	Obstruction and pain; tumour	Eight years	Colostomy and resection; subsequent closure of colostomy; two years later a short circuit had to be done because pericolitis had spread up descending colon; well two years later

TABLE OF CASES OF DIVERTICULITIS (*continued*).

	Sex	Age	Diagnosis confirmed by operation	Region	Main symptoms	Length of history	Treatment and results
11	F.	53	Yes	Sigmoid	Chronic obstruction	—	Colostomy; alive
12	M.	48	Yes	Sigmoid	Chronic obstruction	Three years	Resected and permanent colostomy left; patient alive and well three years later
13	M.	59	Yes	Recto-sigmoidal junction	Chronic obstruction; tumour	Two years	Colostomy performed, recovered; very large concretions were present in the diverticula
14	F.	58	No	Sigmoid	Tumour; temperature; obstruction	Twelve years	Alive; has to take large doses of petroleum daily
15	M.	59	Yes; colostomy	Sigmoid and recto-sigmoidal junction	Tumour; abscess; chronic obstruction, frequent high temperature; *perforation into bladder*	Four years	Colostomy; died of secondary carcinoma two years later
16	F.	56	No	Sigmoid	Temperature at frequent intervals to 103° F.; chronic obstruction; tumour	Two years	Alive and well two years later
17	M.	56	Yes; colostomy	Recto-sigmoidal junction	Pain; chronic obstruction; *perforation of bladder;* temperature to 103° F. often; tumour	Eleven years	Colostomy; patient completely recovered: quite well three years later
18	M.	55	Yes; colostomy	Recto-sigmoidal junction	*Perforation into bladder;* tumour; high temperature; obstruction	Two and a half years	Colostomy, transverse
19	F.	57	Yes; resection	Sigmoid	Obstruction, thought to have cancer	Six months	Involved bowel resected and end-to-end union; patient died some time later from bronchitis
20	M.	64	No	Sigmoid	Chronic constipation; attacks of obstruction; temperature; tumour	Twenty years	Doing well on petroleum
21	M.	47	No	Sigmoid	*Perforation of bladder;* chronic obstruction; tumour; temperature	Four years	Alive
22	F.	54	Yes; colostomy	Sigmoid	Chronic obstruction; acute obstruction	—	Colostomy; well six years later
23	M.	62	Yes; colostomy	Sigmoid	Chronic obstruction; high temperature; tumour	—	One and a half years after colostomy patient developed secondary carcinoma
24	M.	56	Colostomy	Sigmoid	Semi-acute obstruction	—	Colostomy; died

Mr. W. Ernest Miles.

It seems that everything that can be said has already been said concerning this very interesting subject, so that there is very little for me to add. Certainly, from what we have seen demonstrated by excellent specimens and skiagrams, there is no room for doubt that diverticulitis is a very real disease and should engage the attention of every abdominal surgeon. Whether it is a comparatively new form of disease due to modern modes of life, or one which has existed, though undetected, throughout the ages is not easy to determine; but, at any rate, its ætiology should not be obscure, and here there seems to be a fertile field for investigation. Dr. Maxwell Telling has told us that, in many cases, there is often an antecedent history extending over many years; so that it may be possible, by careful inquiry, to associate with its onset certain conditions or habits which might prove to be definite causative factors. I am inclined, with Professor Rutherford Morison, to put the idea of congenital origin out of court. The fact that these diverticula often exist in considerable numbers, as shown in the skiagraphs, supports this view. Much more likely a cause is increased intracolonic pressure, the diverticula being hernial extrusions of the mucosa through the muscular coats of the bowel at the weak points created by the passage of blood-vessels, the disposition of which has been so ably demonstrated by Mr. Drummond. If, then, this be the correct interpretation of the ætiology, and it seems feasible enough, it follows that any circumstance which tends to increase intracolonic pressure is a possible factor in the causation of diverticulitis. It has been suggested that flatulent distension of the colon may be the determining factor; but I doubt it, because gas-pressure is so evenly distributed. It appears to me that, in everyday life, intracolonic pressure, especially in the pelvic colon, may be increased in three ways at least:—

Firstly, by forcibly resisting the desire to evacuate the bowels. In these days of rush, when men and women have to catch trains in order to get to business, it often happens that late rising leaves only sufficient time for a hurried breakfast, and, in consequence, forcible contraction of the external sphincter muscle has to be relied upon to avert calamity during the train journey. During this period intracolonic pressure must be enormously increased. Secondly, intracolonic pressure is greatly increased by straining at stool. Forcible straining occurs when a formed motion is passed through a narrowed anal orifice. Apart from stricture of the rectum, which of course would have that effect, there is a very common condition of the anal canal which limits the normal degree of expansibility of the external sphincter muscle and necessitates straining during defæcation. I allude to a deposit of fibrous tissue in the submucosa of the pecten which I have described under the name of the pecten band in an article entitled "Observations upon Internal Piles."[1] In that article I pointed out that the two chief indications of the presence of the pecten band were (1) difficulty in emptying the rectum during defæcation, and (2) diminished size in the calibre of the fæces passed. I also pointed out that both of these symptoms disappeared after the pecten band had been divided. The presence, therefore, of a well developed pecten band, by causing straining at stool, would undoubtedly be a factor in the production of increased intracolonic pressure.

Lastly, in the constipated, there is a great tendency to the habitual use of copious enemata in order to procure an action of the bowels. An enema consisting of two or three pints of fluid is often resorted to, and there can be no question that under such circumstances intracolonic pressure is greatly increased.

It would, therefore, be highly interesting if the previous histories of cases of diverticulitis were to reveal that either of these causes were operative, as then prophylaxis would be easy. When the disease has become established surgical intervention is inevitable and risks must be taken; but, if definite causes can be recognized, prophylactic measures can be adopted on the principle that prevention is better than cure.

Dr. ALFRED C. JORDAN.

This most interesting and valuable discussion has thrown much light on the nature of the changes which occur in the large intestine in diverticulitis. There has been remarkable agreement in the views of speakers as to the pathology of the disease, the only important point of difference being as to whether the disease is congenital or acquired. In my small share in the discussion I shall endeavour to show how far the X-ray investigation of these cases helps in determining the ætiology and the pathology of diverticulitis.

All the patients in my series conform to the rule that typical diverticulitis is a condition peculiar to stout elderly persons. They are not, however, those born to wealth and an easy life—those who "laugh and grow fat"; but those who have won high position and affluence by a life of strenuous effort.

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[1] *Surg., Gynæcol. and Obstet.*, November, 1919.

(1) The first slide shows the iliac colon in a lady, aged 63, whose history is, in my experience, a very typical and suggestive one. Twenty years previously she had suffered from dysentery while living in Mexico. Ever since she had complained of attacks of "colitis" with mucus and blood in the stools. She was given careful medical treatment, to which she responded so well that now, eight years later, she is free from intestinal trouble.

(2) The next case is one well known to several of those who have taken part in the discussion. A man, aged 54, with typical diverticulitis of the iliac colon, presented the usual X-ray appearances of a constricted central lumen and isolated pockets in which bismuth was imprisoned. There was enough hyperplasia to give rise to a tumour, and to produce considerable obstruction. Mr. Lockhart Mummery diagnosed the nature of the condition. In conjunction with Mr. Mummery, Sir Arbuthnot Lane operated, short-circuiting the ileum into the rectum below the obstruction. As the bowel above the union was greatly reduced in lumen the hope was entertained that the ileal contents would not make their way up into the descending colon. A few months later, however, it was found that the iliac colon had opened up almost to the normal diameter, so great had been the benefit produced by the relief of pressure. By adopting very regular habits[1] the patient has overcome the undesired penalty of his great improvement, and at the present time, ten years after the operation, he is well and able to perform an abnormally large amount of work.

(3) The next case is that of a man, aged 46, who was sent to me by Mr. Mummery to investigate the condition of the big bowel. Two years previously a diverticulitic pelvic colon had been excised and colostomy performed, and a year later a lateral anastomosis had been made between the pelvic colon and the middle of the transverse colon. I found, as usual in such cases of anastomosis without division of the bowel, that the intestinal contents did their best to ignore the existence of the artificial opening. In this case, however, the obstruction to the

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[1] He has established the habit of evacuating after each meal and insists on the importance of the "evening stool." Each morning, beginning at the lower end of cæcum, he gently massages the whole of the cul-de-sac colon to the rectum and so gets rid of the results of regurgitation.

normal passage was so severe that the new way could not be ignored. Nevertheless, a thin line of bismuth material forced its way into and through the descending colon. In this case a condition of severe colitis recurred after the closure of the iliac colostomy.

(4) Leaving now the most usual site of the disease, we come to an instance of diverticulitis in the splenic flexure region in a very stout man, aged 67. The obstructed portion of bowel in this case was the splenic flexure, and, as usual, it was the length of bowel immediately proximal to the obstruction which had become subject to diverticulitis. The splenic flexure is seen to take a spiral course. The transverse colon was found to be adherent at a point close to the splenic flexure, so that in the upright posture a sharp kink appeared at the tethered point. The diverticula are seen to be just proximal to the kink. This case exemplifies the well-known tendency for the occurrence of malignant disease, stated by William Mayo to amount to 30 per cent. of all cases of diverticulitis of the large intestine. Some months after I saw this patient the liver began to enlarge, and it became evident that malignant disease was present. The patient has since died.

(5) The next case illustrates diverticulitis as it occurs in the hepatic flexure region. This is the hepatic flexure of a man aged 43, under the care of Mr. Donald Armour. There was a history of fourteen years of attacks of colic. There is the typical narrowed lumen of the upper part of the ascending colon, and a row of isolated sacs along the inner side of the ascending colon and in the concavity of the hepatic flexure. Note especially the spasmodic contraction of the transverse colon with a tendency to the extrusion of diverticula from it.

(6) The next slide illustrates the mode of origin of diverticulitis in the large intestine. We radiologists are told that we must distinguish between true and false diverticulitis. The chief points of distinction are said to be two: (1) False diverticula are the saccules of the big bowel, brought into unusual prominence by extra tight contraction of the haustra of the bowel; if an opaque enema be introduced into the bowel, these "false" saccules disappear as the lumen of the bowel widens to the normal diameter. (2) The false saccules, being protrusions of the entire bowel wall, consist of all the coats of the bowel, whereas true diverticulitic sacs consist of mucous and serous coats with no muscular coat.

Now all my observations have led me to the conclusion that the origin of diverticulitis is in colitis. Not only does colitis precede diverticulitis, but it continues to affect other parts of the large intestine after the appearance of diverticulitis. The slide now on the screen illustrates the manner in which diverticulitis comes into being. The catarrhal state of the mucous membrane induces tight haustral constriction of the bowel, with exaggerated sacculation between the haustra: in fact the condition known as "false" diverticulitis. After this condition has persisted for years it becomes permanent, the haustra never relaxing, and the lumen of the bowel never opening up fully. The bowel forces its contents through its narrowed lumen in small amounts suspended in mucus. The iliac colon is the part of the large gut which becomes most tightly and persistently contracted as the result of long-continued catarrh. Small-cell infiltration occurs, leading to the formation of a tumour. When this stage has been reached it is easy to see that the saccules must lose their muscular coat, for:—

(1) The whole bowel wall is fatty and sodden.

(2) The circular muscle, being in a state of spasm, cannot remain on the sides of the saccules, but must become retracted down to the bowel wall at the base of the saccules.

(3) The muscle layer over the far end of the saccule is attenuated by pressure from within the bowel, and must be absorbed gradually, leaving perhaps a vestige such as has been described by more than one speaker.

Thus we have a complete explanation (so it seems to me) of the origin of diverticulitis, of its pathology, of its most usual situations, and of the type of patient in whom it occurs.

The catarrh of the mucous membrane which leads to colitis is most obstinate; it persists in many cases in spite of all efforts to allay it. The catarrh itself is due primarily to stasis in the large intestine; secondarily to microbic infection. It follows, accordingly, that diverticulitis is a special manifestation of chronic intestinal stasis.

If these views be admitted, then there is little need for a discussion as to the congenital or acquired nature of the disease. Those who regard it as congenital wish us to infer not that the saccules are present at birth, but that the weakness of the bowel wall, which later in life results in diverticulitis, is present at birth. Now the saccules of the large intestine, once they are established, are permanent, for the circular muscle is far thicker in the position of the haustra, thinning away over the saccules. The exact position of the diverticula is determined solely by the weakest part of the circumference of each affected saccule. Some of the beautiful pictures we have been shown indicate beyond doubt that the place where the arteries perforate the bowel wall is the weakest part of the circumference of the saccule. The case upon which I have founded my argument should be described as in a "prediverticulitic" stage; it may never go on to the stage of true diverticulitis, but given the right conditions it must certainly do so.

The next two slides illustrate the persistent, almost permanent constriction of the iliac colon, which goes on eventually to diverticulitis under suitable conditions. The two next show early diverticulitis in the upper and the lower part of the ascending colon.

Dr. Spriggs showed a beautiful instance of duodenal diverticulum; I feel bound to devote a word to it since he expressed the rather astonishing view that there was no analogy between it and the diverticula of the large bowel which form the subject of this discussion. His view is especially astonishing since it was expressed in connexion with a case which, as he said, constitutes almost a perfect proof that they *are* connected—viz., a case in which there were both large intestinal and duodenal diverticula. The subject of the slide I am showing was a lady aged 59—very stout, brought to me by the good friend we have all lost—Sir William Osler. It seems quite evident that these duodenal diverticula are produced by pressure, as in the case of the large intestine, but they differ in two main respects: they are not so apt to become the foci of tumours, and their origin is perhaps to be regarded as congenital, inasmuch as they generally occur close to the ampulla of Vater. A very small sac enlarges under pressure with advancing years until it becomes large enough to give rise to symptoms, and to be a noticeable object.

As an example of a truly congenital diverticulum there exists no better instance than Meckel's diverticulum, as in the instance now shown on the screen of a man aged 55, brought to me by Mr. Armour for lifelong constipation, and recent attacks of subacute obstruction. Mr. Armour removed the diverticulum, and the result was completely successful.

Other pressure sacs, comparable in origin with those of the large intestine, occur in the œsophagus: (*a*) At the upper end—pharyngeal pouch; (*b*) just below the bifurcation of the trachea—digital pouch.

Lastly, in the bladder, when there is a urethral stricture in stout, elderly men, multiple diverticula occur with small orifices protruding through the muscular coat. These were admirably described by Sir Arbuthnot Lane as long ago as 1884, and published by him in the *Guy's Hospital Reports* for 1886.[1] Sir Arbuthnot Lane is the greatest prophet in the land; it will surprise no one to learn that this paper also contains a most full and true description of the appearance of diverticulitis of a loop of large intestine found in the sac of an inguinal hernia, and a complete explanation of its pathology.

Dr. F. HERNAMAN-JOHNSON.

Dr. Spriggs and Dr. Jordan have already said most of what can be said from the X-ray point of view; but I should like to lay stress on the fact that the most satisfactory radiological evidence of the existence of permanent diverticula (or saccules if the word be preferred) is the presence of small round barium masses in clusters after evacuation of the meal or enema as a whole. The appearance presented would appear to be quite characteristic. And it is never, so far as I know, mimicked by spasm or by chance residues.

Another point is that, if diverticulitis is suspected, but not revealed by a meal, the administration of belladonna to the limits of safety for thirty-six hours, followed by thorough cleansing of the lower bowel by oil enemas, will greatly increase the chance of demonstration by a barium clyster. In fact the same care should be taken to secure patency of the diverticula as is done in the case of the appendix, when it is desired to show it by X-rays. The administration of belladonna has also the advantage of relieving many forms of spasm which might otherwise cause one to suspect the presence of organic stricture.

Mr. LIONEL NORBURY.

I have very little to add to what has already been said, but there are one or two points which I would like to mention. I have only had one case which I have operated upon for diverticulitis, and that was the case of a lady aged 43, who gave a fifteen years' history of constipation, and an attack of diarrhœa eleven years previously to the operation, this attack having persisted for a month. In addition, for six months prior to the operation she had severe griping pains in the abdomen. A lump was evident in the left iliac region, and its nature being doubtful, the operation must be called an exploratory one. She had previously had her right ovary removed on account of tuberculous disease, and one suggestion made about her condition when I saw her was, that she had a similar tuberculous condition in the other ovary. At the operation, a large adherent mass was found in the region of the iliac colon, which looked very much like carcinoma, though there were one or two features about it which negatived that view. If it was carcinoma, it was inoperable. As there was a chance it was diverticulitis, I resected 5 or 6 in., closed the ends and performed a lateral anastomosis, and she got well. On examining the specimen, which will be found on the table here, the gut was found to be much contracted, and the walls showed much hypertrophy and there was an abundance of fatty tissue.

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[1] *Guy's Hosp. Reports*, 1886, 3rd ser., xliii, pp. 29-52.

Description of Specimen.—The gut is much contracted. In the lower right-hand half there is an irregular cavity in the surrounding fat, due to "Perforating Diverticulitis." In the recent state the cavity contained pus and pellets of inspissated fæcal matter. Microscopical examination showed all the signs of acute and chronic inflammation. No evidence of malignant disease.

I think that an important point about diverticulitis is its ætiology, and, personally, I think enterospasm is a predisposing cause, that it produces hypertrophy of the coats of the bowel, and that this is increased by the constipation which ensues. The bowel wall becomes weakened at certain points, owing to excessive pressure; infection takes place, and therefore, in such cases, the diverticula may be looked upon as "pressure diverticula." A section through the wall of one of these diverticula shows a well-marked muscularis mucosa and mucous membrane with Lieberkühn's glands also well marked. I saw a very good specimen of this a short time ago. The wall of the diverticulum may give way, resulting in infection of the surrounding fat, which in these cases is often superabundant. I think it important to try to ascertain what is the cause of this excess of fat in these cases; it is seen in several of the specimens on the table. One likely explanation, I think, is that it may be due to passive congestion the result of enterospasm. In many parts of the body one gets much fat as the result of passive congestion.

Another point is, that I think the sigmoidoscope may be of use in detecting certain cases of this condition, for in it the lumen of the bowel is very much narrowed. Recently, in the Out-patient Department of St. Mark's Hospital, I have seen several cases, by means of the sigmoidoscope, in which the pelvic colon was suddenly narrowed, not as the result of the occurrence of a fibrous stricture, but apparently due to spasm and hypertrophy. I know that with the sigmoidoscope one sometimes has a difficulty in getting round bends, but that was not the case in the instances I am referring to. It is in cases such as those that one expects to find diverticulitis.

I have recently had another case which I presume to be one of "diverticulitis": Male, aged 36, with a history of long-standing constipation, more severe for one month previous to examination, with the passage of large quantities of mucus and occasional hæmorrhage from the bowel. Persistent pain and some tenderness beneath left costal margin, with an increased area of splenic dullness. Very large offensive clots passed a few days before operation. Considerable loss of flesh during the last month. Sigmoidoscopy revealed several large internal hæmorrhoids, a much dilated rectum, with enlarged veins, but no ulceration nor growth in the rectum or pelvic colon. At the operation a fixed mass was found involving the splenic flexure of the colon, with much enlargement of the liver and spleen. The condition was thought to be a carcinoma and inoperable. A lateral anastomosis between the lower part of the ileum and pelvic colon was performed on August 28, 1919. The patient has since put on several stone in weight and is quite well and strong, and personally I am inclined towards a diagnosis of "diverticulitis."

MR W. SAMPSON HANDLEY.

I think the success of this discussion is largely due to the clear and complete outline of the disease which was laid down by the opener, and which has provided such an efficient skeleton for the subsequent debate.

My own experience of diverticulitis is but a small one, and it happens to relate chiefly to cases in which local peritonitis arose from perforation or bacterial penetration of the diverticulum. A perforation just above a malignant growth will produce exactly similar conditions, and the differential diagnosis becomes a matter of great importance.

It has seemed to me that in diverticulitis one does not find the "tied string" which is so characteristic in sigmoid carcinoma, except in its very early stages—i.e., an appearance as if string had been tied round the gut. In diverticulitis, so far as I have seen these cases, the induration is generally restricted to one aspect of the bowel, while at the same level other aspects of the bowel are not contracted. In this particular type of diverticulitis, namely, after perforation or bacterial penetration, I feel sure from my experience that simple drainage of the peritoneum is, often, all that is necessary. If obstruction be present, which is not usually the case, then a colotomy is the right treatment. But I feel that resection of the bowel would be most dangerous in such cases, and in this respect I am in entire agreement with the remarks which our President has made.

One difficulty in a discussion of this kind is, that one meets clinically with cases in which, though diverticulitis may be suspected, the interests of the patient are incompatible with the steps necessary either to establish or to disprove the tentative diagnosis. Thus I saw, with Dr. Thornton, of Acton, a lady aged 45, who had a tumour in the right renal region and symptoms of partial obstruction, with visible coiling. Operation disclosed a local lump in the ascending colon, adherent to the kidney, with cellulitis of mesentery and colon at that level. Section of a piece of the mesentery showed no sign of tubercle, just the appearance of cellulitis. A lateral anastomosis from the ileum to the transverse colon permanently relieved the patient, and the diagnosis remains uncertain, though, I take it, it is extremely probable that it was a case of chronic diverticulitis.

I have once seen a specimen which showed a diverticulum with carcinomatous infiltration around it. I made a thorough hunt through my specimens the other day in order to find this one, but I failed, and therefore this observation can have little value, but I thought it was of sufficient interest to relate it to the meeting.

Sir CHARTERS SYMONDS.

Some of the remarks the President has made induce me to at least ask one or two questions.

Looking back upon my surgical experience of many years, I can now, I think, as a result of what I have heard at these two meetings, explain several cases which, up till now, have always puzzled me. I believe they have been instances of diverticulitis.

One, a man aged 65, had obstruction, for which I performed colostomy. From the rectum a mass could be felt, and on opening the abdomen a large irregular mass was found attached to the pelvic colon, which I took to be malignant, and I considered the case to be inoperable. After doing the colostomy, therefore, I left the man to his fate. What was my surprise, therefore, when, a year later, he walked into my room and asked for a certificate. He was perfectly well, and his colostomy wound had closed spontaneously.

Another was a very interesting case, which confirms the statement which the President has made, namely, that communication with the bladder is rarely, if ever, due to malignant disease, but is due rather to the process which he has described. It occurred in the person of a tutor at one of our colleges. There was a mass which could be felt *per rectum.* He had a communication with the bladder, and passed fæces and gas by that route, and tubercle bacilli had been found in the fæces. I did a colostomy, and he got perfectly well; he put on weight, and the communication with the bladder closed. He lived for some years, his end coming in one of the influenza epidemics. He declined to have his colostomy closed, for, as the President himself has said, the cases which get well are very comfortable.

I recall, also, the case of a lady upon whom I had to operate. I found an abscess in connexion with the transverse colon, which I never could explain, but I now consider it was a case of diverticulitis.

The President also referred to communication with the bladder, and I would like very much to learn what he regards as the early symptoms of that process taking place. What symptoms are there which would lead him to suspect that a communication with the bladder was imminent or that it had become a possibility? I ask because I can recall the cases of several people who experienced pain on the left side when the bladder was full. I recall now the instance of a patient who has pain in the left iliac fossa when his bladder is full, and it is relieved, sometimes completely, by the emptying of the bladder, though sometimes the pain remains, perhaps a whole day, afterwards. Associated with that, there is pain—not of a very severe character, but quite definite—before an action of the bowels, and that pain is relieved by such action. As I say, I have seen several of these cases, and it has been difficult to explain them. The knowledge I have gained in these two meetings, however, indicates that probably they were early cases of diverticulitis in which adhesion to the bladder has occurred. And my question in this connexion is: Ought one to interfere, by colostomy or other measures, in that stage?

The President has also spoken of the value of colostomy in these cases, and has told us that, in his opinion, it was unwise to close the colostomy. I think that is a very important piece of advice. I have mentioned one case in which the colostomy closed spontaneously and the patient remained well, and I was rather of the opinion that it was sound practice to close colostomies after all the local symptoms had disappeared. But I gather that the President, and others, would deem it wiser to retain the colostomy, for fear of any recurrence of the diverticulitis.

The PRESIDENT.

In reply to the interesting remarks of Sir Charters Symonds, I think he somewhat misunderstood me in regard to the question of closure of colostomies. I did not mean to convey that they should not be closed, but that it is seldom we get a case in which the symptoms disappear sufficiently to warrant such closure. It is really a matter of the degree of disease we have to deal with in any given case. I saw one patient four years after colostomy had been done, and examined him very carefully. I found there was still such a dense stricture that a probe could not be passed through: it seemed to have a density equal to that of cartilage. I think it is seldom the local condition disappears sufficiently to justify closure.

I am very interested in what Sir Charters Symonds has said about these bladder cases, and I think he has correctly described the symptoms of early attachment to the bladder. I am afraid I cannot answer his question very well, because I think in all four of the bladder cases I have seen the bladder perforation occurred suddenly: in one it occurred while I had the patient in a nursing home, and that patient certainly had no symptoms suggestive of bladder trouble, beyond some frequency of micturition, until he had a sudden rigor and gas in the bladder.

As to whether one should treat these bladder cases by early operation so as to anticipate perforation, I think perforation into the bladder is such a ghastly condition, and causes such acute cystitis and discomfort, that if one is able to anticipate its occurrence, one ought to use every means of doing so. The real difficulty is to persuade a patient to have colostomy done for a possible trouble which he has not yet experienced.

Sir GORDON WATSON.

The discussion on this subject has been so complete that I do not propose to take up any time except to call attention to a wet specimen which I have brought here this evening. The case was diagnosed as one of carcinoma of the pelvic colon, by sigmoidoscopy, in the out-patient room. The patient was admitted to the ward at St. Mark's

Hospital, and I examined with the sigmoidoscope and confirmed the diagnosis. I opened the abdomen and found a sausage-shaped tumour which had more the appearance of diverticulitis than carcinoma. I resected about 6 in. of the gut and performed colostomy. You will see the microscopical section, which suggests a carcinoma, though even now, looking at the specimen with the naked eye, it is impossible to say whether it is a carcinoma or an inflammatory swelling, and certainly it was impossible to do so at the operation. The case, therefore, illustrates the extreme difficulty which may occur in making a diagnosis between diverticulitis and carcinoma.

With regard to the question of the closure of the colostomy wound in these cases, I should like to refer to a case which Dr. Jordan has spoken of this afternoon. In this case I resected a sausage-shaped tumour for diverticulitis and performed colostomy. The specimen is exhibited here. Mr. Mummery, during my absence at the war, closed the colostomy and the patient subsequently developed recurrent diverticulitis above the anastomosis. Mr. Mummery then had to perform an anastomosis between the transverse and pelvic colon in order to get the patient out of his secondary difficulty. Presumably in this case some stenosis at the line of anastomosis was responsible for the recurrence. The case shows that closure of the colostomy in these cases is not always advantageous.

Dr. MAXWELL TELLING (in reply).

I think the Sub-section of Proctology is to be very much congratulated on the excellent way in which the subject has been talked out, so that there remain very few i's to dot and t's to cross by anyone like myself who replies.

I have listened with very great interest to the various speeches, and I have learned a great deal more about the subject by so doing. One thing which has, I think, been definitely settled is that diverticulitis is a clinical entity, and I think this discussion will have served to set it on its feet, so to speak, and to ensure for it that universal recognition to which its clinical importance entitles it.

There are one or two points which struck me in the various papers. The first concerns the name which should be given to the condition. I think "sacculitis" is likely to cause confusion with the condition of sacculation of the colon, which I think one has seen brought out well in some of the X-ray photographs which have been exhibited. The term "diverticulitis," which has now passed pretty well into current usage, serves sharply to distinguish this condition from mere sacculation of the colon.

With regard to causation, I did not go very much into that question. One speaker pointed out that one of the great reasons that these diverticula entered the appendices epiploicæ was the fact that these appendices were frequently fat-loaded. That was demonstrated in 1893 by Sir John Bland-Sutton, when he described certain cases in which foreign bodies entered these appendices, and he also drew attention to the fact that the fat-loading of these appendices was probably the cause of their becoming hollowed out. I think that is a great reason why these points are areas of weakened resistance, thus leading to diverticula entering them.

Mr. Hamilton Drummond quoted a number of cases in which "saccules" had been found in other hollow viscera. The consensus of opinion seems to be that these diverticula are acquired, not congenital : but I do not think we have really quite sufficiently investigated these cases in which diverticula are found in the small gut and other parts of the intestinal canal. If these diverticula are acquired, it seems to me there is an undue proportion of cases in which we find diverticula in the small intestine, well beyond what one would expect if they were purely pressure diverticula. That is a point which requires more study and the investigation of a larger number of cases.

It is as yet early to say what is the precise frequency of occurrence of these diverticula; but in this connexion I think one point needs to be borne in mind. The diverticula themselves, I believe, will be found to be, after middle life, of some considerable frequency ; but I think only a small proportion of the cases in which diverticula occur go on to the production of symptoms or give rise to trouble ; and it has seemed to me that confusion between the mere presence of diverticula and actual diverticulitis has been present in the minds of one or two speakers. For instance, Dr. Spriggs, in his interesting series of photographs which he showed, wondered whether I would suggest total resection of the gut in all those cases in which diverticula were revealed in the X-ray picture. Most certainly I should not, and I have never done so. I do not think any operation whatever is called for in those cases, whatever the X-rays may reveal, unless clinical symptoms are occurring, and I do not think many of these patients would consent to operation if they were made aware of all the facts. Until the condition is clearly causing trouble, I do not think operation should be entered upon.

I hoped to hear, and I have heard, a good deal that is of interest in regard to the diagnosis of this condition by X-ray examination; the slides and diagrams which have been shown have been very interesting, though I am not sure I have been convinced in every case. In some cases I think the appearances are a little deceptive and fallacious; I also think one has to be particularly careful about calling a sacculated colon in spasm an example of diverticulitis. Some of the diverticula which have been shown have not been examples of what I should regard as "diverticular disease" of the sigmoid segment, and I feel there will have to be a great more work done in the X-ray examination of a larger series of cases, proved by operation, before we can to any great extent rely on the X-ray findings.

Dr. Jordan, discussing the ætiology, said, I think, that every case started as a colitis. I am not sure that I know what colitis is when the term is used in that general sort of way. I suppose no one of us has had a very large clinical experience of these cases; only one case have I seen which seemed to bear out the truth of that assertion. It was a patient of mine who was operated upon, two years ago, by Mr. Dobson, of Leeds, and she definitely had had catarrhal colitis for some seven or eight years. In the other cases which I have come across, however, there was nothing which one could reasonably say was a colitis, judged by the symptoms. And in a number of these cases there has been no constipation, nothing on which one could fix in in support of the idea that colitis had pre-existed.

I have been very interested in the remarks of surgeons on the operation of election in these cases, and I suppose that colostomy, at any rate as a preliminary operation, must be that elected procedure. In the cases which I have personally observed which have come to operation, and subsequently to post-mortem examination, the parts have been so adherent, so septic as to have been enormously difficult or impossible to deal with other than in this preliminary way.

The relationship to malignant disease is twofold. I am sure some cases have developed carcinoma secondarily to the presence of diverticula. In several specimens I have examined I think there has been good ground for supposing that a moderate diverticulum formation has resulted from increased pressure above the site of the carcinomatous stricture. It is a point about which it is difficult to be certain in a given case, but I think it is likely that carcinoma can cause diverticula, and it is certain that diverticula may be followed by carcinoma formation. The case mentioned by Mr. Sampson Handley takes us far in that direction.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Birth Palsy in a Boy.[1]

By R. C. Elmslie, M.S., F.R.C.S.

The boy shown is aged 11, the eldest of three children. Delivery was instrumental, and three days after birth it was noticed that the arm hung at the side and was never used. No treatment by splinting has ever been carried out and no treatment has been given at all for several years past.

At the present time, all the muscles of the right upper limb appear to act, with the exception of the external rotators of the shoulder. The arm is held in a fixed position of abduction, forward flexion and internal rotation at the shoulder joint, only a small range of movement being possible. The elbow is flexed, complete extension being impossible. There is a subluxation of the head of the humerus backwards, so that it forms a prominence below the spine of the scapula.

X-ray examination shows that the head of the humerus is flattened and deformed.

The case is of interest because of the marked forward flexion and abduction of the shoulder.

The line of treatment proposed is, first, massage and mobilization of the elbow-joint, with faradic treatment of all the muscles of the limb and active use of the hand, and later the performance of Mr. Fairbank's operation for dividing the anterior part of the capsule of the shoulder-joint and the tendon of the subscapularis muscle, with after-fixation of the arm with the shoulder abducted to a right angle and fully externally rotated for a period of three months, following this up by active physical treatment.

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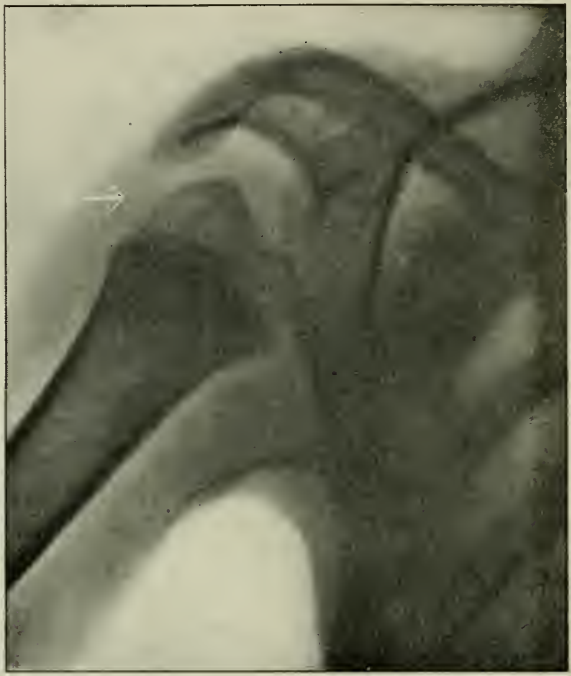
[1] At a meeting of the Section, held November 4, 1919.



Mr. Elmslie's case of birth palsy. Skiagram of shoulder-joint showing flattening of head of humerus and hooking of acromion.

DISCUSSION.

Mr. H. A. T. Fairbank: The position of the limb is not unusual, but is difficult to explain. The subluxation is due probably to a lack of support at the back of the joint, associated with a contracture of the pectoralis major and subscapularis. The abduction is difficult to understand. During the operation, the supraspinatus and the coraco-humeral ligament must be divided before abduction becomes possible. The operation for the division of these contractures and of the subscapularis is advisable in this case. The results on the whole are good, but are not perfect, partly because of the difficulty of getting patients to attend regularly for after-treatment. As to the position for fixation after the operation, I do not now favour Whitman's position with the arm externally rotated, but close to the side, because a prolonged period of fixation in this position fails to secure the necessary abduction of the arm. I prefer to use this position for one month and then to abduct the arm to a right angle and fix this new position for two months.

Mr. A. S. BLUNDELL BANKART: If these cases were all due to a birth palsy, the occurrence of a contracture very soon after birth would be difficult to explain. I have seen cases in which contracture comes on within a few days, and these, I think, must be cases of congenital contracture and not birth palsy at all. In this case the contracture was noted early, and there was apparently some history of trouble with the legs. This, combined with the fact that there is no evidence now of any paralysis, suggests that this is a case of congenital contracture and not of birth palsy at all. In carrying out the operation suggested by Mr. Fairbank, I fix the arm in a position in which the hand lies behind the head.

Mr. LAMING EVANS: The X-ray shows very clearly the projection downwards of the tip of the acromion process as a hook. This is frequently seen in cases of birth palsy and might possibly be found to be pathognomonic of this condition.

Mr. ELMSLIE (in reply): I have seen cases of primary birth palsy with a flail limb develop a contracture within a few weeks. To counteract this I have, as a routine, treated cases of birth palsy with a plaster splint which holds the arm abducted to a right angle and externally rotated. In some cases it is difficult to secure this position when the child is only a few weeks old, because a contracture has already set in. In one case an assistant of mine fractured the humerus by very slight violence in attempting to secure such rotation. I believe the subluxation to be due to shortening of the anterior part of the capsule and of the anterior muscles which lever the head of the humerus backwards out of the joint.

Case of Os Tibiale Sesamoideum.

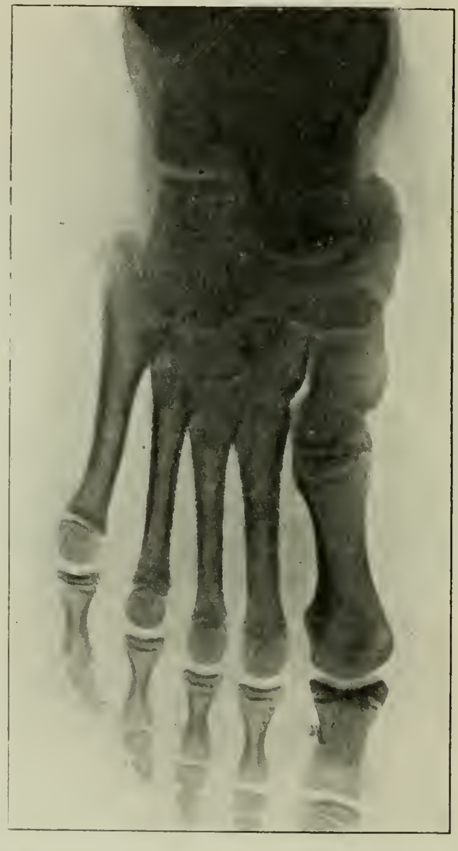
By R. C. ELMSLIE, M.S., F.R.C.S.

THE patient is a girl, aged 11. Three weeks before coming to the hospital she fell down some steps and doubled her left foot under her. From that time the inner side of the instep has been painful. Palpation showed that the tender spot was over the tubercle of the scaphoid.

X-ray examination of both feet led to the discovery that there was a separate ossification for the tubercle of the scaphoid upon either side. On the left side the separate piece of bone appears to be a little rough on its deep surface.

So far the only treatment that has been carried out is immobilization of the foot in plaster of Paris for a fortnight. The plaster was removed a week ago and the pain is still present.

This case appears to be one of os tibiale sesamoideum in which an injury has loosened the separate piece of bone upon the left side and rendered it temporarily painful.



Mr. Elmslie's case of os tibiale sesamoideum.

I propose to fit an outside iron, so as to throw the weight on to the outer side of the foot and so rest the scaphoid. I have seen two previous cases with similar symptoms. In one of these, in a middle-aged woman, I removed the affected piece of bone. The bone was rough upon its deep surface, as if it had been fractured or torn away from a ligamentous attachment.

DISCUSSION.

The PRESIDENT: These cases are interesting, especially in their medicolegal importance. I quite agree with the suggested line of treatment which should relieve the pain.

Mr. H. KELLER: The X-ray shows a small chip off the base of the fifth metatarsal. This might possibly have something to do with the pain. As to treatment I consider that it is necessary to immobilize the foot by some mechanism which cannot be removed. This is preferable to the use of an appliance.

Mr. H. A. T. FAIRBANK: I have here some X-ray photographs showing this condition. The first is from an officer who hurt one foot in a fall, but could not remember which. The separate ossification is unilateral. The second is a case of Mr. Waugh's, in which there appears to be a definite separate ossification. The third is the result of an injury, the scaphoid being obviously fractured.

Mr. LAMING EVANS: I have had a similar difficulty in some cases in distinguishing between a separate ossification for the os trigonum and a fracture of the corresponding portion of the astragalus.

Skiagrams of an Injury to the Neck of the Femur produced in straightening a Flexed Hip in a Case of Infantile Paralysis.

By E. LAMING EVANS, F.R.C.S.

IN this case, whilst I was manipulating a flexed hip under an anæsthetic, after subcutaneous division of the tensor fasciæ femoris, something seemed to give way in the joint. The child was aged 5 and was suffering from infantile paralysis affecting both lower limbs. Only very slight force was being used at the time.

X-ray photographs seem to show that there is both a fracture of the neck of the femur and a separation of the epiphysis of the hip. It is possible, however, that the latter condition is apparent only and that the real lesion is a simple fracture. The double injury, if present, must be very rare.

DISCUSSION.

The PRESIDENT: Injuries of the neck of the femur produced in this way are interesting events both from the point of view of the care that should be taken in the manipulation and from the point of view of treatment. In manipulating a hip in those cases in which the bones are poorly developed, fractures are very apt to occur. They often assist in correcting the deformity.

Mr. A. S. Blundell Bankart: It will be interesting to have this case watched and shown again at a later meeting, so that the progress of any deformity of the neck of the femur, such as coxa vara, may be noted.

Case of Chronic Synovitis of both Knee-joints.

By H. A. T. Fairbank, F.R.C.S.

The patient is a young soldier who has now had chronic swelling of both knees for two and a half years. Three years ago he had rheumatic fever in Egypt, many joints being affected. All, however, recovered by February, 1917, with the exception of the knees. These have remained swollen ever since, in spite of many varieties of treatment. He has never suffered from dysentery nor from any other disease of importance. The knees have been treated by periods of rest, by massage, gymnastic treatment, ionization, radiant heat, firm bandaging, graduated faradic treatment of the quadriceps and by four months' treatment at Bath. The Wassermann reaction is negative. A skiagram shows some lipping of the joint surfaces on the right side, but not on the left.

On September 9 both knees were aspirated; 5 c.c. of ether were injected into the right knee-joint. The fluid obtained from this was slightly turbid and sterile. The effusion returned soon after the aspiration.

I should like suggestions as to the treatment of this case. Two proposals have been made: (1) excision of a portion of the synovial membrane of the joint; (2) excision of the joint with a view to securing fixation.

DISCUSSION.

The President: I think that treatment similar to the firing of horses—that is light touching with a linear cautery—is worth trying. In my opinion the osteo-arthritic changes present are more important than the persistent hydrops.

Mr. W. R. Bristow: I have seen several cases of chronic synovitis of the knees in men who have returned from France with some illness—presumably some form of infection. All forms of treatment are disappointing, recurrence of the effusion usually occurring. Operation is almost certain to fail. I have not seen satisfactory results from excision of portions of the synovial membrane. Excision of the joint in such a case can only be justified when there is severe pain. I think the best course will be to apply a firm compression bandage and to allow the patient to get about.

Mr. A. S. BLUNDELL BANKART: The effect of excision of the synovial membrane can only be to produce a fibrous ankylosis. This could be produced in some other way—for example, by the injection of formalin and glycerine.

Mr. H. KELLER: It is advisable to repeat the Wassermann reaction after giving a provocative dose of salvarsan. I should treat the case with the latter drug in spite of a negative Wassermann.

Mr. G. R. GIRDLESTONE: Have the knees been immobilized for any considerable period? [Mr. FAIRBANK: Certainly, for periods of four weeks at a time.] Considering that the condition is undoubtedly an arthritis, I think complete immobilization for a long period such as six months, with treatment by ionization during this time, is quite worth trying, following this up by treating the thigh muscles.

Mr. ELMSLIE: I suggest that it might be worth while opening the joints and washing them out. It seems to me possible that the joints contain cartilaginous loose bodies, like melon-seed pods, the right one more probably than the left.

Mr. LAMING EVANS: It might be worth while opening the joint and turning back the edges of the synovial membrane in an attempt to drain the latter into the subcutaneous tissue.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Two Cases of Multiple Congenital Deformities.[1]

By H. A. T. Fairbank, F.R.C.S.

Case I.—The boy is aged 11. He has bilateral talipes equinovarus and dislocation of both hip-joints. In the upper limbs there is a posterior subluxation of the left shoulder-joint which is not reducible. The condition of this joint is very similar to that seen in the late stage of a birth palsy. The right elbow-joint has, across the flexor aspect, a bridge of skin stretching between the arm and forearm. Extension of this joint is limited. Both wrists are flexed and the fingers clawed, the metacarpo-phalangeal joints being extended and the interphalangeal joints flexed. Sensation is normal throughout. The limbs are very thin and the musculature feeble, but all the muscles react to a faradic current, except the extensors of the wrists. Although the condition of the left shoulder resembles that seen in birth palsy, it must probably be regarded as developmental, because of its association with so many true congenital deformities.

Case II.—This boy is aged 4 months. In the upper limbs the extension of the left elbow is limited, the left hand was originally in the position of dorsiflexion with ulnar deviation. In the right hand there was palmar flexion with slight ulnar deviation. These deformities have been to some extent corrected by treatment with splints. The left hip was dislocated, the left knee rigid and the patella absent. There was bilateral talipes equinovarus. The deformities of the hands and feet are yielding well to treatment.

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[1] At a meeting of the Section, held December 2, 1919.

Case of Multiple Congenital Deformities.

By R. C. ELMSLIE, M.S., F.R.C.S.

THE patient is a girl aged 5. The head, neck, trunk and abdomen show no deformity nor wasting. The upper limbs hang at the sides with elbows extended, forearms in a position of mid-pronation and wrists flexed, the whole limb being rotated in. The pectoral muscles appear well developed, but the deltoid and posterior scapular muscles are much wasted. The muscles of the upper arm are moderately developed, those of the forearm and intrinsics of the hand are very feeble. The wrist-joints are flexed to a right angle, the fingers being held fully extended. The right and left arms are very similar in appearance and in the mobility of the joints, but the left is slightly smaller and shorter than the right. Most active movements are possible through a small range. At the shoulder-joint abduction can be carried nearly to a right angle, strength being poor. Adduction is fairly strong, being the movement most used by the child. Rotation in and out are both present, but very feeble. The action of the serratus magnus appears to be normal. Extension of the elbow is present but weak. Flexion in the right arm can be carried out from the fully extended position to an angle of 35°. In the left elbow the movement can only be carried to 160°. These movements through this limited range are fairly strong. Pronation of the forearm is complete and fairly strong; supination weak and possible only through a small range. At the wrist-joint there is a small range of weak active extension. The flexor muscles appear, however, to be quite strong. The movements of the fingers are very limited, but extension, flexion, abduction and adduction of all the fingers appear to be present, being better in the right hand than in the left. The adductors of the thumbs work quite strongly; the child is in the habit of holding things between the thumb and the palm or between the index and middle fingers. The latter fingers are concave towards each other, probably as the result of this use. In the lower limbs there is a dorsal dislocation of both hips. The knees are held fully extended, and the right cannot be flexed at all; the left can be flexed voluntarily through a small angle. The feet show a talipes equinovarus partially corrected by operation. The muscles of the lower limbs are weak throughout—those on the right side being the worse. No knee-jerk can be obtained. Electrical examination was too difficult and painful to permit of completion. X-rays show a doubtful dislocation of the right shoulder and a dislocation of the head of the right radius forwards, in addition to the dislocation of the hips. The individual bones appear to be well formed and five carpal bones are present on either side.

The condition of the upper limbs in this case is one which I have now seen several times, and have been in the habit of calling "seal flapper" deformity. In the cases that I have seen the condition of the lower limbs has varied. In most there has been a congenital talipes—in some a dislocation of the hips and in some stiffness of the knees. In one case there was a spastic condition of the lower limbs. The deformities of the upper limbs form a sufficiently definite type to deserve differentiation under a specific name, although the pathology of the condition is apparently unknown.

Case of Sternomastoid Tumour in a Child born by Cæsarean Section.

By H. A. T. Fairbank, F.R.C.S.

This child, aged 4 months, presents a hard fibroid mass in the lower part of the right sternomastoid. There is at present no torticollis. Dr. Hubert Roberts has kindly furnished notes of the birth of the child. The mother was extremely ill with vomiting, convulsions, albuminuria, and finally coma. Cæsarean section was performed to save her life, and in this the operation was successful. The head was tightly engaged in the pelvis and considerable difficulty was experienced in extracting it. In Dr. Roberts's opinion the injury of the sternomastoid muscle most likely occurred in disengaging the head from the pelvis and not as the result of uterine contraction during the removal of the child.

DISCUSSION.

The President: The theory as to whether the sternomastoid tumour is of traumatic origin is still a matter of controversy. I have never heard that there could be so much obstruction to delivery by Cæsarean section as to cause an injury to a muscle.

Mr. Laming Evans: Is it not possible that this is a case of true intra-uterine wry-neck? The time appears too short to account for the contraction already present. It would be interesting if Mr. Fairbank would undertake to watch the case, and either to show it again or to report what degree of torticollis developed.

Mr. ROTH: I think it would be better to treat the case rather than to watch it. In my experience a sternomastoid tumour treated by gentle movements from the first recovers without any torticollis arising.

Mr. FAIRBANK (in reply): At the present time there is little, if any, shortening of the muscle. Treatment by movements is being carried out, and I hope that recovery will take place without a torticollis developing.

Case of Arthritis of the Knee with Persistent Flexion.

By E. LAMING EVANS, F.R.C.S.

THIS girl is aged 17. At the age of 2 she developed an arthritis of the right knee, in which flexion deformity was a prominent symptom. She has been treated in several hospitals by various methods, but the flexion remains. A recent slight injury necessitated rest in plaster of Paris in a general hospital. The knee is now flexed at an angle of 20°, the ankylosis being fibrous and a very small range of movement remaining.

X-ray examination shows that the epiphyseal cartilages are synostosed. In the external condyle of the femur there is a translucent area suggesting an old abscess. The joint line is hazy, and shows an obliquity downwards and inwards from collapse of the external condyle.

I think that conservative treatment by splinting has perhaps been carried out too long, and that excision of the joint with a view to obtaining bony ankylosis is desirable. An important point, however, is the possible existence of a focus of disease in the external condyle, which may necessitate the removal of enough bone to leave considerable shortening.

DISCUSSION.

The PRESIDENT: This is probably a case of caries sicca. I agree that an excision should be performed and the joint fixed in the extended position. It is interesting that the X-rays appear to show that the epiphyseal disks have disappeared.

Mr. Elmslie: Some years ago I had the opportunity of investigating the results of excision of the knee in children, both as shown in the literature and by investigation of about 100 children in the London County Council schools. In the latter the operation of excision has been carried out in various London hospitals by many different well-known surgeons. The results were extremely bad. Hofmeister laid down certain rules for excision of the knee. Under the age of 9 he considered it an unjustifiable operation; between 9 and 13 it should only be done as a means of saving the limb. After this age the operation is a good one. My observations led me to agree with these conclusions completely. In this case I think that excision should be performed, but it may be difficult because the focus of disease on the outer side may be opened in dividing the femur.

Case of Traumatic Myositis Ossificans.

By W. T. Gordon Pugh, F.R.C.S.

About eight weeks ago, the patient, a boy aged 15, whilst playing in a gymnasium, caught his foot in a mat and fell sideways on to his hand, dislocating the left elbow. This appears to have been completely reduced without an anæsthetic. Cold compresses were applied and the arm slung in a flexed position. About a fortnight later, the doctor finding that any attempt at movement caused pain, administered chloroform, moved the joint, and fixed the elbow in an extended position upon a splint. For a few days the elbow was flexed and extended several times each morning, but the movement possible became steadily less in extent and more painful.

When he was admitted to hospital a few days ago, the arm was fixed with the elbow within 40° of full extension, and the forearm in a position midway between pronation and supination. There was considerable swelling around the elbow, due chiefly to a firm thickening, extending from the olecranon upwards for about 3 in., and to a similar thickening in front of the joints.

X-ray examination shows considerable areas of opacity in positions corresponding to the triceps, brachialis anticus and supinator brevis.

The history is that usually associated with a traumatic myositis ossificans, and the treatment must evidently take the line of securing absolute rest, so as to prevent the development of additional new bone and to assist the absorption of that already present. In the presence of such extensive alterations from the normal, an ultimate stiffness of the joint seems not improbable, and it is therefore advisable to get the elbow into a standard position for fixation.

I should like to hear opinions as to whether this alteration of position should be carried out and, if so, whether this should be done at once under an anæsthetic or gradually by the use of a hinge splint. I should like also to hear the experience of others as to the final result in cases with such marked changes, and how long it will probably be before the patient is able actively to use his elbow?

DISCUSSION.

The PRESIDENT: I think it is improbable that the boy will ever regain a mobile elbow-joint. I should, therefore, alter the position immediately to that which is the most useful.

Mr. ELMSLIE: The case is a lesson against the employment of forced movements in the treatment of elbow injuries. This joint appears to resent such movements more than any other. I consider that there is a considerable chance of improvement in this case, because the new bone is light and casts little shadow. I should, therefore, fix the joint in its present position on the ground that any alteration would tend to increase the bone formation. If the joint remains stiff it will be quite possible to alter the position later on.

Mr. FAIRBANK: I agree with what Mr. Elmslie has said. The position of the arm should be left alone for the present. The presence of a shadow around the neck of the radius, probably representing ossification in the supinator brevis, is interesting and certainly very rare. I have an X-ray showing a shadow of a bone in this situation in a dislocation in which the ulna had been reduced, but not the radius. At any rate the head of the radius is still dislocated.

Mr. ROTH: I hope this case will be taken as an example of how not to treat an elbow. I think it would be better to flex the joint now, because it will take two to three years for the bone to absorb, and during that time the arm, if flexed more, will be in a more useful position. The slight movement of bending the joint should not cause much increase in bone formation.

Mr. LAMING EVANS: The case is of interest as one of myositis ossificans following a pure dislocation. I think the X-ray shows that there may possibly have been a separation of the epiphysis of the olecranon. In my opinion, it will be better to fix the elbow in its present position, both because any movement will increase the tendency to bone formation, and because tension upon the triceps may pull the epiphysis of the ulna further away from the shaft.

Mr. JOCELYN SWAN: I do not agree with Mr. Laming Evans that myositis ossificans after simple dislocation without fracture is especially rare. I have had two recent cases under my care in which ossification in the brachialis anticus has followed simple dislocation. In neither case was there any change in the supinator brevis. They were treated by rest and the ossification became reabsorbed.

Mr. A. S. BLUNDELL BANKART: I think the elbow should be flexed now, because the limb will be so much more useful during the period of immobilization. I hope Mr. Pugh will show this case again later on.

Recovery after Suture of Median and Ulnar Nerves in the Forearm.

By R. C. ELMSLIE, M.S., F.R.C.S.

MISS H. D., aged 16, cut her wrist by falling with it through a window on July 1, 1918. The tendons were sutured at the time at the Huntingdon Hospital and the ulnar artery tied but the nerves were not sutured.

She first came to me for treatment on September 9. There was a cruciform incision above the wrist, the transverse scar being about 3 in. above the joint (due to the injury); the longitudinal scar was that of the operation. All the long tendons were acting satisfactorily, but all the intrinsic muscles of the hand were completely paralysed; the hand was clawed, the metacarpo-phalangeal joints being hyperextended, the interphalangeal joints flexed. The anæsthesia to light touch included the whole of the palmar surface of the hand and the front of the ulnar side of the forearm as high as the transverse scar; also the back of the ulnar half of the hand, the whole of the dorsal surface of the little and ring fingers, the last two phalanges of the index and middle fingers, and the last phalanx of the thumb.

On September 10 the longitudinal scar was excised and the median and ulnar nerves were explored; the ends of the median nerve were found displaced laterally from each other by about $\frac{3}{4}$ in.; the ends of the ulnar nerve were continuous through a strand of fibrous tissue which was about $1\frac{1}{2}$ in. long. The nerve ends were freshened and sutured, the median meeting easily, the ulnar only with some tension when the wrist was flexed. After the operation the wrist was kept flexed for six weeks and this joint and the fingers were then slowly extended on a splint. To counteract the tendency to clawing of the hand and to produce a palmar arch an "egg" splint was used for some time.

Treatment by daily massage and galvanic stimulation of the intrinsic muscles was commenced as soon as the wound was healed, and as soon as voluntary power returned re-educational exercises were added. The forearm muscles were treated by faradic stimulation throughout.

At the present time there is an almost complete return of sensation to light touch in the whole of the affected area. All the intrinsic muscles respond to a faradic stimulation, and all show some voluntary power; the ulnar muscles recovered more slowly than the median, and the interrossei are still weak. The hand is now of a natural shape, there is a palmar arch and the fingers can be flexed at the metacarpo-phalangeal joints whilst the interphalangeal joints are extended. Opposition of the thumb is good. The circulation in the hand is now good. Throughout the winter of 1918-19 it was very bad, the fingers being constantly blue and shiny. The pads on the last phalanges are now returning.

Dupuytren's Contraction in a Man aged 31.

By Paul Bernard Roth, F.R.C.S.

The patient was wounded in January, 1916, with a rifle grenade splinter in the ring finger of the left hand, there being a small flesh wound. The wound was healed in a month, although there appears to have been a little suppuration. In the following December he began to feel something tight pulling down his finger, and it became bent towards the palm. This has gradually become worse. The ring finger is flexed at its metacarpo-phalangeal joint to 45°, the interphalangeal joints not being affected. There is a very prominent ridge in the palm, due to the thickened palmar fascia, which leads to the base of the ring finger, and to which the skin is adherent.

Note.—Both little fingers have been contracted ever since the patient can remember, but they have caused no inconvenience.

The points of interest in this case are the manner of causation, the rapid onset, the youth of the patient, and the treatment to be adopted. I think it possible that the organisms of suppuration gained entry to the prolongations of the fascia on the ring finger, and spread down it to the palm, setting up a septic fibrosis.

As to treatment, I prefer to adopt Adams's method, followed by bandaging the finger to a malleable iron splint in nearly complete extension.

DISCUSSION.

The PRESIDENT: I think that it is doubtful whether the injury really had anything to do with the origin of the contraction. Adams's operation is tedious and the results uncertain, the alternative would seem to be by an open operation.

Mr. MORTIMER WOOLF: I have recently seen two cases of Dupuytren's contraction following wounds, but I do not think that the wounds had anything to do with the contraction. In each case the man had been in the habit of using a chisel, and I think that the pressure in the palm is important. I have treated a case by very extensive excision of the fascia, but had a recurrence of the contraction in spite of this. I re-operated and transplanted a piece of fascia lata and fat from the thigh, with great improvement. The method of transplanting fat has recently been reported in the *Annals of Surgery*.

Mr. MAYNARD HEATH: I have recently seen a case in a boy aged 16, in whom the contraction followed a crush of the fingers and was apparently due to sepsis tracking down into the fascia. I think that treatment should be by excision of the fascia. I have treated a case by this method in which no recurrence took place after ten years.

Mr. LAMING EVANS: I have seen the condition in a younger man, the contraction following trauma, due to gripping the bottom of a boat after his ship had been torpedoed. I have always removed the fascia, and have found it necessary to open the flexor tendon sheaths in order to make sure of complete removal.

Mr. ELMSLIE: I agree with Mr. Laming Evans that the removal should be complete; I think that recurrences are often due to insufficiency of the initial operation. Although I cannot see the necessity for opening the tendon sheaths, I do not find that in my dissection they are as a rule exposed.

Mr. W. R. BRISTOW: Bad results are often due to insufficient after-treatment. Simple tenotomy followed by stretching and splinting gives good results. An extensive dissection often leaves a keloid scar.

The PRESIDENT: The use of thiocyanin, rubbed in after operation, has been advocated in order to prevent the formation of a keloid. As an example of the defects of an incomplete operation I may allude to the case of a

Roman Catholic, who preferred to have the operation done without an anæsthetic, because it would be good for his soul. The operation was hurried and a recurrence resulted.

Scoliosis of Uncertain Origin.

By P. Maynard Heath, M.S.

The patient is a girl, aged 10. She had a fall in the playground in 1918; the accident was apparently trivial, but no details are available. Very little notice was taken of it, and the child was not incapacitated. Some months later the mother noticed that the back was not straight.

In the erect position there is very little abnormal to be seen but when the spine is flexed the whole trunk is carried clearly to the left. At the same time rotation occurs, so that the right lumbar region becomes prominent, and a secondary curve, convex to the left, is produced in the dorsal region. Flexion of the spine directly forwards or to the right is impossible. There are no nervous symptoms and the condition is not progressive. The hip-joints and lower limbs are normal and there is no evidence of abscess formation. The deformity of the spine is obviously due to some lesion in the lower lumbar vertebræ.

X-rays show that the articular processes between the fourth and fifth lumbar vertebræ on the left side are apparently fused into a structureless mass of dense bone, and that the fourth lumbar vertebra is set obliquely upon the fifth. No fracture of the articular processes can be seen. There is a deficiency in the laminæ of the fifth lumbar vertebra and its left transverse process is longer than the right and comes into contact with, and is possibly joined to, the left lateral mass of the sacrum. The bodies of the vertebræ are normal in outline and no rarefaction of bone is visible. The X-ray appearances may be due to fracture, congenital abnormality, or caries. If the cleft in the laminæ of the fifth lumbar vertebra is due to fracture, it is possible that the articular processes between the fourth and fifth lumbar vertebræ have been fractured and have subsequently fused. The slightness of the accident is somewhat against this view. On the other hand the increase in size of the left side of the fifth lumbar vertebra is clearly developmental and suggests that the cleft in the laminæ of that vertebra and the condition of the affected articular processes are also developmental. Caries seems improbable in view of the symptoms and X-ray appearances.

The child has been treated for six weeks in a plaster jacket by another surgeon. Since then she has been given exercises and massage, with improvement in her comfort and general health, but without any influence on the lateral deviation of the spine.

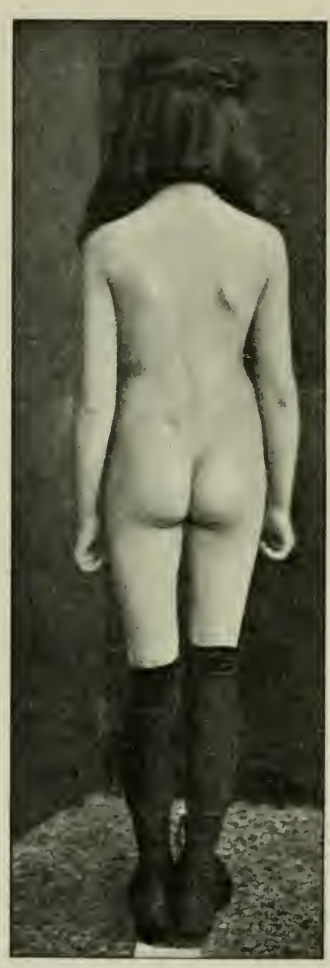

FIG. 1.

Position when erect.

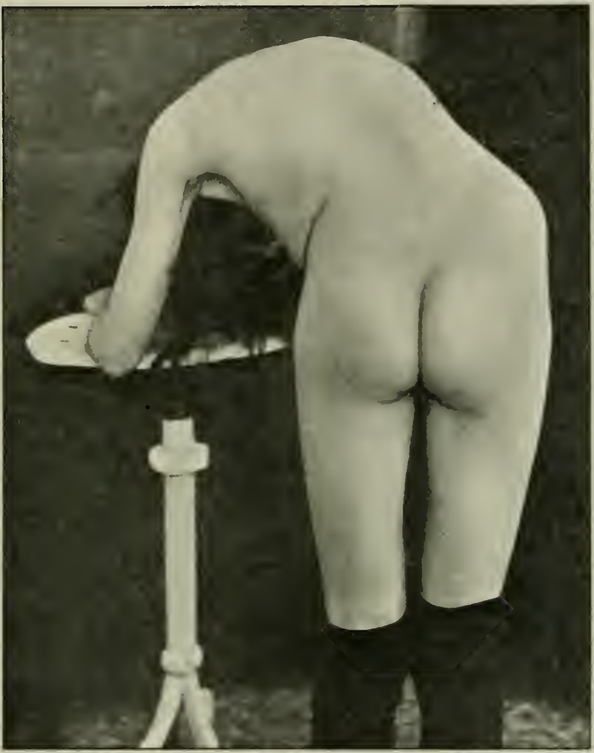

FIG. 2.

Position when attempting to bend directly forwards.

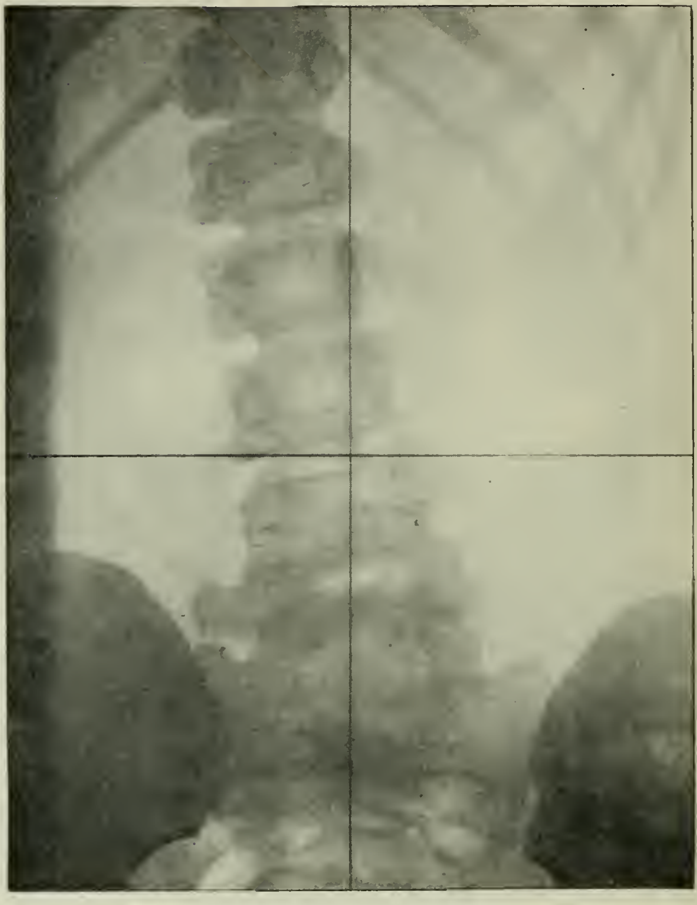

Fig. 3.

Section of Surgery.

President—Sir JOHN BLAND-SUTTON, F.R.C.S.

Congenital Diverticulum of the Colon.[1]

By GARNETT WRIGHT, F.R.C.S.

WHILE congenital diverticula not infrequently occur in the small intestine, similar conditions are extremely rare in connexion with the large bowel. The following case appears to me to be worthy of record as being an almost unique example of such a diverticulum :—

The patient, a married woman, aged 27, was admitted to the Salford Royal Hospital on September 20, 1917. She had suffered from constipation and abdominal pains ever since she could remember. The griping pains and the constipation had steadily increased until latterly life had become very miserable. For two or three days before admission constipation had been absolute and the patient had vomited once or twice.

On examination there was definite visible peristalsis to be seen, sometimes in the upper and sometimes in the lower abdomen. The bowels were moved on the 22nd and again on the 26th, but the griping pains and the visible peristalsis still continued.

On September 27 I opened the abdomen by a median laparotomy and found the whole of the large intestine greatly dilated, and the mesosigmoid occupied by a cystic swelling, which I thought at first was a mesenteric cyst. On further examination a longitudinal band was seen to bifurcate at the commencement of the sigmoid colon, and it was evident that a diverticulum was originating at this point, and was occupying the mesosigmoid. On tracing this onward it could be felt to leave the mesosigmoid at its lower end and turn upwards behind the posterior parietal peritoneum and end in a large sac on the posterior abdominal wall behind the stomach. It was evident that the diverticulum could not be removed without excising a large portion of the colon, and as the whole colon was in a condition of megacolon I decided to do a complete colectomy. Accordingly the terminal ileum was divided, the upper end closed and united by lateral anastomosis to the lower part of the ileopelvic colon. The colon was then resected and the lower end closed by invagination. The patient recovered well from the immediate effects of the operation. The temperature rose to 100·4° F. on the 28th and 100·6° F. on the 29th. It fell to 99° F. on the 30th and to 98° F. on October 3. Thereafter it remained slightly below normal until the evening of October 6, when it fell to 97° F. and remained at that level until just before death on October 8, when it rose to 101·8° F. The bowels were moved after an aperient on September 30, and there was an average of five daily motions after that. The pulse-rate fluctuated between 112 and 140. The patient appeared to be progressing fairly satisfactorily until the evening of October 6, when the temperature fell to 97° F., and she vomited. She continued to vomit on the 7th and the abdomen was reopened on the 8th, when it was found that suppuration had occurred in the anastomosis, which had given way. The patient died that evening.

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[1] At a meeting of the Section, held February 4, 1920.

DESCRIPTION OF SPECIMEN.

The specimen removed consisted of the termination of the ileum and the large intestine from the cæcum to the upper end of the rectum, together with the diverticulum to be described below. The colon removed was in a condition of moderate idiopathic dilatation, and in the recent state it measured 8 ft. in length.

The diverticulum, which when fresh was 37 in. long, commenced at the upper end of the sigmoid loop of the colon, running at an acute angle downwards and entering between the layers of the mesosigmoid. Here it occupied the concavity of the sigmoid colon and became of very large size, being very much greater in diameter than the bowel. On reaching the lower end of the sigmoid loop, the diverticulum separated from the colon, to which it was up to now intimately adherent, and narrowing abruptly it passed from between the layers of the mesosigmoid to lie behind the posterior parietal peritoneum. Here it turned upwards and expanded into a large blind sac, reaching a position on the posterior abdominal wall behind the stomach. In the recent state, the circumference of this portion of the diverticulum was 17 in. For purposes of description we can divide the diverticulum into three parts:—

(1) The first part is about 6 in. long, with sacculations and tænia such as we see in normal large intestine. This portion merges gradually into—

(2) The second portion, which lies between the leaves of the mesosigmoid. This is the longest part and is of wide diameter and smooth walled. For the first part of its course it is intimately blended with the sigmoid colon, but becomes separated from it below. It is joined on to the third portion by a very narrow neck.

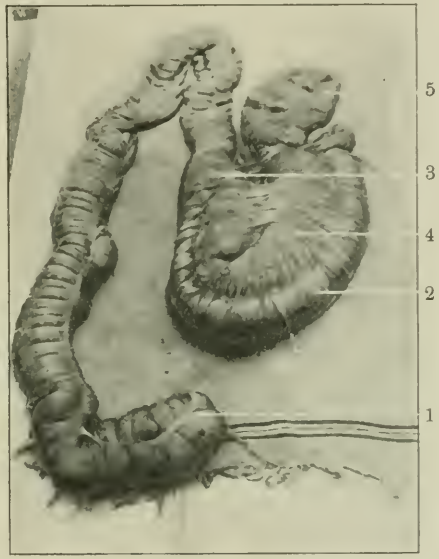

FIG. 1.

Portion of intestine removed at operation. Note vessels running over diverticulum to supply the sigmoid colon. 1, cæcum; 2, sigmoid colon; 3, origin of diverticulum; 4, diverticulum between layers of meso-sigmoid; 5, blind sac.

(3) The third part is a large blind sac, which was entirely retroperitoneal in position and ends at the upper extremity in a small button-like projection. Before the peritoneum was dissected away, the two limbs of the loop formed by the first and second parts of the diverticulum were bound together by strong bands of thickened peritoneum.

The connexion of the tænia with the diverticulum is very interesting. The *tænia libera* is continued from the descending colon on to the sigmoid, and does not reach the diverticulum at all.

The *tænia omentalis* bifurcates at the commencement of the diverticulum, one limb descending on to the sigmoid, and the other running off it at an obtuse angle to reach the anterior surface of the diverticulum. Here it gradually widens out, losing its band-like appearance and forming a fairly uniform longitudinal coat, though the main mass of the fibres can still be seen near the convexity of the diverticulum.

The *tænia mesenterica* is continued from the descending colon directly on to the posterior surface of the diverticulum and widens out in a similar manner to the band on the anterior surface. It does not have any connexion at all with the sigmoid colon.

Thus we find two bands on the sigmoid colon: (1) The *tænia libera*; (2) one limb of the bifurcated *tænia omentalis*—and two on the diverticulum—(i) the *tænia mesenterica*, (ii) the other limb of the bifurcated *tænia omentalis*.

The blood supply of the diverticulum was derived from the inferior mesenteric artery. This was of large size and divided into large branches which lay in the concavity of the diverticulum, forming rudimentary arches from which vessels ran transversely over the diverticulum to reach the colon, which they supplied in the usual way. Several irregular arterial arches were present on the surface of the diverticulum. The vessels of supply to the diverticulum entered (1) from the arches in the concavity, (2) in a row half-way across the diverticulum, (3) as a series of well-defined branches near the convexity.

The large blind sac was supplied in an irregular fashion by large branches from the vessels in the concavity of the diverticulum.

When the peritoneal coat is dissected away it is seen that the colon and the first and second parts of the diverticulum are intimately blended, it being impossible to separate them from each other without tearing muscle fibres. Muscular bands can be seen running from one to the other, and the septum between the two is common to both and consists of circular muscle fibres with mucous membrane on each side.

The fusion between the diverticulum and the colon ceases about 3 in. above the narrow neck between the second and third parts of the former, and the rest of the diverticulum is quite independent of the colon.

STRUCTURE OF DIVERTICULUM.

The first and second parts are covered with peritoneum except where they are fused with the colon, and in the concavity of the loop. The third part has no peritoneal coat, and in life occupied a position behind the posterior parietal peritoneum. The muscular coats are: (1) An outer longitudinal layer which in the first part is represented by the tæniæ described above; in the second part there is a more uniform outer coat, with no distinct bands; in the third part the outer coat consists of coarse muscular bundles running irregularly, but generally speaking in an oblique manner, and ending in a whorl at the button-like projection at the apex. (2) An inner layer of circular fibres.

A section taken where the colon and the diverticulum fuse together in a common septum (fig. 2) shows that the outer longitudinal coat gradually fades away, till in the mid-line of the septum only circular fibres can be distinguished.

The inner coat of the diverticulum consists of a mucous membrane thrown into irregular folds lined by epithelium of the usual large intestine type. It is interesting to observe that this epithelial lining is continued into the button-like projection in the apex of the blind sac of the diverticulum.

From the anatomical structure of this diverticulum it is evident that we are dealing with a congenital abnormality of the hind-gut. The blood supply, from the inferior mesenteric vessels, proves that the structure is a portion of the hind-gut, while nothing but a developmental error could explain the intimate connexion between it and the colon. How it arose is difficult to explain. Professor Keith has pointed out to me that in the Hyrax double colonic cæca are normally present, arising from the transverse colon, in addition to the usually placed cæca. In this connexion I would suggest that the arrangement of the longitudinal bands on the colon and diverticulum may throw some light on the development of this abnormality. The bands on the colon and the diverticulum combined represent the bands of a single primitive intestinal tube, since, although there are four in all, two on the colon and two on the diverticulum, two of these are derived from the bifurcation of a single tænia omentalis. It would appear that the original error in development has been in the form of longitudinal split in the primitive hind-gut, the septum being placed coronally, and running from the centre of the primitive tænia omentalis to the opposite side of the canals. If this is so, it would seem to me to be against the view that the diverticulum in this case is a throwback to the colonic cæca of the Hyrax, as these presumably arise by lateral buds from the intestinal canal. Lewis and Thyng [1] describe knob-like diverticula arising in connexion with the small intestine in embryos of the pig, rabbit, and

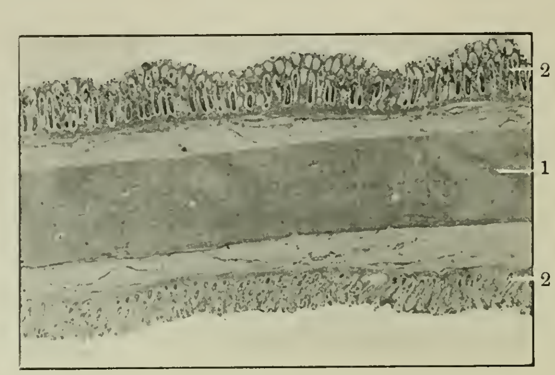

FIG. 2.

Septum between colon and diverticulum. 1, muscular coat of circular fibres only; 2, mucous membrane of colon and of diverticulum on each side of muscular coat.

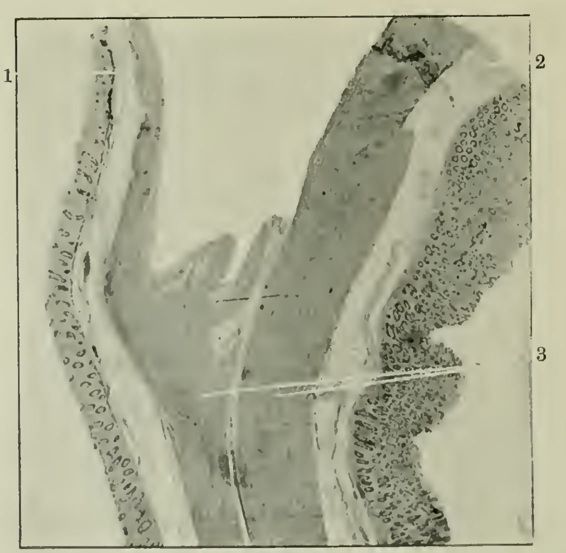

FIG. 3.

Transverse section at angle of junction between colon and diverticulum. 1, colon; 2, diverticulum (note hypertrophy as compared with colon); 3, circular muscular coats of colon and diverticulum fusing to form septum.

man, but, with one exception of a diverticulum arising near the ileo-cæcal valve, they found none in the large intestine. These diverticula disappear as development proceeds, but Lewis and Thyng suggest that they may occasionally persist and be the source of diverticula. It is difficult, however, to conceive that a diverticulum of this size in the large intestine could arise in such a manner. I confess that I can arrive at no satisfactory explanation of the phenomenon.

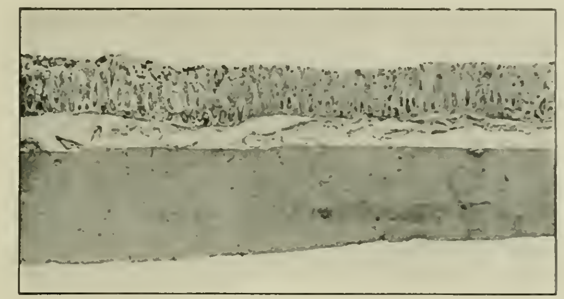

Fig. 4.

Section of wall of second part of diverticulum; moderate degree of hypertrophy.

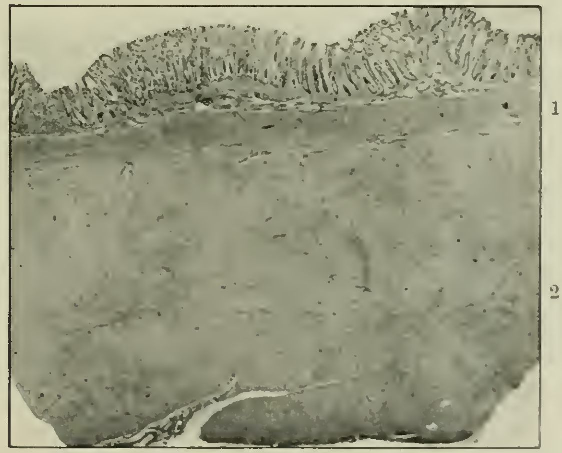

Fig. 5.

Section of wall of third part of diverticulum; extreme hypertrophy. 1, inner coat of circular muscle fibres; 2, outer coat of muscular bundles running obliquely.

I have been unable to find more than one or two cases in literature of a similar nature. Meckel [2] mentions a case of a fœtus with two cæca described by Boerhaave in 1757. Grawitz [3] describes the case of a man aged 55 who died of peritonitis due to intestinal obstruction, in whom a right inguinal hernial sac was occupied by a diverticulum arising from the middle of the ascending colon. It consisted of a double loop of bowel, opening by a common opening into the ascending colon. Its length was 20 cm., circumference at the neck 8 cm. and at the fundus 13·5 cm. The two loops of bowel were separated at the fundus, but were fused near the neck of the diverticulum, having an imperfect septum of mucous membrane only between them at this point. There was also a transverse occlusion in the loop of the diverticulum. Grawitz considered that this diverticulum might have been formed from a loop of bowel being caught in the hernial sac, and from a secondary fusion and breaking through of the lumen at the site on constriction. Lockwood [4] described a case of double descending colon where the lower ends of both limbs were lost in a mass of carcinoma, but gives no anatomical details. Hedinger [5] described a post-mortem examination on a newly-born child in whom the distal third of the appendix was thickened and nodular, owing to numerous small diverticula of the mucosa, some of which reached the muscularis mucosæ only, while others extended through it and produced projections of the serous coat. He considered that these diverticula were congenital.

REFERENCES.

[1] Lewis and Thyng. *Amer. Journ. of Anat.*, 1907-08, vii, pp. 505-519.
[2] Meckel. *Tabulæ Anatomico-Pathologicæ*, 1822, Fasc. 3, Tab. XXIII, fig. IV, p. 13.
[3] Grawitz. *Virchow's Archiv*, 1876, lxviii, p. 506.
[4] Lockwood. *Brit. Med. Journ.*, 1882, ii, p. 574.
[5] Hedinger. *Virchow's Archiv*, 1904, clxxviii, pp. 24-43.

DISCUSSION.

Professor A. Keith, F.R.S.: I agree with Mr. Garnett Wright in regarding the abnormality as being the result of a developmental duplication or splitting of the sigmoid colon. Splitting or dichotomy of the buccal of the rectal ends of the alimentary tract is known to occur, but there has been no clear recognition that a developmental cleavage can affect a limited and intermediate tract such as the transverse colon or sigmoid. Mr. Wright's specimen may be regarded as an example of this exceedingly uncommon type of malformation. Various forms of cleavage or septal formation are known to occur during the development of the alimentary tract of the embryo. The foregut is cleft horizontally or coronally, so that the ventral compartment becomes the trachea, while the dorsal part forms the first part of the œsophagus. Cleavage may occur abnormally so that the first part of the œsophagus ends blindly while the second part springs from the bifurcation of the trachea. Such is not an uncommon congenital abnormality of the œsophagus. The second part of the duodenum may be the seat of another kind of septal malformation—the septum lying transversely across and completely occluding the lumen of the bowel. Another type is represented by a specimen in the Museum of the Royal College of Surgeons. In this specimen a piece of small bowel from an ox has set side by side with it a long sausage-shaped cyst, 17 in. in length, exactly similar to the small bowel in structure, save that it is lined by stratified epithelium. In this case the cleavage of the bowel has apparently taken place in a sagittal plane, for both bowel and cyst lie side by side and are suspended at the free margin of the common mesentery. In Mr. Garnett Wright's specimen the cleavage has been in a coronal plane, the primitive sigmoid being split so as to form a dorsal or abnormal sigmoid and a ventral or normal sigmoid colon. Thus we see that abnormal cleavage may affect several parts of the alimentary tract of the embryo and may take place in a sagittal coronal or transverse plane. It must be remembered that a mere point of adhesion between opposite walls of the embryonic gut will, when the embryonic gut is drawn out to form the adult bowel, become a long septum or cleft. In trying to explain such a malformation as has been shown by Mr. Wright it is vain to seek light from comparative anatomy. In no animal whatsoever is duplication of the sigmoid a normal occurrence. The abnormal parasigmoid of Mr. Wright's case shows a remarkable condition of hypertrophy. The muscular coats have evidently striven to force the fæcal contents out at the cæcal end of the parasigmoid—at least the degree of hypertrophy increases as one passes from the open mouth to the closed end of the abnormal bowel. At the open or proximal end of the parasigmoid the mucous or glandular coat measures 0·8 mm. in thickness; in the neighbouring part of the normal colon this coat has a thickness of 0·25 mm. At the same sites the muscular coats measure 0·95 mm. (0·8 mm. circular coat, 0·15 mm. longitudinal) in the first, and 0·26 mm. (0·2 mm. circular coat, 0·06 mm. longitudinal) in the second. At the distal or cæcal end of the abnormal sigmoid the mucous membrane measures 0·6 mm., and is provided with numerous solitary follicles. The muscular coats at this point measure 3·7 mm.—fourteen times the normal thickness. The inner or circular coat measures 2·2 mm., the outer or longitudinal 1·5 mm. The mucous coat is bound rather firmly to the muscular coat, the submucous tissue being densely packed and much reduced in amount. At the cæcal end the muscularis mucosæ is 0·14 mm. in thickness—almost as thick as the outer muscular coats of a normal sigmoid. The inner circular and outer longitudinal coats of the muscularis mucosæ can be clearly distinguished. Throughout the greater length of the parasigmoid there is hardly more than a trace of Auerbach's (myenteric) plexus. This is particularly the case where the muscular hypertrophy is greatest. We know that addition of new muscular fibres in the embryonic gut takes place from the stratum in which Auerbach's plexus lies. It is a remarkable fact that with such muscular hypertrophy as is seen in the distal half of the abnormal parasigmoid, Auerbach's plexus should have almost disappeared. Very large isolated ganglion cells occur at various sites in both circular and longitudinal coats.

The PRESIDENT: On reading Mr. Wright's account of this rare diverticulum I at once suspected that it was associated with malformation of the colon. The suspicion was converted into a certainty on my examining the specimen. It is an example of doubling of the large intestine. The enormous size and thickness of the affected section are explained by the fact that it has served for some years as a reservoir of fæcal material much in the same way that a section of large intestine serves for the retention and accumulation of a retrograde flow of fæces after it has been put out of action by an ileo-colic anastomosis. The portion of the colon thus put out of action in many cases becomes enormously distended and its walls thickened. The distension and thickening are only equalled by the alterations that take place in the stomach when the pylorus is the seat of chronic, non-cancerous obstruction. Doubling of the large intestine is a rare malformation. An accessible record is to be found in the *Glasgow Medical Journal*, 1912.[1] It is carefully described by Mr. A. Blair Aitken, and it is illustrated by good figures. The transverse section of the pelvic colon resembles the barrels of a double-barrelled gun. Roux de Brignolles[2] discovered a double pelvic colon whilst operating for appendicitis.

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[1] *Glasg. Med. Journ.*, 1912, n.s., lxxviii, p. 131.

[2] *Ann. internat. de Chir. gastro-intest.*, 1914, viii, pp. 19-23. I have been unable to obtain this journal.

Appendicectomy by a New Route, and Simplified Procedure.

By R. H. Anglin Whitelocke, M.D., F.R.C.S.

The operation here described is designed for the removal of the appendix vermiformis through the right iliac fossa in cases where no general exploration of the abdominal cavity is called for—i.e., in acute rather than in chronic cases. The method is a muscle-splitting one and has proved so successful that the author employs it in preference to all others. Indeed its advantages—anatomical, surgical and clinical—appear sufficient to justify its general adoption in suitable cases as a substitute for the procedures in general use.

For the sake of clearness the operation may be described under the following headings:—

(I) Incision and approach.
(II) Treatment of the appendix.
(III) Advantages and drawbacks of the method.
(IV) Conclusions.

(I) Incision and Approach.

The incision used for opening the abdomen may from its anatomical situation be described as the *para-ilio-spino-inguinal* (or *ilio-inguinal* for brevity's sake), and is made ½ in.—or less—internal or medial to, and and parallel with, the anterior superior spine of the ilium, *or as near to it as practicable*. The anterior iliac spine, a constant and definite landmark, which is easily felt or demonstrated, however obese the subject, forms the centre. The cut is carried above and below this point to an equal extent and parallel with the iliac crest and spine, and the attached inguinal (Poupart's) ligament (fig. 1).

The length of the incision varies with the size of the individual, but usually need not exceed 2½ in., although it may be extended in either or both directions, if required.

The cut at once divides the skin, subcutaneous fascia and aponeurosis of the external oblique muscle to its full length, when an assistant, armed with a pair of blunt rectangular retractors (fig. 2), pulls and holds the edges apart, whilst the muscular fibres of the internal oblique muscle are defined and exposed to view. At this stage a white line of varying distinctness may usually be seen passing across the muscle almost horizontally, in a direction from the anterior superior spine of the ilium to the middle line of the body (roughly in about 68 per cent. of cases noted); this line, which is bloodless, indicates a natural division of the muscle into an upper and lower section. With this line as a guide (the same direction must be followed when no line is visible) the

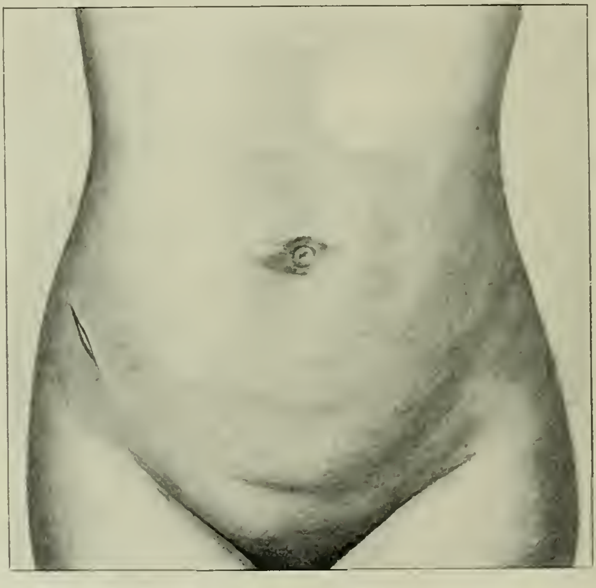

Fig. 1.

thick fibres of the muscle are incised and then separated by the aid of a blunt instrument such as a Kocher's director, or by the gloved index finger (fig. 3). The subjacent transversalis muscle is then similarly treated, its fibres being separated at the same time and by the same manœuvre to the full extent allowed by the skin incision. As soon as the fibres of these horizontal muscles have been freely separated and any bleeding staunched, the retractors are removed and replaced at right angles to their former position, in such a way as to pull and hold the fibres apart, thus exposing the transversalis fascia (fig. 4). This fascia, usually thin, may at times be somewhat thickened and resistant, and through it the exploring finger ascertains the precise condition of the subjacent peritoneum. Up to this stage the procedure is identical for all cases of appendicitis, whether acute or chronic, simple or "interval," with or without abscess. In uncomplicated cases and whenever there is no evidence of suppuration, as may usually be determined by palpation of the peritoneum through the transversalis fascia, this fascia together with the peritoneum are seized with blunt-pointed catch-forceps and divided in a direction parallel with the skin incision, medial or internal to the reflection of the parietal peritoneum on to the iliac fossa, deep to and parallel with the inguinal (Poupart's ligament).

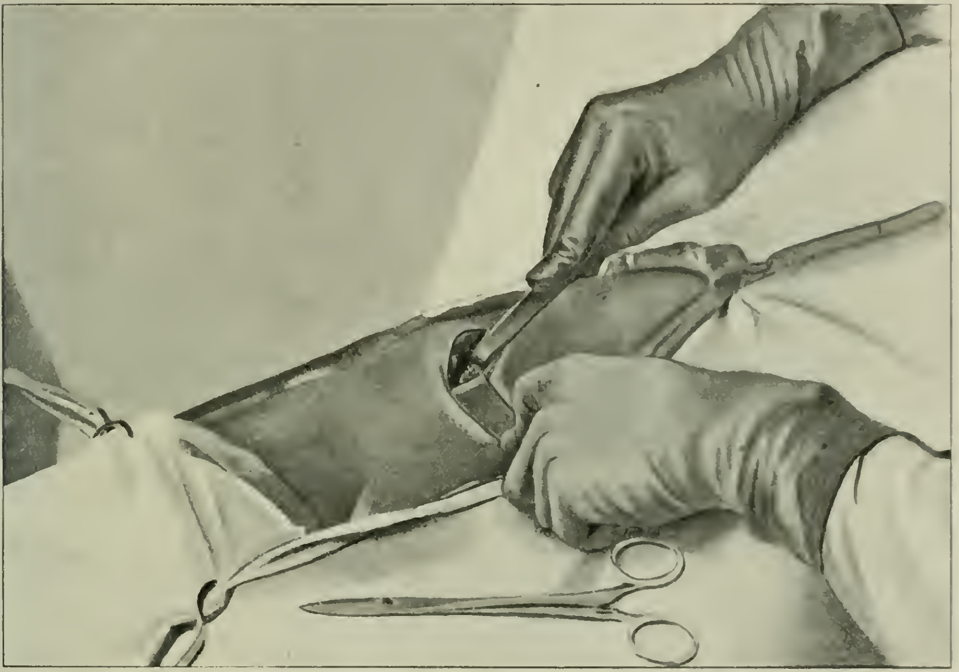

Fig. 2.

Where, however, an abscess is present or suspected, it is safer to turn the peritoneum inwards or medialwards from its seat of reflexion behind Poupart's ligament and to open it *posteriorly* from the iliac fossa. The transversalis fascia and peritoneum are usually divided together and in a line parallel with the skin incision, but they may be cut transversely as with the deep muscles, in case the incision is likely to require enlargement. The peritoneal opening should be made as free as the skin and muscle-divisions allow, and the edges caught and securely held apart with blunt-pointed catch-forceps (fig. 5). These instruments are attached near to the extremities of each edge, and serve to retain the cut edges of the peritoneum, acting as retractors during the subsequent manipulations. The forceps should be *blunt*, lest they wound the bowel; moreover, unless the peritoneal opening is ample, there may be difficulty in replacing the viscus at a later stage. When the abdomen is opened, the viscus first seen is generally (86 per cent. of cases noted) a division of the large intestine, either the first part of the ascending colon or less frequently the cæcum. Except in children and in abscess cases the omentum or transverse colon are

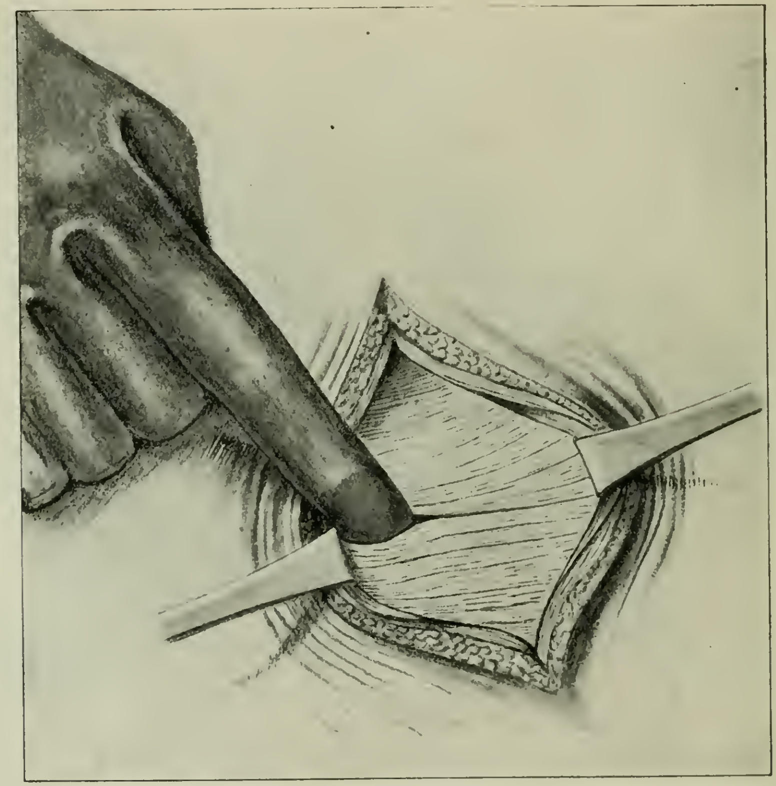

Fig. 3.

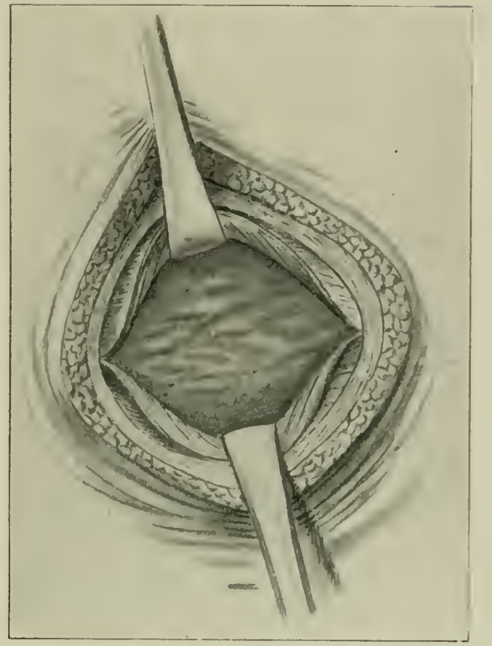

Fig. 4.

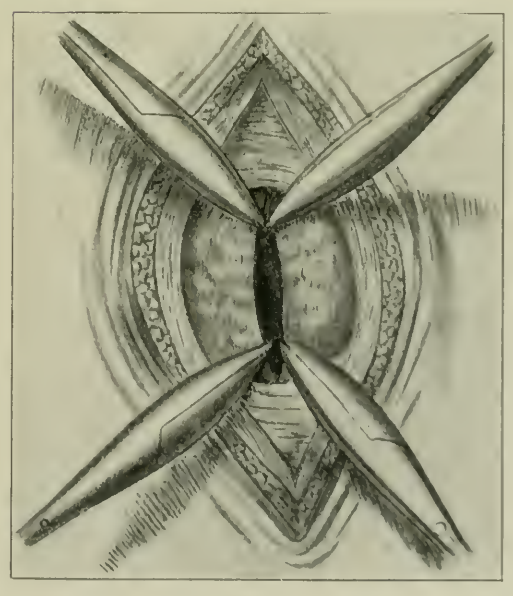

Fig. 5.

rarely seen, still less often is the small gut observed, except in cases of excessive intestinal paralysis and distension due to a generalized septic peritonitis.

(II) TREATMENT OF THE APPENDIX.

The large intestine is now seized and drawn gently upwards through the wound, its anterior muscular band serving as a guide until the cæcum with its appendix is delivered through the wound. When the appendix happens to be retrocæcal—a condition more frequently found than is reported—and is therefore sessile, or is otherwise adherent so that it cannot be delivered complete through the opening without risk or injury, it is wiser to detach the cæcum at once, to deliver it through the wound and to leave the appendix *in situ* to be dealt with afterwards.

In a simple or "interval" case, the appendix is removed outside the abdomen by clamping and dividing its mesentery up to its root. The mesentery is then ligatured, the appendix crushed at its base with a clamp, ligatured with catgut and divided with a sharp knife at a point distal to the ligature but proximal to a pair of forceps so placed as to prevent any escape of contents. The stump is now carefully disinfected with pure phenol and buried between the peritoneal folds of the cæcum and small intestine or invaginated and stitched into the cæcum by means of two superimposed layers of Lembert's catgut sutures. The cæcum is then cleansed with hot saline swabs and rapidly returned to the abdomen.

Some difficulty is at times experienced at this stage in returning the bowel through the small opening, especially in prolonged operations and in cases with a large movable cæcum, although the difficulty may be readily prevented or overcome if the following details are attended to :—

(1) The opening through the peritoneum should always *be as free or freer* than that through the muscles.

(2) The exposed and delivered bowel must be kept warm by gauze or lint wrappings soaked in hot saline lotion, whenever the manipulations are likely to be prolonged.

(3) The edges of the wound must be freely held apart by an assistant, whilst the operator is engaged in returning the cæcum.

The wound is closed in layers with catgut sutures (fine for the peritoneum, stout for the muscles), the skin being brought together with silkworm ligatures, these being *interrupted* so as to allow of the escape of serum. Only a few sutures are required, since there is but little strain on the edges, and the muscles tend to fall together of their own accord.

When the appendix is so adherent that it cannot readily be delivered with the cæcum through the wound, as for instance in cases of chronic abscess, the safest and easiest plan is to deal with it as a foreign body by detaching the cæcum from its root, and completing the operation by burying the stump as already described. The adherent and detached appendix is then seized at its divided end with the fingers or forceps and gently pulled upon until it comes away.

In many cases in which the inflamed viscus lies in retrocæcal or retroperitoneal situations, the muscular and mucous portions of the organ may be readily withdrawn from its inflamed and thickened peritoneal sheath, a firm rubber tube of sufficient calibre being then inserted into the track. When the appendix is approached by this route and its base sought for at the junction of the large and small intestines, it can *always* be found and removed. Only once out of a series of over 800 consecutive operations was there any insuperable difficulty in discovering the appendix, and in this case, after the ingestion of a barium meal, the Röntgen-rays showed a transposition of the viscera.

Drainage is now seldom used except in cases of abscess. The practice of removing the appendix as a routine, and with it the focus of sepsis and infection, is a sound one, for not only does it minimize the immediate risks and any tendency to relapse, but it shortens the period of drainage and, indeed, usually enables drainage to be dispensed with altogether.

(III) ADVANTAGES AND DISADVANTAGES OF THE NEW METHOD.

(A) Advantages.

Since the technique described above differs but little from that in general use in muscle-splitting operations, the question may be asked: What are the special advantages of this route of approach? The advantages are (*a*) anatomical, (*b*) surgical, and (*c*) clinical.

(*a*) *Anatomical Advantages.*—The anterior superior spine of the ilium is an unfailing landmark which can readily be felt in all circumstances of life. An incision or scar running through the external oblique muscle within half an inch of, and parallel with, its bony origin is practically free from strain, since it lies in a natural groove or fold of the body. The deep muscles—internal oblique and transversalis—are divided where their fibres are thickest and strongest, and more capable of bearing strain, i.e., along the line which indicates their natural division into an upper and lower section. As a rule no blood-vessels are present to require ligature, nor is any muscle-nerve liable to be divided. Moreover, the parietal peritoneum is thick and taut, and well supported by extraperitoneal fat at its line of reflexion to the iliac fossa, hence it is less likely to approach the skin wound and so predispose to hernia, even after prolonged drainage. Such approximation of the peritoneum towards the skin through the muscles is perhaps the most important predisposing cause of ventral or other forms of hernia. A further advantage is that the ascending colon or cæcum is situated in the direct route, so that the surgeon may rely on discovering the appendix at its *origin from the viscus, no matter where its body lies* (except in the rare event of visceral transposition). There is probably no viscus which varies more than the appendix in its position and movements under the ever-changing circumstances of life. Hence an assurance that the appendix will be found in almost all conditions is a great recommendation of the procedure here advocated. If the large bowel is traced downwards or drawn upwards through the wound, with the anterior muscular band as a visible guide, until its junction with the small intestine is reached, the *root* of the appendix may be found, while if it is covered by peritoneum it may be felt and readily dealt with.

(*b*) *Surgical Advantages.*—The iliospinal approach to a great extent prevents the risk of ventral hernia, since the two main factors concerned in the process—pouching of the parietal peritoneum and muscle-strain—are almost wholly eliminated or rendered improbable by the anatomical facts already described. In the oblique incision of McBurney, on the other hand, the scar from its position is exposed to considerable intra-abdominal strain, and as a result the peritoneum often pouches; in fact, in cases of prolonged drainage the peritoneum often approaches the skin through the weakened muscles and may even become adherent to it. As stated before, both these circumstances seriously predispose to ventral hernia. Further, the not infrequent division of, or injury to, the twelfth dorsal nerve, supplying the region of the internal inguinal ring, and the ligature of the deep epigastric vessels predispose to inguinal hernia. Even more important than the prevention of hernia is the fact that the ilio-inguinal route obviates the necessity of *involving the general peritoneal cavity* while dealing with the appendix. Such a violation defeats Nature's efforts at self-preservation or cure, efforts which are revealed by the tendency of inflammatory processes within the abdomen to become limited and shut off by protective lymph. Any procedure, therefore, which counteracts this natural and conservative tendency must be condemned as un-surgical. This criticism is applicable to most of the operations practised to-day in which the appendix is approached through the *general peritoneal cavity*, whether the organ is healthy, inflamed, or even gangrenous; especially is this true of the straight or paramedian incision, to a less extent of the oblique. By the iliospinal route, on the other hand, the peritoneum may be opened and the abscess easily reached and evacuated through the iliac fossa without the difficulties and dangers of soiling or infecting the general peritoneal cavity. For this reason the new route now used is unquestionably safer, and in harmony with the soundest principles of surgery. Another advantage may be found in the possibility of almost always removing the appendix *at the time as a primary undertaking*. The necessity for a subsequent operation is thus obviated, with its concurrent risks, waste of time, increased expenditure and worry, apart from the risks of hernia. When *drainage* is established through the iliac fossa down to the stump of the appendix, or even into the pelvis itself, it is simpler and safer than when an abscess is drained through the general peritoneal cavity: an excluded fæcal concretion may also be more readily found and safely extracted. Again, the procedure as a whole is simpler and takes less time; in uncomplicated cases the saving of time is remarkable. The appendix is so quickly found and the wound requires so few sutures that more than once four such operations have been performed at the Radcliffe Infirmary within sixty minutes, all the details, including dressings and bandages, being carried out by the operator. Indeed, few abdominal operations will be found as simple. No doubt speed may matter little in an ordinary operation. On the other hand, it may be of the first importance when a patient is *in extremis*, cyanosed or ashy white in colour, with an irregular or flickering pulse, and severe meteorism, where even the anæsthetic adds appreciably to the danger. Simplicity of method and rapidity of execution are of priceless value when life hangs by a thread. Further, the ilio-inguinal route of approach, whether for exploration or removal, enables the surgeon to make a fair estimate of the gravity of the case before the peritoneum is opened. When the transversalis fascia is reached, digital palpation makes it possible to determine not only the presence but the precise situation of an abscess. The extraperitoneal fat (in cases of abscess) is often considerably thickened and œdematous, and offers distinct resistance even in the early stages; hence, if the fingers are pressed gently through the fat against the peritoneum, the resistance encountered enables the surgeon to form some idea as to whether the abscess is immediately subjacent or has passed upwards, behind, or to the outer side of the colon, or downwards and forwards into the pelvis. When the resistance is slight the peritonitis is almost certainly localized, and the peritoneum may be incised *anteriorly* and internally or medially to its anatomical line of reflexion, so that the general cavity can be opened with but little risk. On the other hand, if the resistance is such as to justify the suspicion of abscess, it is safer to turn the peritoneal fold inwards and forwards, to open the abscess *from behind* through the iliac fossa, and then to deal with the appendix or its concretion, and to establish drainage through the opening.

(*c*) *Clinical Advantages.*—The principal clinical gain lies in the lessening of the period of convalescence. Prolonged drainage is seldom needed, since the actual source of disease is as a rule removed at the time, and consequently recovery is usually rapid. When no drainage is necessary the patient may be allowed out of bed as soon as the wound is quite healed (usually from the tenth to twelfth day), and is able to walk about soon after.

(*B*) *Disadvantages of the New Method.*

What are the drawbacks to the procedure advocated above?

First.—One possible objection is that by this route no satisfactory exploration of the contents of the abdominal cavity can be made. This objection, however, is scarcely to the point, since, as stated before, the operation is recommended for the *removal of the appendix*, and not for general exploration. Laparotomy and exploration of the abdomen may be superadded by means of another incision, if required, and without detriment.

Secondly, it may be urged that, when the mischief in the appendix is insufficient to account entirely for the symptoms, the return of the cæcum after removal of the appendix may be difficult through the small peritoneal opening. This objection is valid, but if care is taken to make a free opening through the peritoneum to begin with, if the edges are well retracted, and if the exposed bowel is kept warm, the difficulty will rarely be formidable and never insuperable.

Thirdly, the provision of adequate drainage is felt to be a difficulty by those surgeons who believe that gravity plays an important rôle in abdominal drainage. Experience, however, proves that an appendix abscess, even in Douglas's pouch, may be quite as efficiently drained through the iliac fossa as through the central abdomen, provided that a sufficiently rigid though pliable rubber tube is used. It is not suggested, however, that every case need be drained in this way, since treatment must always be adapted to existing circumstances, and it is quite possible that in a few cases it may be advantageous to supplement it by another incision.

(IV) CONCLUSIONS.

The value of the operation above described is attested by a wide experience, having been practised by the writer for over ten years; during the last eight years it has been used almost *exclusively*. The procedure was at first reserved for simple, quiescent, or "interval" cases, the oblique muscle-splitting incision of McBurney being substituted whenever an abscess was suspected or evident. With increasing experience, however, the special method was found to be suited for almost every condition and adaptable to almost every emergency. Out of 843[1] consecutive operations performed at all stages of the disease, whether simple or complicated, and at all ages ranging from 3 to 79 in both sexes, there have been in all eighteen fatal cases, yielding a mortality of approximately 2·25 per cent. In a recent sequence of 117 cases there was no death, whilst the 118th and 119th were unsuccessful. The immediate mortality would appear, therefore, to be no higher than that pertaining to any of the other modes of procedure, while as regards remote sequelæ not a single case of hernia nor of abnormally weak scar has occurred. These results amply justify an appeal that the suggested procedure should receive at least a trial. In fact, now that it is becoming the rule to operate in almost every acute case as soon as the diagnosis is established and the necessary arrangements can be made, there is every reason to anticipate a fall in the immediate mortality, while the necessity for prolonged drainage and convalescence will be so lessened that any permanent abdominal weakness will be rarely met with.

The results, immediate as well as remote, obtained from any particular method of operation must, however, depend upon clinical details, as well as on operative technique. Did space allow a full account of the post-operative treatment and clinical details would have been added. It is thought advisable, however, to record these later in a special article.

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[1] Since this paper was written fifty-one additional operations have been performed with one death.

DISCUSSION.

Mr. R. P. Rowlands: For definite cases of acute appendicitis I always use a gridiron incision placed far out, but not so far out or so small as recommended by Mr. Whitelocke, believing that this incision is more satisfactory than any incision placed further in. I agree with Mr. Whitelocke that hernia is extremely unlikely to follow his type of incision. It is specially suitable for acute appendicitis with abscess when drainage may have to be adopted. I always carry the incision in the deep muscles inwards for 1 in. into the rectus sheath and thus obtain plenty of room. With this incision I have never failed to find and remove the appendix in many hundreds of cases, and, if more room is required the deep part of the rectus sheath in front of the muscle can be slit downwards or upwards for several inches without weakening the abdominal wall or dividing any nerves. After thus enlarging the wound downwards, I have been able to remove fairly large tumours from the pelvis, and there is no difficulty in cleaning out the pelvis when peritonitis is present. I agree with Mr. Whitelocke that another advantage of the gridiron incision is that the patient can be got out of bed early without any risk of hernia. He says that he gets his patients up on the tenth day. I invariably get them up on the fifth day and make them walk about a little more every day. I have not seen any disadvantages of this method, and I think it is far better to get these patients up before the period of thrombosis and risk of pulmonary embolism sets in. In a large number of cases at Guy's Hospital pulmonary embolism nearly always occurred between the ninth and seventeenth day, except in some instances of large pelvic tumours with pre-operative clots in which embolism may occur earlier and often apart from an operation. By getting patients up early and keeping them stirring I believe that clotting in the large veins, and especially in the right auricular appendix of the heart, is often prevented, with the result that pulmonary embolism becomes a rare event. Moreover, the recovery of the patient is not only quicker but more complete.

Mr. Herbert Paterson: I think that the small extent of the incision is a serious drawback. With a small incision it would not be possible to deal adequately with the conditions found in all cases. Personally, in operations for appendicitis I prefer the middle line, which gives good access to the appendix, makes it possible to deal satisfactorily with any unexpected complications, and gives a sounder scar than a side one. *In medio tutissimus.*

Section of Surgery.

President—Sir JOHN BLAND-SUTTON, F.R.C.S.

DISCUSSION ON THE REMOTE RESULTS OF THE SURGICAL TREATMENT OF GASTRIC AND DUODENAL ULCERS.[1]

Mr. HERBERT J. PATERSON.

THE remote results of the surgical treatment of gastric and duodenal ulcers may be considered from three points of view :—

(I) Clinical, or the result of the operation as regards the patient.

(II) Physiological, or the result of the operation as regards the secretions of the stomach and the metabolism of the body.

(III) Pathological, or the influence of operation on the healing of the ulcer, and the complications which may ensue from surgical treatment.

I propose very briefly to consider these points in the light of my own experience of the operation of gastro-jejunostomy, as this operation is, I think, still our sheet-anchor in the treatment of gastric and duodenal ulcers. My experience of partial resection for simple ulcer is limited to five cases, in which I did a resection in the belief that the disease was malignant.

(I) CLINICAL.

The conclusions herein are based on my first 450 cases, all performed over seven years ago. Of these sixteen died as the result of the operation, and four died later, of gastric cancer. In these four cases I was probably mistaken in considering the condition due to simple ulcer.

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[1] At a meeting of the Section, held March 3, 1920.

An analysis of this series gives the following results: Fifty-one of the patients have died of other diseases, having lived more than two years after the operation without any recurrence of their gastric disorder; 270 are alive and quite well. The number of those who are dead may seem large, but the series dates from twenty years ago. (One patient, operated on in 1901, is now 81 years of age.) Adding these two classes together gives a percentage of 80 who may be regarded as cured by the operation. In 9 per cent. the result may be classified as fairly good—that is, the patients have been relieved of their pain, but either suffer from occasional attacks of indigestion or have to exercise care in their diet. In 7 per cent. of the cases the result of the operation may be classified as bad: in four cases a jejunal ulcer occurred, in one there was a recurrence of a duodenal ulcer, and twenty-three cases were complete failures. In some of these cases this result was probably due to leaving in the abdomen a diseased appendix in the days before the rôle of the appendix in producing gastric symptoms was appreciated.

There is a tendency at the present time to decry the value of gastro-jejunostomy. Considering that 89 per cent. of the patients are relieved, 80 per cent. of them completely, I think one is justified in repeating an opinion expressed ten years ago: The results of gastro-jejunostomy performed in suitable cases are as satisfactory as those of any other major operation.

There must, however, be a definite pathological indication. Some at least of the failures are due to disregard of this vital rule. I have undone twenty-three gastro-jejunostomies in which there was no evidence of a gastric or duodenal lesion, the symptoms being due to such various conditions as chronic appendicitis, intestinal stasis, locomotor ataxy, gall-stones and Bright's disease. An operation performed on a clinical diagnosis, unconfirmed by the pathological findings, is a mistake both in diagnosis and in operation.

(II) PHYSIOLOGICAL.

A study of the physiological effects of gastro-jejunostomy is important as showing us how gastro-jejunostomy acts, and teaching us the proper indications for its use.

There are two striking and constant effects of gastro-jejunostomy on the gastric contents: (1) The diminution of the total acidity; (2) the increase of mineral chlorides. These are shown in the following typical gastric analysis:—

GASTRIC ANALYSIS.

Before gastro-jejunostomy—	
Total chlorides	0·412
Free hydrochloric acid	0·036
Protein hydrochloric acid	0·266
Mineral chlorides	0·109
After gastro-jejunostomy—	
Total chlorides	0·332
Free hydrochloric acid	0·000
Protein hydrochloric acid	0·146
Mineral chlorides	0·186

The diminution of the total acidity is due, partly to the diminution of the total chlorides secreted by the gastric mucosa, and partly to the neutralization of free hydrochloric acid by the small quantity of bile and pancreatic juice which back-flows into the stomach through the anastomosis. This increase of the mineral chlorides is constant and permanent. It is not the result of increased activity of the gastric mucous membrane, it must, therefore, be due to chlorides added from without. Therefore the inference is irresistible that the increase is due to the mineral chlorides of the bile and pancreatic juice, which gain entrance into the stomach through the anastomosis.

Further proof of the correctness of this hypothesis is afforded by the effect on the gastric contents of undoing a gastro-jejunostomy. The result has always been the same, an increase in the mineral chlorides after gastro-jejunostomy and a decrease towards the normal after the restoration of the alimentary canal to its normal condition. This is shown in the following analyses:—

Before gastro-jejunostomy—	
Total chlorides	0·420
Free hydrochloric acid	0·051
Protein hydrochloric acid	0·289
Mineral chlorides	0·080
After gastro-jejunostomy—	
Total chlorides	0·365
Free hydrochloric acid	0·000
Protein hydrochloric acid	0·153
Mineral chlorides	0·211
After gastro-jejunostomy was undone—	
Total chlorides	0·343
Free hydrochloric acid	0·018
Protein hydrochloric acid	0·270
Mineral chlorides	0·055

Further, if this hypothesis be correct, in those cases in which, in addition to gastro-jejunostomy, an entero-anastomosis is performed, we should not expect to find this increase of mineral chlorides, inasmuch as the bile and pancreatic juice are diverted through the entero-anastomotic opening. This, in my experience, is invariably the case, as exemplified in the following analyses :—

Before gastro-jejunostomy—	
Total chlorides	0·335
Free hydrochloric acid	0·003
Protein hydrochloric acid	0·175
Mineral chlorides	0·156
After gastro-jejunostomy and entero-anastomosis—	
Total chlorides	0·350
Free hydrochloric acid	0·000
Protein hydrochloric acid	0·226
Mineral chlorides	0·124

If my hypothesis as to the cause of the increase of mineral chlorides be correct, then it is important to compare the effects of the different types of operation on the increase of mineral chlorides, as this gives us an indication of the amount of bile and pancreatic juice which regurgitates into the stomach. The average increase of mineral chlorides after the different types of gastro-jejunostomy is shown in the following table :—

AVERAGE INCREASE IN MINERAL CHLORIDES AFTER GASTRO-JEJUNOSTOMY.

Posterior (Mayo)	0·082
Posterior (iso-peristaltic)	0·096
Posterior (vertical)	0·087
Anterior (transmesocolic)	0·080
Anterior (long-loop)	0·070

The differences are so small as to be negligible. These observations indicate that, physiologically, the type of operation performed is unimportant, with the exception that all types of Y-operations and gastro-jejunostomy with entero-anastomosis are unsound. The conclusion to be drawn is that gastro-jejunostomy is not a mechanical but a physiological operation, and its action is to enable nature to counteract hyperacidity, and neutralize the effects of hypersecretion.

As to the effect of gastro-jejunostomy on the metabolism of the human body, I have elsewhere recorded a series of experiments showing that the operation of gastro-jejunostomy does not appreciably diminish the proportion of the nitrogen or fat absorbed from the food. We are therefore justified in concluding that both clinical and chemico-pathological evidence demonstrate that the operation of gastro-jejunostomy does not appreciably interfere with the metabolism of the human body, and is compatible with the enjoyment of perfect health.

(III) Pathological.

In five instances I have had an opportunity of examining post mortem the condition of affairs four or five years after an anterior gastro-jejunostomy with a long loop. Those cases show how remarkably Nature adapts herself to altered conditions. At first sight it is somewhat difficult to believe that an anterior operation has been performed, as the anterior surface of the stomach becomes drawn downwards and posteriorly, while the afferent limb appears to become shortened, so that instead of measuring 10 or 12 in. it measures only 4 or 5 in. Further, although the afferent limb at the time of the operation is drawn up round the colon, later it appears to be displaced outwards so that it emerges from under the colon at or near the splenic flexure. Thus Nature appears to protect the afferent loop from the influence of the excursions of the movable part of the transverse colon.

As to the result of gastro-jejunostomy on the healing of the ulcers, I have had an opportunity of investigating this point post mortem on sixteen occasions: in all of them the ulcer, whether gastric or duodenal, had healed.

As regards the anastomotic opening, there is usually some contraction: I have found the opening which was originally made $2\frac{1}{4}$ to $2\frac{1}{2}$ in. long has diminished to $1\frac{3}{4}$ to 2 in. in length.

Jejunal Ulcer.

Jejunal ulcer is perhaps the most serious after-complication of gastro-jejunostomy. The term "jejunal ulcer" has been applied somewhat loosely to ulcers round the anastomotic opening, and ulcers situated in the jejunum. A clear distinction should be made between ulcers in these two situations. For those situated round the anastomosis I have suggested the description "gastro-jejunal ulcers." The proportion of cases in which gastro-jejunostomy is followed by jejunal or gastro-jejunal ulcer is difficult to estimate with accuracy. I have had four cases of my own, and have operated on six other cases and have seen several others in consultation. From the records available I think we are justified in assuming that the proportion is between 1 and 2 per cent.

The appearance of a jejunal ulcer resembles that of a gastric ulcer so closely that no description is necessary. The important point to discuss is the ætiology There are strong grounds for believing that jejunal ulcer is the result of the toxic action of free hydrochloric acid on the jejunal mucous membrane. The hydrochloric acid so injures or kills the cells of the jejunal mucous membrane that they are readily digested by the intestinal juice. There are two very suggestive circumstances with regard to jejunal ulcer:—

(1) Jejunal ulcer is usually (in my own limited experience invariably) associated with hyperacidity of the gastric contents.

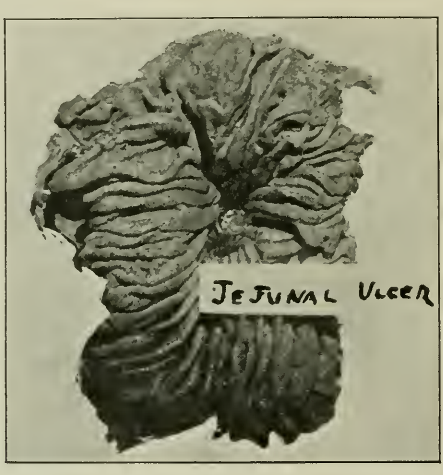

FIG. 1.

A jejunal ulcer, which perforated the anterior abdominal wall five years after anterior gastro-jejunostomy. Symptoms of indigestion began three years after the gastro-jejunostomy. The portion of jejunum affected (about 6 in.) was resected. The patient lived eleven years after the second operation without further gastric symptoms.

(2) In the majority of instances the ulcer is situated either opposite the anastomosis or just below it in the efferent limb—that is, just where the acidity of the gastric contents when they reach the jejunum is greatest.

Those facts are very significant. Usually after gastro-jejunostomy, the small amount of bile and pancreatic juice which back-flows into the stomach, is sufficient to neutralize or reduce to normal the free hydrochloric acid present in the gastric contents. If, however, the amount of free hydrochloric acid in the gastric contents be excessive, then the bile and pancreatic juice may not neutralize the free hydrochloric acid sufficiently to prevent its toxic action on the jejunal mucosa when it comes in contact with it. Thus, we get a jejunal ulcer usually in the situation to be expected if this hypothesis be true—i.e., either opposite or just below the anastomosis.

Some years ago I had an opportunity of making some observations on one of my patients in whom a jejunal ulcer perforated through the abdominal wall. Investigations of the gastric contents showed the presence of 0·072 per cent. of free hydrochloric acid, as seen in the following analysis :—

GASTRIC ANALYSIS (JEJUNAL ULCER).

Total acidity	85·0
Total chlorides	0·433
Free hydrochloric acid	0·072
Protein hydrochloric acid	0·208
Mineral hydrochloric acid	0·153

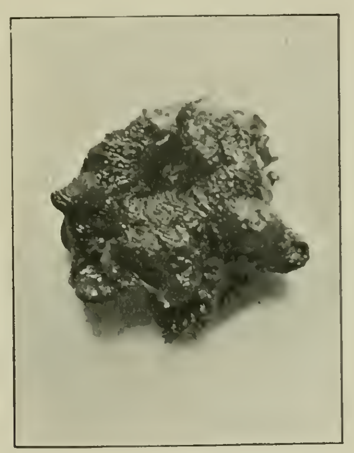

FIG. 2.

A portion of jejunum showing a deep punched-out jejunal ulcer, which was resected seven years after post-gastro-jejunostomy. The patient had had symptoms on and off for about two years. The ulcer was situated immediately opposite the anastomosis. The walls of the jejunum are greatly thickened.

We would expect that such a high degree of acidity would not be neutralized by the bile and pancreatic juice. This proved to be the case as shown by an analysis of the fluid escaping from the fistula which contained 0·044 per cent. of free hydrochloric acid.

FLUID FROM FISTULA (JEJUNAL ULCER).

Total chlorides	0·405
Free hydrochloric acid	0·044
Protein hydrochloric acid	0·175
Mineral hydrochloric acid	0·186

After a week on a purely milk diet the percentage of free hydrochloric acid in the gastric contents was reduced to 0·18, that is about normal.

GASTRIC ANALYSIS (JEJUNAL ULCER).

Total acidity	80·0
Total chlorides	0·361
Free hydrochloric acid	0·018
Protein hydrochloric acid	0·187
Mineral hydrochloric acid	0·146

With this strength of hydrochloric acid the bile and pancreatic juice were able to deal, so that the fluid flowing from the fistula contained only 0·004 per cent. of free hydrochloric acid.

FLUID FROM FISTULA (JEJUNAL ULCER).

Total chlorides	0·372
Free hydrochloric acid	0·004
Protein hydrochloric acid	0·092
Mineral hydrochloric acid	0·276

From observations I have made we may, I think, draw three inferences:—

(1) That the presence of an ulcer in the jejunum is usually correlated with the presence of free hydrochloric acid in the jejunal contents.

(2) That as a high percentage of free hydrochloric acid in the gastric contents diminishes rapidly on a milk diet, the excess of free hydrochloric acid is due probably to errors in diet.

(3) That the bile and pancreatic juices in the jejunum usually neutralize the free hydrochloric acid which passes through the anastomosis. When, however, free hydrochloric acid reaches the jejunum in great excess, the alkaline juices are insufficient to neutralize all the free hydrochloric acid present. It may be objected that in some of the recorded cases gastric acidity is stated to have been normal. There is no difficulty in explaining such cases, even if the statement be accurate. Although the acidity of the gastric juice may be normal, the amount secreted may be abnormal, in other words, hypersecretion may exist without hyperacidity. If there be hypersecretion, although the percentage of free hydrochloric acid in the gastric juice be normal,

the quantity entering the jejunum may be so great that it is not completely neutralized by the bile and pancreatic juice. In other words, the prolonged or continuous action of a weak solution of hydrochloric acid may have the same effect as the intermittent action of a strong solution.

Jejunal ulcer is sometimes attributed to damage to the intestine caused by the use of clamps. There are two objections to this hypothesis:—

(1) The interval between operation and the onset of symptoms of jejunal ulcer. In my own cases the interval varied between two and five years.

(2) The ulcer usually occurs in a situation not gripped by the clamps.

In a few cases jejunal ulcers are probably of infective origin. Such cases present the following features: (*a*) The ulcer occurs within a very short period after gastro-jejunostomy; (*b*) Usually the ulcers are multiple. Probably those ulcers recorded as having occurred in the afferent limb of the jejunum belong to this group.

SCHEME OF CAUSES OF JEJUNAL ULCER.

Hyperacidity, normal flow of bile and pancreatic juice	Jejunal ulcer.
Normal acidity, but hypersecretion, normal flow of bile, &c.	
Normal acidity, diminished flow, or diversion of bile, &c.	
Normal acidity, normal flow of bile, &c.; toxic agent other than hydrochloric acid	
Infective processes	

The conditions under which jejunal ulcers may arise may be summed up as follows:—

(1) Hyperacidity of the gastric contents, so that the bile and pancreatic juice are unable to neutralize completely all the acid entering the jejunum.

(2) Normal percentage of hydrochloric acid in the gastric juice but excessive secretion, so that the amount of hydrochloric acid discharged into the jejunum is greater than can be neutralized, and there is a continuous action on the jejunal mucosa of a weak solution of hydrochloric acid.

(3) Diversion of the course of the bile and pancreatic juice so that part of the jejunum is exposed to the action of the gastric contents unmixed with bile and pancreatic juice, as after operations of the "Y-type," and gastro-jejunostomy with entero-anastomosis.

(4) Normal acidity and amount of gastric secretion, but incomplete neutralization in the jejunum owing to temporary diminution of the flow of bile and of the secretion of pancreatic juice.

Dr. Charles Bolton has made a series of most instructive and valuable experiments on the artificial production of gastric ulcers, and his observations appear to me to support the above views as to the ætiology of jejunal ulcer. Dr. Bolton found that feeding guinea-pigs on a solution of hydrochloric acid up to 0·7 per cent. produced no effect whatever, but a solution above that strength produced gastric lesions identical in character with those produced by a gastro-toxic serum. He concludes that the gastric juice is not only concerned in the initiation of a gastric ulcer, but also in its tendency to spread and to become chronic; and that the gastric juice owes its destructive power primarily to the hydrochloric acid contained in it, and the damage done to the wall of the stomach varies in proportion to the strength of the acid present.

I think we may argue fairly that these conclusions as to the action of hydrochloric acid on the gastric mucous membrane may be applied with even greater force to the action of hydrochloric acid on the jejunal mucous membrane which under normal conditions is unused to the presence of acid gastric contents.

With regard to gastro-jejunal ulcer, much of what has been said applies also to this variety of ulcer. Hyperacidity is the important factor in both, but there is this difference: jejunal ulcers are a result of altered physiological conditions produced by operation; gastro-jejunal ulcers are probably a direct consequence of operation.

Whether or not primary union takes place between mucous membranes of the stomach and jejunum is a disputed point. But this is certain: if the mucous membrane be roughly handled or injured, or unduly constricted by too tight a suture, or if coaptation of the gastric and jejunal mucous membranes be imperfect, ulceration will follow at the line of suture. If hyperacidity or hypersecretion exist then the ulcer instead of healing, may persist or spread, and eventually give rise to symptoms, and may lead to great constriction or even closure of the anastomotic opening.

The view that the use of a non-absorbable suture is a cause of gastro-jejunal ulcer, I regard as non-proven. It is true I have seen the end of the thread hanging through the anastomosis at a subsequent operation on more than one occasion, but there was no ulceration; but in the cases I have examined post mortem, the thread had often become encapsuled, and there was direct continuity between the gastric and jejunal mucous membranes.

If these views be correct, the practical point which arises is this: What are the causes of hyperacidity after gastro-jejunostomy? We must draw a clear distinction between two classes of cases:—

(1) Cases in which hyperacidity is present before, and persists after, operation.

(2) Cases in which, after a period of normal or subnormal acidity following operation, the gastric contents subsequently become hyperacid.

(1) Hyperacidity persists after gastro-jejunostomy for one or two reasons: (*a*) Extreme hyperacidity before operation, so that the bile and pancreatic juices are unable to reduce the acidity of the gastric contents to normal, unless the patient is kept on a milk diet for some months. (*b*) A too small or inefficient opening. If the opening be too small hypersecretion may persist. Likewise any kink or twist at the site of the anastomosis will prolong the contact of the gastric contents with the jejunal mucosa.

(2) Hyperacidity of the gastric contents succeeding a period of normal or subnormal acidity is due probably to one of two causes: too small an opening, or indiscretions in diet. In some cases both these factors may be present.

It is sometimes forgotten that operation is only an incident in the treatment of gastric and duodenal ulcer, and too much emphasis cannot be laid on the importance of a carefully regulated diet after operation. I believe that in most cases—certainly in all of my own—indiscretions in diet play the chief part in causing post-operative hyperacidity, and so predispose to the occurrence of gastro-jejunal and jejunal ulcers.

In conclusion, let me recapitulate briefly what appear to me to be the practical conclusions at which we may arrive:—

(1) Gastro-jejunostomy is not an obsolete operation. When performed with proper indication the results are as satisfactory as those of any other major operation in surgery. The failures are not more than 7 per cent.

2) Gastro-jejunostomy is a physiological operation, and it is as

efficient a treatment for ulcers of the body of the stomach as for ulcers near the pylorus.

(3) Operations of the "Y-type," or gastro-jejunostomy with entero-anastomosis, are wrong in principle, and unsatisfactory in practice, as they permit of the direct contact of the acid gastric juice with the jejunal mucous membrane, before it has been mixed with the alkaline bile and pancreatic juices.

(4) With the above exception the type of gastro-jejunostomy performed is of less importance than the manner in which it is performed.

(5) Gastro-jejunostomy has no appreciable effect on the metabolism of the human body.

(6) There is good pathological evidence that ulcers heal as the result of gastro-jejunostomy.

(7) The risk of jejunal ulcer following gastro-jejunostomy is probably under 2 per cent.

(8) Clinically there are two groups of cases: (*a*) Those in which perforation into the general peritoneal cavity occurs; (*b*) those in which general peritonitis is prevented by the formation of adhesions.

(9) Pathologically, the cases may be classified as follows: (*a*) jejunal ulcers; (*b*) gastro-jejunal ulcers or ulcers at the site of the anastomosis.

(10) Occasionally jejunal ulcers are of infective origin. In these cases ulceration commences within a short period after gastro-jejunostomy, and usually the ulcers are multiple.

(11) In the great majority of cases a jejunal ulcer is single, and is probably the result of the toxic action of hydrochloric acid, which injures the cells of the mucous membrane so that they are readily digested by the intestinal juice.

(12) Gastro-jejunal ulcers are a direct consequence of the wound made in affecting the anastomosis, and their persistence is probably the result of hyperacidity of the gastric juice.

(13) Every case of recrudescent pain of a constant character after gastro-jejunostomy, especially when associated with hypersecretion, should be regarded as a case of potential ulcer and treated accordingly.

(14) Gastro-jejunostomy is but an incident in the treatment of gastric and duodenal ulcers. A prolonged period of restricted diet is imperative, especially in cases in which there is hyperacidity before operation. In all cases of hyperacidity increase in diet should be controlled by gastric analysis at regular intervals.

Sir HAMILTON BALLANCE.

I was interested in this subject because I have operated upon no less than four cases of jejunal ulcer during the last six months. One of them had had, fourteen and a half years previously, a gastro-enterostomy performed. Personally, in doing gastro-enterostomy, I use very fine silk. I agree with the opener that it is not proved that the use of silk, rather than absorbable sutures, is the cause of jejunal ulcers following gastro-enterostomy. I have generally advised patients to use, after this operation, an alkali, such as bicarbonate of soda, before meals and last thing at night just before getting into bed. They have generally taken ½ dr. ten or fifteen minutes before meals. A few weeks ago I operated upon a patient who had carried that treatment out for four and a half years, following on a gastro-enterostomy, yet that had not prevented the onset of jejunal ulcer. I am sorry to say she died after the operation for its relief.

I do not know at all how to prevent jejunal ulcer forming. It is undoubtedly the gravest remote complication of gastro-enterostomy, and operations for its relief are certainly among the gravest in surgery. They may take a very long time, because multiple anastomoses may be required. If anyone here can let us know how to prevent this very grave complication, I think this discussion will by no means have taken place in vain. I have brought with me two specimens of jejunal ulcer, and in thanking the President for asking me to speak, I must apologize for the very fragmentary character of my remarks.

Mr. GARNETT WRIGHT.

In a large subject such as we have under consideration to-night it is impossible and inadvisable for each speaker to endeavour to cover the whole ground. The particular aspects with which I am concerned are (1) the effect of gastro-jejunostomy on lesser curvature ulcers, and (2) secondary ulceration.

(1) *The Effect of Gastro-jejunostomy on Ulcers of the Lesser Curvature.*—While it is true that in a number of cases the operation of gastro-jejunostomy will effect a cure—and I may say that I was rather agreeably surprised at the number of favourable replies I received—yet there are too many other patients in whom temporary relief is followed by a disappointing return of symptoms, and subsequent operation shows

the presence of an unhealed ulcer. I have performed secondary operations on several such cases of my own and other surgeons' during the past twelve months. The curious inefficiency of gastro-jejunostomy in such cases is well illustrated by a patient, a man aged 45, on whom I did a posterior gastro-jejunostomy for duodenal ulcer in August, 1918. Inspection of the stomach failed to reveal any ulcer except the duodenal ulcer. For five months his symptoms were completely relieved, and then pain recurred, and in March, 1919, perforation took place and was operated on, the ulcer being situated high up on the lesser curvature near the œsophagus. The ulcer was very inaccessible and was merely closed. His symptoms of indigestion recurred in a month or two, and on November 4, 1919, I again operated on him, and excised the ulcer by a transgastric method. Since then he has remained well. Here an ulcer developed in the stomach after gastro-jejunostomy had been established. The fairly frequent failure of gastro-jejunostomy alone in this type of case has led me to adopt one or other method of excision, either wedge-shaped excision and gastro-enterostomy or partial gastrectomy. This last method is the one I prefer. It has the advantage of removing the ulcer-bearing area, as Rodman pointed out as long ago as 1904. Technically, I find it as easy, if not easier, than wedge-shaped excision, and though my number of cases, sixteen, is too small to enable me to form a just estimate of the mortality, as I have not yet lost a patient from this operation for gastric ulcer, I am hopeful that the mortality will prove to be pretty low. The largest statistics (Mayo clinic) give the mortality as 7 per cent. Troell gives 8 per cent. from the statistics of the Seraphim Hospital, Stockholm. The operation which I do at present is the anterior Polya operation.

(2) *Secondary ulceration* is the most important of the harmful remote sequelæ of the operation of gastro-jejunostomy. The frequency of this complication has been estimated by Paterson as 1·5 per cent., but it is almost impossible to get the data for a reliable estimate. I am convinced that in its slighter manifestations it is a much commoner condition than is usually suspected, and that many patients in whom a recurrence of the original ulceration, be it duodenal or gastric, is suspected, the real lesion is a new ulceration on the anastomosis, or in the jejunum. The surgeon who re-opens his failures systematically will recognize this troublesome complication more frequently than his fellows who are shy of secondary operations. There seems to be an opinion that this complication is more frequent in the practice of

inexperienced surgeons, but a review of the published cases does not support this view. Most of them have occurred in the practice of well-known surgeons. In considering the causation of such secondary ulceration we have to take three factors into account: (1) The digestive action of the gastric juice; (2) sepsis; (3) trauma. A combination of two or more of these factors is necessary to the production of secondary ulceration.

(1) The *influence of gastric digestion* is shown in the distribution of the ulcers in a position subject to the action of the non-neutralized gastric juice and in the greater liability to ulceration in operations complicated by some form of entero-anastomosis. There is a steadily diminishing power of resistance to gastric digestion as one descends the small intestine. Thus, no secondary ulcers have been reported after gastro-duodenostomy, and more occur after anterior than after posterior gastro-jejunostomy.

(2) *Sepsis* is of importance, just as it is in the production of the original ulceration. We may exclude gross sepsis introduced at the time of operation in most cases, though the possibility of an infected suture must not be overlooked. There is, however, a potent source of infection in the presence of an unhealed gastric or duodenal ulcer. Rosenow has shown that streptococci are constantly present in such ulcers, and my colleague, Dr. Langley, has made cultivations from patients on whom I have operated for gastric and duodenal ulcers, and has recovered streptococci in large numbers from the ulcers, very few colonies being recovered from the rest of the mucosa. We have here an argument in favour of a direct attack on the ulcer at the original operation. An anastomosis established with an unhealed ulcer in existence in the stomach or duodenum, is exposed to the risk of infection from the ulcer, and I think that a certain number of secondary ulcerations, especially of the gastro-jejunal type, can be explained in this way.

(3) *Trauma* is often an accessory agent in the production of secondary ulceration, and this leads us to the operative technique of the preliminary operation. In fourteen out of 155 cases Murphy's button or some form of bobbin had been used.

In thirteen a continuous non-absorbable suture was considered to be the exciting cause of the ulceration, but cases have occurred when only absorbable suture material has been used. I have previously elaborated my views on the part played by such a continuous un-absorbable suture in a paper in the *British Journal of Surgery*, and I pointed out that even though used for the external line of sutures only, such material works its way into the lumen of the alimentary canal in course of time, and may cause abrasions, &c., of the mucosa, which form the starting point of secondary ulceration. The bruising of the bowel or stomach produced by the use of heavy clamps has been blamed, and Hartmann considers this a very important factor.

It is a curious fact that the more recent cases seem to be chiefly of the gastro-jejunal type, and one wonders whether the through-and-through hæmostatic stitch does not lead to a stoma in which the mucosa is adherent in a thin line all round. Such a stoma would be more liable to injury by hard pieces of food than one where the mucosa was freely movable over the deeper parts.

When all these possibilities have been taken into account, we have to remember that the cause of the original ulceration, whatever it may be, is probably still active, and where this is so, patients will go on developing fresh ulceration in spite of all our efforts. This is clearly evident in reading the reports of many of the published cases, in which numerous operations failed to give relief.

The *morbid anatomy* of secondary ulceration presents some points of interest. Paterson's original classification into gastro-jejunal and jejunal is a sound and valuable one. The number of cases of jejunal ulceration slightly exceeds the gastro-jejunal variety. In 142 cases—gastro-jejunal sixty-three, jejunal seventy-nine. Of the jejunal in fifty-four the ulcer was on the efferent loop, in two on the afferent loop, in ten opposite the stoma—i.e., on the mesenteric border of the jejunum. In nine cases the ulcers were multiple, and in four the site was not definitely stated.

Acute perforation into the peritoneal cavity took place in thirty-one cases, and is more liable to happen after anterior gastro-enterostomy (28·7 per cent. of the cases as against 15·3 per cent.).

Chronic perforation into the abdominal wall is often seen after anterior gastro-enterostomy, large inflammatory tumours and sometimes external fistulæ being produced.

After *the posterior operation* chronic perforation into the mesentery is frequent, the mesentery of the colon becoming much thickened and contracted, until finally the colon is brought into contact with the ulcer, perforation into the colon and jejunocolic fistula resulting. Fistula into the colon is almost invariably secondary to a posterior gastro-enterostomy. It occurred in twenty-one out of 142 cases which I collected, and in only two instances was the preliminary operation an anterior one, and in one of those it was of the retrocolic variety.

Ulcers opposite the stoma—i.e., in the mesenteric border of the jejunum are of interest, in that the contraction of the ulcer tends to produce a spur at the gastro-enterostomy opening, and consequent obstruction. The question of pressure of heavy clamps as a factor in the causation of such ulcers also arises, as they are situated just where pressure of clamps is apt to be exerted.

Mr. Grey Turner has some interesting specimens here, showing an unabsorbable suture hanging from an intestinal anastomosis and a pylorus which had been ligatured with silk, the silk having disappeared, this showing that such material eventually finds its way into the lumen of the intestinal canal.

Mr. G. Grey Turner.

I am sorry the subject chosen for debate to-day is not more limited in its scope, because I feel it would have been sufficient if we had tried merely to elucidate some points on the important subject of jejunal ulcer and its late sequelæ. However, the whole question of ulceration in the stomach and duodenum is of great importance, and I cannot help feeling, like the last speaker, that there are a number of underlying factors which we have not yet been able to fathom.

The subject impresses me as one of very great difficulty indeed. For instance, only recently a patient returned under my care suffering from a gastric ulcer. She was a young woman, aged 27, on whom I had operated eight years previously for an acute perforation of a chronic ulcer on the lesser curvature of the stomach. At that time she was very ill, and I felt very much relieved that she recovered. Gastro-enterostomy was not performed, and she not only made a good immediate recovery, but remained in excellent health, got married, and had a family. She had no further stomach trouble until two months before her return to the hospital. At the second operation I found a large ulcer at the original site which had all the appearances of an ordinary chronic gastric ulcer, with some superadded infection.

In another case, a lady, aged 33, was operated upon for an ulcer on the posterior surface of the stomach by pyloroplasty—it was in the earlier days of stomach surgery, when the indications as to operation were not so fully understood as they are now. She made a perfect recovery, and for six years was entirely free from stomach symptoms. At the end of that time there was a recurrence, and, unfortunately, the patient succumbed after an operation for gastro-enterostomy. The post-mortem examination disclosed a large chronic ulcer which was adherent to the pancreas of just such a size and position as could scarcely be expected to have ever spontaneously healed. What had been the condition of these ulcers in the years between the operations?

With regard to jejunal ulcers we now know that they occur in what I think is an alarming proportion of cases after gastro-enterostomy, and that they may recur, in spite of our efforts for their cure. All these things make me feel there are many underlying factors in the production of ulceration which we do not understand, and, until we know more about these factors, our surgical efforts are not likely to be attended with complete success.

Some of the cases of ruptured ulcer provide us with interesting food for thought. I want to refer to the case of a boy who, at different times, had no less than three perforations. The first occurred at 13 years of age. It was on the anterior surface of the stomach, near the pylorus. After the operation of simple suture, he was well until 16 years of age. Then he had his second perforation, this time on the posterior surface of the stomach, away from the pylorus. He remained in excellent health for a year, when he had a return of indigestion, terminating in acute perforation two years after the second operation. I found a large chronic ulcer at the cardiac end of the stomach, just in front of the hilus of the spleen. It was a very difficult perforation to close, and I had to apply the spleen to reinforce the suturing. He made a good recovery, but at the end of three weeks from that operation, when we hoped he was nearly well enough to enable us to do a secondary gastro-enterostomy, he suddenly developed acute symptoms suggestive of a further perforating lesion. On re-opening the abdomen, I found an abscess in the left flank which had ruptured, and caused localized peritonitis. In spite of this, I performed gastro-enterostomy, and he again made a good recovery. Twenty-nine days later he had acute intestinal obstruction, which was dealt with by further laparotomy, and now, ten years from the date of his first operation, he is in perfect health, having been on active service abroad all through the war.

And now to deal with non-perforating ulcers. One hears much about the disappointing results following gastro-enterostomy, but we must admit that in certain cases those disappointing results are deserved, while in other instances they are as undeserved as they are inexplicable. I have had to operate in two instances which come under the first head the original interference having been carried out elsewhere. In one, the stomach had been united to the small intestine within 12 in. of the ileo-cæcal valve. As bearing upon the question of secondary ulceration, the piece of ileum (which had been united to the stomach for two years) does not show any macroscopic sign of ulceration, though there are areas of inflammation, and when I go home I shall make a careful examination of that intestine, to see if I can learn anything from it with regard to the onset of peptic ulcer. In the other instance of a well-deserved bad result, the jejunum had been attached to the large intestine as well as to the posterior wall of the stomach.

The disappointing results which are not deserved are exceedingly difficult to explain. I have seen re-activity of the ulceration, a fair proportion of jejunal ulcers, and some examples of recurrent vomiting.

Some years ago I made a careful investigation into a considerable number of cases which were operated upon for chronic ulcer of the stomach and duodenum. In a series of 149 cases in which the operation was duly indicated and properly carried out, there were 135 patients who stated they were clinically cured. But these results only extended over a period of from one to nine years, and I cannot help admitting that now, as gastro-enterostomies have been performed for a longer time, and in a larger proportion of the population, we are getting more unfavourable secondary results than we saw in the earlier stages of the operation considered historically. I do not wish to say anything against the magnificent results presented in the majority of cases in which the operation has been properly indicated ; but there is the fear of secondary ulceration, which should make us pause and consider if we are doing the best operation in the majority of our cases, or whether we ought not rather to select some particular operation for each special type of case. I have been brought up in an atmosphere in which gastro-enterostomy has been looked upon as a makeshift and unphysiological operation, and in which surgeons have been particularly anxious, when they could, to make a direct attack on the pylorus or an anastomosis at that end of the stomach. And that has made me greatly interested in the often-forgotten operation of pyloroplasty. Some years ago I followed up twenty consecutive cases which had been operated upon by this method, for periods of from ten to fifteen years. Out of the series followed for that long time, there were 83 per cent. of complete cures.

With regard to the question of technique, Mr. Garnett Wright has mentioned to you the specimen I show this evening, in which an unabsorbed suture of celluloid thread is hanging loose in the intestine seven months after operation. I do not think that can be good. I do not know what pathological change may result from it, but it is not an ideal condition, and I see no reason for the use of unabsorbable sutures. For many years I used fine chromicized catgut, and without untoward results. Later, I was persuaded to use silk and now I have come back to catgut, which I regard as perfect. Another point in technique has not been mentioned, and that is the question of excision of the mucous membrane. I think that is a mistake in doing gastro-enterostomy or any operation involving intestinal anasomosis, for we require as complete a covering for the submucous tissue as possible, and that is best secured by leaving the excess of mucous membrane; it protects the vessels and makes a soft movable layer over the line of suture.

In certain of the cases, surgeons have reinforced the suture-line by stitching the edges of the hole in the mesocolon directly to the anastomosis. I think this plan came from the Mayo clinique. But if this is done as a routine, then in certain cases, especially in patients with a fat mesocolon, a fibrous ring develops, which may cause constriction of the anastomosis, or lead to a sort of recurring intussusception into the stomach, which, I am certain, accounts for some bad results in which the patient has recurring vomiting.

Mr. James Sherren.

The remote results of the surgical treatment of chronic gastric and duodenal ulcers rival those of any major operation. If the appropriate procedure is adopted the ulcer heals permanently, and any symptoms which may arise later are the outcome of ulcerative or mechanical changes associated with the anastomosis.

I have dealt with the question very fully in my recent Hunterian Lecture, and intend this evening to give my views very briefly and without all the evidence on which they are based.

Chronic ulcers of the stomach and duodenum heal as the result of gastro-jejunostomy correctly performed, unless chronic perforation has occurred, the floor of the ulcer being formed of liver or pancreas, thus preventing the contraction necessary to sound healing. In my experience recurrence of a simple ulcer after suitable surgical treatment is unknown. I have not seen it at post-mortem examinations nor at second operations carried out for other conditions.

I have been able to examine, post-mortem, nine cases I have operated upon for chronic duodenal ulcer from six days to eleven years later and found the ulcer healed in all but the first. I have had similar opportunity in eight of my cases of chronic gastric ulcer from fourteen days to eight years after operation: in all the ulcer had healed. I have examined the site of the ulcer at second operations in forty cases: the ulcer had healed in every one. The only instances in which I have seen the ulcer unhealed have been at second operations for posterior eroding ulcer treated by gastro-jejunostomy alone; these I now invariably deal with by partial gastrectomy.

I have here a specimen showing healing of a chronic duodenal ulcer more than eleven years after gastro-jejunostomy. I obtained it last week from a male upon whom I operated on June 4, 1908. He gave a typical history of chronic duodenal ulcer extending over ten years. Three days before operation he had profuse hæmatemesis and melæna. I found extensive ulceration on the anterior and posterior surfaces of the duodenum at the junction of the first and second parts. He made an excellent recovery, and during the war served in a labour battalion in France. He had no abdominal trouble until about six weeks before death. He came to see me on February 9 on account of swelling of the abdomen of about a month's duration. Post-mortem examination revealed a primary carcinoma of liver. You will see the smooth deep scar on the posterior surface and the thinner one of the anterior surface opposite to it. The anastomosis is large and smooth and the jejunum normal.

The two other specimens also show the ulcers healed at over eight and three years after operation.

I believe that the action of gastro-jejunostomy is twofold. It reduces gastric acidity and leads to a more rapid emptying of the stomach. Its good effects are not solely nor mainly mechanical.

Malignant disease of the stomach may occur at remote periods after treatment of chronic gastric ulcer by gastro-jejunostomy alone. It was the probable cause of death in three of my series at seven, five and four years after operation. I was not able to obtain a post-mortem in either so that I am unable to say if it arose at the seat of the simple ulcer. They were all cases that I should treat by partial gastrectomy at the present time. In 3 per cent. of chronic ulcers of the stomach which I have removed at operation, the naked-eye diagnosis being simple, there being no evidence or suspicion of malignancy, carcinoma starting in one edge was discovered on microscopic examination.

I know of no case of carcinoma of duodenum arising after operation for chronic duodenal ulcer, but in two cases in which the ulcer was involving the stomach further operation became necessary within eighteen months and revealed carcinoma of stomach, the duodenal ulcer being healed. I now always treat this type of case by partial gastrectomy.

Post-operative vomiting of the regurgitant type is due to errors of judgment or technique. In all my cases, except the first, some abnormality was present at the first operation and some definite mechanical change found at the second.

Secondary ulceration will, I believe, become as rare as regurgitant vomiting is at the present time. Gastro-jejunal or anastomosis ulceration is due to the use of unabsorbable suture material for the inner or outer layer and is the only cause of stricture of the opening. I believe that an anastomosis ulcer may become malignant. Jejunal ulcer is due to failure of the operation sufficiently to lower gastric acidity at the same time that it fails to remove the cause of the original ulcer or damages the jejunum. It is almost unknown after gastrojejunostomy for chronic gastric ulcer. In all cases of true jejunal ulcer gastric acidity is higher than it should be when compared with the pre-operative, unless the ulcer is communicating with the colon, when a low acidity is the rule.

The use of a jejunal clamp is one of the causes of jejunal ulcer, but this of itself would not be enough unless the acidity remained high. I noted bruising by clamp in six cases in which I have records of test-meals, both before and after operation, after I had begun to suspect this as a cause. In three gastric acidity remained high, and ulcer developed in all; in the others the acidity is distinctly lowered and there have been no symptoms pointing to ulceration. Operation was over two years ago.

I have two specimens bearing on the question of jejunal ulcer, one showing the result at a late period after operation; the other, a common sequence, perforation into the colon.

The first came from a deaf and dumb man. I operated on him in January, 1911, and noted bruising of the jejunum distal to the anastomosis. This is not included among the six mentioned, as no test meal was given on account of the difficulty of communication. He was readmitted seven months after operation with a profuse hæmatemesis. I operated and found an ulcer the size of sixpence on the anterior wall of the jejunum in the situation of the hæmatoma. It was quite free from the anastomosis, which showed no sign of ulceration. I excised the ulcer and he remained well abdominally, but I again took him into hospital last year with a malignant tumour of neck. He died from carcinoma of a parathyroid with secondary deposits in right lung. The specimen shows thin scars on the anterior and posterior surfaces of the first part of the duodenum 1·3 cm. beyond the pylorus. The gastro-jejunostomy opening and the jejunum are normal. This illustrates the development of jejunal ulcer as a purely local complication.

The next specimen shows a sequel that I have found in over a third of the cases of true jejunal ulcer-perforation into the colon. I operated on this patient in the latter part of 1911. Within a few months he began to get the pain in the lower left abdomen so typical of jejunal ulcer. In 1914 I saw him with painless diarrhœa, which he had had for several months. I diagnosed a jejuno-colic fistula, and urged him to come into hospital for further treatment; this he refused and was admitted moribund from perforation of another ulcer into the general peritoneal cavity four months later. A thin scar is present on the upper and anterior surface of the first part of the duodenum constricting its lumen. The ulcer which has perforated into the colon is seen free of the anastomosis, which is healthy and shows no sign of scarring. The ulcer, which by its perforation caused death is opposite to it.

I have so recently given details of all my cases operated upon over two years that I will give very brief figures. Out of 389 cases of chronic duodenal ulcer operated on over this period I have been able to keep 348 under observation, 318 are and have remained quite well and may be claimed, I think, as successes. This is over 90 per cent. of those traced or 80 per cent. of those operated on. I had to re-operate on nineteen for secondary ulceration, ten being jejunal. Of the remainder, all except one—who has, I believe, an anastomosis ulcer—express themselves better for the operation.

The results of the cases of chronic gastric ulcer operated on during the same period is not so good, but it must be remembered that the surgical treatment of chronic gastric ulcer was not standardized during most of this time, and it therefore includes cases treated by excision alone, which were failures in most cases, requiring a second operation within eighteen months, and also those of adherent posterior ulcer, which required second operation within the same time to bring them to a successful issue and are not considered with the successes.

The cases done over two years number 310; over eighty of those kept under observation were successful, or 75 per cent. of those operated on; fourteen had second operations, five for recurrence after excision alone, five for excision of posterior adherent ulcers that had not completely healed after gastro-jejunostomy alone. All these became successes but are not included. Four were operated on for secondary ulcers, only one of which was jejunal. In the remainder complaints are few and all express themselves as better for the operation.

The remote result in the treatment of chronic gastric ulcer improves as individual experience ripens.

Dr. Robert Hutchison.

I am a whole-hearted believer in the surgical treatment of chronic gastric and duodenal ulcer, but we must not expect too much. I would not myself regard the operation as even comparatively unsuccessful merely because the patient has occasional indigestion afterwards. It must be remembered that gastro-enterostomy does not make a patient immune to functional indigestion or to attacks of gastritis. I have seen, of course, a good many failures, and a disproportionate number of these seem to come from certain districts. The bad results are perhaps more often due to injudicious selection of cases than to errors of technique, so perhaps the physicians are to blame for them more than the surgeons. Apart from gross mistakes in diagnosis, the overlooking of other pathological conditions in the abdomen, such as chronic appendicitis or gall-stones, it seems as if the co-existence of gastroptosis or marked gastric atony militates against a very successful result. In gastroptosis this may be due to the greater curvature sagging down so that the anastomosis comes to be too high up and a pouch forms behind it in which stagnation of contents takes place. I have never seen recurrence of a duodenal ulcer after the operation, and in recent years anastomosis ulcers seem to be commoner than jejunal ulcers. I accept Mr. Paterson's explanation of the production of the latter as being due to a continued hyperacidity of the gastric contents but cannot agree that this is the result of errors of diet. In so far as it is not due to the anastomosis opening being too small, it is to be regarded I think as a functional disorder of the stomach, the result of nervous disturbance. In order to prevent its occurrence the state of the patient's nervous system has to be considered and at the same time alkalies should be given in all cases in which the gastric acidity is excessive. I prefer the use of the earthy carbonates for this purpose rather than bicarbonate of soda as recommended by Sir H. Ballance. I would like to have an expression of surgical opinion as to the relative advantages and disadvantages of occluding the pylorus at the time of the operation and its effect, if any, in preventing or promoting remote ill-effects later on.

Sir D'Arcy Power.

It will be generally agreed that short-circuiting for dilated stomach—the result of duodenal ulceration—is rarely followed by any untoward results. I hoped to show the specimen preserved as No. 1951F in the Museum of St. Bartholomew's Hospital, but the preparation unfortunately is not forthcoming. It is interesting on account of the completeness of the history. From 1874 until death in 1913 the patient had suffered from numerous attacks of abdominal pain which had been diagnosed at different times as "congestion of the liver," "biliousness," "appendicitis," "gastric neuralgia" and "neurosis." He was kept in bed for many weeks at a time and was sent to Egypt in the hope of obtaining permanent relief. In 1903 he had lost 2 st. in weight and presented the typical symptoms of pyloric obstruction. A posterior gastro-enterostomy was performed. He regained his normal weight, resumed full work as a solicitor, walking to and from his office daily—a distance of ten miles—and was able to eat a City dinner without subsequent discomfort. He died in 1913—ten years after the operation—at the age of 58, from acute retention of urine due to an enlarged prostate. The description of the preparation in the museum catalogue is "stomach and transverse colon with a coil of jejunum showing a posterior gastro-enterostomy. The stomach is dilated and slightly hypertrophied; about midway along the greater curvature a window has been cut to expose the opening between the stomach and jejunum. The opening is large and patent and the mucous membrane of the stomach and small intestine appears healthy. Microscopic examination of a section taken at the point of suture shows a sharp line of demarcation between the mucosa of the stomach and intestine." I take it that the results shown in this specimen are those which follow normally in similar cases. The opening remains of the size it was made, the stomach does not entirely recover its normal size, but the patient is able to resume an active life. From time to time he becomes constipated or he eats what he knows will give him indigestion. He then suffers from abdominal discomfort, becomes depressed and believes that his former condition is returning. Gentian and rhubarb and a little advice as to dietetics soon cure him.

It may be quite otherwise when gastro-enterostomy has been done for an actively bleeding duodenal ulcer. In many cases the patient is relieved by the operation but I have had two or three in which the bleeding recurred and in one case the hæmorrhage was so repeated and severe that the patient died some months after I had operated upon him.

In cancer of the stomach and pylorus the operation is palliative and, in hospital cases at any rate, does not appear to prolong life though the pain and sickness is relieved. I do not advocate it therefore, except when these symptoms are prominent, although it is surprising how well the operation is borne even by patients who are greatly emaciated.

Neither gastro-enterostomy nor gastro-gastrostomy has given me satisfactory results in well marked cases of hour-glass stomach, for in every case the patient has returned after a longer or shorter period with a return of the symptoms. I am glad to say that so far as I know none of my patients have suffered from symptoms of "peptic ulcer" and for this I can assign no reason. I have kept in touch with most of them and when they suffer from abdominal discomfort they come and see me, so that it is unlikely that those with more serious trouble would stay away. I operate rather quickly, I do not use intestinal clamps, I suture with No. 2 silk, and I have shortened by jejunal loop from 9 in. in 1903 to 3 in. in 1920; otherwise the technique has remained unchanged.

Dr. C. Bolton.

During the past four years I have had four cases of jejuno-colic fistula, and I propose confining my remarks to this subject, more particularly as most of the literature on the subject is foreign, and very little appears to be known about it in England. Of the four cases, three recovered after operation by Mr. Wilfred Trotter, and the fourth was found at the post-mortem examination. The latter specimen I am exhibiting to-night. The first case was recorded in 1903, and to 1919 twenty-seven cases in all have been published. The addition of my four cases brings the total up to thirty-one. All the cases described have been of the male sex, from 30 to 50 years of age chiefly. The time after the performance of gastro-jejunostomy, at which the fistula appeared, varies from a few weeks up to ten years. My own cases have occurred at the following times: four months, fourteen months, four years, and five and a half years.

I propose to speak more particularly about the diagnosis, and to appreciate my remarks you must first consider the *exact conditions present.*

The cause of the fistula is the perforation of a gastro-jejunal or a jejunal ulcer into the transverse colon, such an ulcer resulting from the operation of gastro-jejunostomy. The position of the fistula depends on the position of the ulcer. Usually the colon communicates *indirectly* with the stomach through the jejunum. The fistulous opening is situated within an inch of the stoma and to the left; the colon lies in front. Occasionally, the colon communicates *directly* with the stomach if the ulcer is of the gastro-jejunal type. It may also communicate in both ways. If the colon communicates *directly* with the stomach, the case is of no great interest to the physician, because the diagnosis is clear from the typical clinical spectacle of fæcal vomiting and eructations of foul gas, passage of undigested food in the motions, and wasting. If, however, the communication is *indirect*, the condition is of very great medical interest, because the diagnosis may be extremely difficult for the following reasons: As the stomach empties, food passes down the efferent limb of the jejunum, only a small amount or none passing into the transverse colon. Leakage from the colon to the jejunum is more marked and more constant, as the colon continually contains liquid fæces, diarrhœa being a constant symptom. The amount leaking into the jejunum depends upon the liquidity of the contents of the colon, the patient being much relieved when he is constipated. Also the fæces leaking into the jejunum pass more easily down into the small intestine than back into the stomach. The small intestine thus becomes infected, and that condition keeps up the diarrhœa. *The diagnosis* turns on the finding of fæcal material in the stomach contents. *Vomiting* may be only occasional and fæcal material is not constantly present in the stomach, so that the physician may never obtain a view of the fæcal vomit. Vomiting may be absent, and fæcal material only obtained by tubage of the stomach. My own patients vomited as follows: (1) Five times in the two months that the fistula had been present; (2) once only in six months, but he acquired the habit of relieving his symptoms by voluntarily bringing up his stomach contents; (3) vomited fæces on the first occasion, and being a medical man, recognized the condition; (4) vomited "on and off" for three months. In case (2) I obtained four different types of material: (1) Ordinary stomach contents after a test meal, such as one would expect in a case of gastro-jejunostomy; the total HCl was 0·144 per cent., of which 0·108 per cent. was combined, and 0·036 free HCl. (2) Dirty yellow vomit containing 0·146 per cent. protein, HCl, and also starch and fat splitting ferments. (3) A similar material from which stercobilin and indol were isolated. (4) Finally, after several attempts he produced typical fæcal material.

Great difficulties may thus be experienced in regard to vomiting and the obtaining of evidence of fæcal contamination.

Similarly, the *diarrhœa*, which is the earliest and most constant symptom, is liable to mislead the physician. The stools are loose, brownish white in colour and not very watery. There is no separate blood nor mucus. They do not occur particularly after a meal, but at any time during the day or night, when they are liable to keep the patient awake. The frequency varies from three or four to about twelve in the day. There is commonly no undigested food present or at most a few muscle fibres or a little starch may be found. The presence of these is probably due to the condition of diarrhœa rather than to direct passage into the colon. Ferments are usually absent and the total fat is about normal; in two of my cases its percentages were 18·8 and 29·7 dry weight. If the fistula is a big one or of the gastro-jejuno-colic type, undigested food may be present, but usually it is no bigger than the little finger, and no doubt the mucous membrane of the colon protrudes into its aperture. There are usually intervals during which the motions are formed, and now the vomiting stops and the patient feels much better.

Eructations of foul gas are present, but it would be a mistake to suppose that large quantities are present in the stomach, for such is not the case, and no more is usually brought up than occurs in simple indigestion. *Intestinal pain* is present and referable to both small and large intestines, but is particularly liable to be marked on the right side where it is of a griping character. When this pain is associated with *visible peristalsis*, a mistaken diagnosis of intestinal obstruction may be made, but the patient is always better when he is constipated and worse when diarrhœa is present. The peristalsis is due to narrowing of the colon in the region of the fistula, which is not an uncommon complication. *Wasting* is constantly present. The *appetite* is not necessarily affected and may be quite good or even excessive.

The X-ray examination in my cases was inconclusive and difficult to interpret. Cases, however, have been described in which the stomach was seen to fill from the colon after an opaque enema, and also in which the meal was seen to pass from the stomach into the colon. These appearances have been found to vary in the same case at different times. *Other methods* of use in gastro-colic fistula have been occasionally successful—for example, the finding of easily recognizable substances in the fæces shortly after ingestion, the recovery of a coloured enema by tubage of the stomach, and the passage of air into the stomach, on insufflation of the rectum, and vice versa.

The *immediate prognosis* after operation on these fistulæ is good—of the thirty-one cases, four had no operation, and all died: one from perforation of another ulcer, another from hæmorrhage, and the other two from inanition. Twenty-seven were operated upon, and of these twenty-one recovered. Of the six deaths, two followed simple exploration of the abdomen, the fistula not being touched.

Mr. W. G. Spencer.

It is unfortunate that there should be so many dogmatic statements, when so little is known about the origin of inflammation and ulceration in the stomach and duodenum. There are, for instance, the signs stated to indicate a duodenal ulcer; it is only in a minority of instances that these serve to distinguish a duodenal ulcer from one at the pylorus, or on the lesser curvature of the stomach; it would appear more likely that the signs are due to pyloric spasm—occult blood is an early sign of cancer of the stomach.

The opener of the discussion laid stress upon an increased formation of hydrochloric acid, but it is difficult to understand how the oxyntic cells can do in disease what they do not in health, when the wall of the stomach has become partly destroyed by inflammation and ulceration. Is not the main question connected with the hyperacidity—that there is a septic fermentation within the cavity of the stomach?

I wish to oppose most strongly the recommendation that gastro-jejunostomy should not be done if the surgeon can find no definite ulceration. The cases in which there is often no evident signs of ulceration are young women who have been unable to keep to their work. They are relieved by rest in bed, but only for a time. After two or three breakdowns a gastro-jejunostomy enables them to continue at their occupation, although they may not be wholly freed from dyspepsia. Indeed they are the cases suitable for gastro-jejunostomy only, whereas duodenal ulcers, ulcers of the lesser curvature, and hourglass stomachs should be excised, whenever the patient can stand the operation.

I have been operating on cases referred to me by medical colleagues at the rate of one or so a month for the last twenty-five years, and I have never been called upon to operate again for jejunal ulcer or to undo an anastomosis. I have always aimed at making the anastomosis directly under the œsophageal opening, where the X-ray photographs show the lowest part of the stomach. I can only suggest that the specimens of jejunal ulcer exhibited were due partly to a continuance of septic infection from ulcers, and partly to the anastomosis being made near the pylorus, not at the lowest point of the stomach. I have followed Petersen's method described at the Paris International Medical Congress in 1900. The jejunum is stitched to the back wall of the stomach, so that it continues in a direct line the horizontal course of the duodenum for 2 in. beyond the anastomosis towards the left. There is then no possibility of a kink.

I have always used fine silk for both rows, and have not cut away mucous membrane. It is important that the margin of the mesocolon should be stitched to the stomach wall, and not to the line of the anastomosis, nor to the jejunum. In the only case I can recall in which I had again to operate, the edge of the hole in the mesocolon had been fixed to the jejunum, which had then been drawn up into the lesser peritoneal sac so as to form a V-shaped kink.

An accident which happened in the case of several weakly patients was the bursting open of the abdominal wall about the tenth day. Instead of inserting the ordinary deep interrupted sutures which have to be drawn tight if the wall is tense, a double loop is made, the two ends are brought out to one side and tied over pieces of rubber tubing. These sutures can be left in for a month or so, and the patients can meanwhile get about.

Dr. Arthur F. Hurst.

My remarks are made from the physician's point of view, with special reference to prophylaxis.

I find myself in agreement with much that has been said this afternoon, but unlike the last speaker, I believe that duodenal ulcer can be diagnosed with a considerable degree of certainty, and that mistakes are very much less commonly made than in the diagnosis of any other abdominal condition. Not only is the clinical history very characteristic, but when doubt exists as to the diagnosis between duodenal and prepyloric ulcer an X-ray examination will settle the question without difficulty. It is somewhat astonishing to be told that the hyperacidity which is so constantly observed in cases of ulcer is due to organic acids produced by fermentation, and not to hydrochloric acid. Surely the least experienced analyst would be able to distinguish between these causes of acidity, and moreover it is easy to prove that gastric fermentation never occurs to an appreciable extent in the absence of pyloric obstruction. I was sorry to hear Mr. Spencer suggest that the ulcer should be excised in cases of hour-glass stomach. This must often be extremely difficult, owing to the large size of the ulcer and the frequency with which it has penetrated into neighbouring organs. On the other hand, the operation of gastro-gastrostomy is comparatively easy, and the results are in my experience uniformly good. I feel much less anxious about the future of a patient upon whom this operation has been performed than about one upon whom gastro-enterostomy has been performed, as there is no danger of any secondary ill-results occurring. I had thought that all surgeons agreed that gastro-enterostomy should never be performed in the absence of gross structural disease of the stomach or duodenum. I was surprised to find that Mr. Spencer took the opposite view. Some of the worst results I have seen following gastro-enterostomy have been in patients for whom the operation was done under these conditions, and the only cases in which I have had to recommend that the anastomosis should be disconnected have been of this nature. Lastly, the dogmatic statement made by Mr. Spencer in a recent review on gastric surgery in *Medical Science* that "everybody now agrees that the only treatment for duodenal ulcer is gastro-enterostomy" requires emphatic contradiction. The vast majority of cases can be cured by adequate medical treatment, particularly if some method of complete neutralization all day and all night like that advocated by Sippey is used. I believe that the necessity for operative treatment of duodenal ulcer will steadily become more rare as the diagnosis is made at an earlier stage, and this treatment becomes more widely adopted.

I quite agree with the speakers who emphasized the importance of studying the original cause of gastric and duodenal ulcer. It is through ignorance on this question that the relapses after medical treatment and many of the unsuccessful results of gastro-enterostomy are due, but I believe that we are already in possession of sufficient knowledge in this direction to be of material help. It is easy enough to cause an ulcer to heal by medical treatment, but if the patient returns to the conditions under which it originally formed, a relapse is almost certain to occur. In the same way if a patient on whom a gastro-enterostomy has been performed is given no instructions as to the precautions he must take in order to remain well, the ulcer may return or a jejunal ulcer may form. I believe that the essential predisposing cause of gastric and duodenal ulcer is inherited, and that certain individuals are born with stomachs, the motor and secretory activity of which is such that they are likely to develop either gastric or duodenal ulcers according to the type of their stomach, under conditions which, in the majority of individuals, would lead to nothing more than ordinary indigestion.

The evidence in the case of gastric ulcer is at present less definite than in that of duodenal ulcer, so I will speak first about the latter. If a large number of normal individuals be examined with the X-rays it will be found that a certain proportion of them have stomachs which are hypertonic; the greater curvature does not reach the umbilicus in the erect position, and they empty themselves abnormally quickly. At the same time they may secrete a juice containing. more than the average quantity of hydrochloric acid, and the secretion often continues after the stomach is empty. As a result of all this the first part of the duodenum is bathed with exceptionally acid material for an exceptionally large proportion of the twenty-four hours. If such an individual develops a source of infection such as septic teeth or chronic appendicitis, or if he bolts his food, has meals at irregular hours, eats indigestible food, if he indulges in alcohol between meals, or smokes excessively, a duodenal ulcer is likely to develop. A hypotonic stomach, which empties slowly and also secretes an excessive quantity of very acid juice, probably predisposes to the development of a gastric ulcer which will form under the same conditions. These types of stomach appear to run in families, and consequently it is not uncommon to find brothers and sisters with either duodenal or gastric ulcers, or with slighter symptoms due to the presence of a pre-ulcerative condition, but I have never seen a duodenal ulcer in one member of a family and a gastric ulcer in another.

After the ulcer has healed the patient still possesses a stomach which predisposes to the formation of an ulcer. It is therefore of the greatest importance that all the exciting causes I have mentioned should be removed, and neither medical nor surgical treatment can be regarded as adequate unless all sources of infection are eradicated, and the patient is given, on the cessation of active treatment, printed instructions as to how to avoid a recurrence of symptoms. If in spite of these a recurrence develops, it is either the patient's own fault for not following the instructions, or it is our fault for giving instructions which are not sufficiently explicit, but as a matter of fact the former is almost always found to be the case. The following is a copy of the instruction I now give to patients at the end of their course of treatment, and I would suggest to surgeons that they might give similar instructions after operation.

Diet to avoid Recurrence of Symptoms.—Avoid alcohol in every form, except, later on, a little light wine or diluted whisky at meals. *Avoid* effervescing drinks. *Avoid* all pips and skins of fruit (whether raw, cooked or in jam, and currants, raisins and lemon peel in cake), nuts and all unripe fruit; for example an orange may be sucked but not eaten, and currants, raisins and figs are particularly undesirable. *Avoid* all raw vegetables, whether taken alone (celery, water-cress) or in salads or pickles; green vegetables are best taken after being passed through a sieve and mixed with butter in the form of a purée: porridge is only allowed if made with the finest oatmeal. *Avoid* vinegar, lemon-juice, sour fruit, soured milk. *Avoid* pepper, mustard, curry, chutney, excess of salt. *Avoid* tough meat, salted fish or meat, pork, twice cooked meat. *Avoid* cheese, except cream cheese; new bread. *Avoid* clear and thick meat soups.

Have some food in the middle of the morning and on going to bed (for duodenal, but not gastric, ulcer).

Eat slowly and chew very thoroughly.

Do not smoke excessively.

If you have the slightest return of symptoms go to bed on a milk diet and alkalies, seek medical advice and do not wait for the symptoms to become severe.

In addition to these general instructions, alkalies should be ordered; but I agree with Dr. Hutchison that sodium bicarbonate is quite the most unsuitable one to give, as although it neutralizes an equivalent quantity of acid it is the strongest of all stimulants to gastric secretion.

If all cases of dyspepsia are thoroughly investigated I believe that it would be possible to institute a real prophylaxis of gastric and duodenal ulcer, as the types of stomach predisposing to these conditions are easily recognizable. Apart from this, early diagnosis and thorough treatment by the complete neutralization method, followed by continued prophylaxis, would make the necessity of operating for ulcer very rare. Lastly, if a jejunal ulcer develops in spite of all precautions the same medical treatment generally meets with complete success.

Mr. Charles Ryall.

The operation of gastro-enterostomy is founded on sound scientific lines, and is as free from immediate risks as any simple abdominal operation, and disappointing after-results are, in carefully selected cases, just as rare as in other abdominal operations. That gastro-enterostomy cures duodenal and pyloric ulcers, and does so rapidly, is beyond argument. I have seen large inflammatory masses surrounding ulceration disappear, as well as the ulcer, in the course of two or three weeks. Gastro-enterostomy cures ulcers at the pyloric end of the stomach, but I am inclined to excision as well as to short-circuiting in ulcers away from the pylorus. The fact that some surgeons meet with gastro-jejunal ulcers following operation more frequently than others do can scarcely be due to lack of observation, and therefore it would be interesting to know if there is any difference in their respective techniques. Does this ulceration occur more frequently where the opening is small, or does it follow any particular method of suturing? Union so rapidly takes place, no matter how the suturing is applied, that surgeons naturally fall back on the quickest method of application. The two-layer line of suturing is most in vogue, all the coats of the stomach being united by the inner suture. I do not favour this bunching together of all the coats of the stomach, as it does not tend to accuracy of apposition of the mucous membrane; I consider that better results may be obtained by three-layer sutures, a separate one thus being used for the accurate apposition of the mucous membrane. As to the use of thread, I agree with Mr. Paterson that the theory of its being a cause of gastro-jejunal ulceration is unproven, though I myself use catgut for suturing the mucous membrane.

In my opinion simple ulcers of the stomach and its outlet are due to hyperacidity, and this hyperacidity is cured and the ulceration disappears as a result of short-circuiting; nevertheless, there is no reason why fresh ulceration should not take place, either in the stomach or its new outlet, should hyperacidity recur and remain unchecked.

Mr. R. H. Jocelyn Swan.

I am in entire accord with Dr. Hurst when he speaks of the difficulty in diagnosis of the true gastric ulcer, and I must urge most strongly that the operation of gastro-enterostomy should not be performed unless some lesion of the stomach or duodenum or the existence of pyloric stenosis can be demonstrated at the operation. If nothing is obvious by palpation of the stomach and duodenum from the outside, I make it a rule to incise directly the anterior gastric wall and examine the interior, and especially the pylorus, with the finger. I have also found this procedure of great value in deciding whether a thickened infiltrated mass in the gastric wall is due to a thickened ulcer and to malignant disease. When no demonstrable lesion is found in the stomach or duodenum gastro-enterostomy should not be performed, but search made elsewhere for disease of other organs which may give rise to the symptoms, such as chronic appendicitis, gall-stones, tuberculous disease of the small intestine, and bands of adhesions about the colon.

With regard to the technique of the operation, I always use very fine linen thread (No. 120, Barber's), and practically always do the anastomosis by means of three layers of suture. I remove no redundant mucous membrane, but pleat it up with the innermost layer of suture, obtaining in this way better hæmostasis and better co-aptation of the edges of the mucous membrane. I have seen no reason for using catgut instead of thread, in fact I still think fine thread much better than catgut. I use no loop of jejunum, sew the mesocolon to the gastric surface around the anastomosis and also hold up the jejunum on the distal side of the anastomosis by suture to the mesocolón. I prefer a stoma of about 2½ in.

In cases of duodenal ulcer I have made a practice of infolding the ulcer, but in my more recent cases of gastric ulcer, especially if they are of the indurative, chronic variety, I prefer a partial gastrectomy to a localized excision of the ulcer. I was led to do this by the excellent results of pylorectomy for large thickened ulcers, combined with anastomosis to the jejunum.

No one can deny that in apparently suitable cases the operation of gastro-enterostomy is followed by excellent results, as both Mr. Paterson and Mr. Sherren have shown in their long series of cases. Yet at the same time a small number of cases remains in which the results are disappointing. In one case, on re-opening the stomach two years after performing gastro-enterostomy for a small prepyloric ulcer, I found the latter healed, but another ulcer on the lesser curvature. The anastomosis was fully patent and I could find no sign of any thread suture. In a second case I found a small jejunal ulcer in the efferent part of the jejunum 2 in. distal to the gastro-enterostomy opening. I have seen no case of ulceration at the margin of the anastomosis. I cannot venture upon any opinion as to the causation of recurrent ulceration, but I think that possibly some common factor may remain which was responsible for the original ulceration.

With regard to Mr. Wright's suggestion that sepsis of an ulcerated surface in the stomach may be a cause of subsequent ulceration after gastro-enterostomy—a suggestion that implies that a direct attack should be made upon the ulcer in addition to an anastomosis—I may mention that I have performed many gastro-enterostomies, as a palliative measure, on cases of carcinoma of the pyloric end of the stomach, that have been too advanced for radical removal: in no case have I seen any subsequent ulceration about the anastomosis or in the jejunum in subsequent examination.

The specimen I show to-night is interesting. A man, T. O., aged 36, was operated upon by me on January 31, 1909, for pyloric stenosis due to cicatricial contraction of an old healed ulcer. He improved so much that he put on 3 st. in weight during the next two years. I saw him again in August, 1918, when he had return of vomiting and I found a hard mass in the epigastrium. On exploration an extensive carcinoma was found occupying the pyloric end of the stomach extending to the edge of the anastomosis with metastases in the liver. The specimen was subsequently removed after death in November, 1918. This case is an instance of the incidence of carcinoma in a healed gastric scar, and it becomes an interesting speculation as to whether this should influence us in advising partial gastrectomy in similar cases.

Mr. A. J. Walton.

My experience of the remote anatomical and pathological results of gastric surgery, and more especially of gastro-enterostomy, is very similar to that of Mr. Sherren, and may I think be regarded as representing the teaching of the London Hospital. It is nevertheless of considerable interest to note, that although my knowledge of the technique of these operations was to a very large extent learnt from Mr. Sherren, the working arrangements as carried out in our London hospitals has prevented a close collaboration of our work since I was appointed an assistant surgeon. Owing to the stress of such work there has been but little opportunity of discussing details with him for nearly seven years and our technique has diverged in certain minor details. The fact that our results, although absolutely independently arrived at, so closely agree is I think strong evidence of their correctness.

My remarks will be based almost entirely upon my own personal experience, which embraces a series of 335 cases of gastro-enterostomy and other operations performed for conditions other than carcinoma of the stomach. Time will not permit me to quote any of the reports from the voluminous literature upon the subject which may support or oppose these views. Those cases in which patients were suffering from carcinoma of the stomach when first seen have been purposely omitted, for the nature of this disease will so affect the results of the operation that they cannot be fairly compared with the operations performed for benign conditions.

The total number of operations is 335 of which 321 were personally treated from the beginning. Of these 321 cases nine required further operations: three for the onset of carinoma, three for the presence of persistent ulcers, and three for gastro-jejunal ulcers; that is to say, 2·8 per cent. required further operative treatment. Every case has been followed as carefully as possible and very few have been lost sight of, so that it is probable that this percentage is fairly correct. The remaining fourteen cases had had their first operation performed by some other surgeon and therefore it is impossible to estimate what percentage of recurrence they represent. It is doubtful whether the subsequent clinical history is in any way relevant to the present discussion, but my own figures, as well as an independent investigation carried out on my cases by a post-graduate student, have shown about 80 per cent. complete recoveries. The post-operative difficulties may be best classified as follows:—

DUE TO ERRORS OF TECHNIQUE.

(A) *The Operation is unjustifiably performed.*—It not infrequently happens that a gastro-enterostomy is performed without any clear evidence of the presence of gastric or duodenal disease. A long list of the diseases in which this error may be made has been given by Sir Berkeley Moynihan [6]: but the disease upon which I chiefly wish to lay stress is visceroptosis. Not only may the symptoms of this condition closely simulate those of a gastric ulcer, as I have already pointed out [8], but at operation local areas of spasm may be seen which appear to be mistaken for chronic ulcers by those not conversant with gastric surgery. A gastro-enterostomy will not only give no relief but will often be followed by an aggravation of the symptoms. There is one exception to this statement and that is an acute ulceration giving rise to severe hæmorrhage which may be found with visceroptosis in young women. Under such circumstances a gastro-enterostomy may be of great value, but even here it has been my experience that it will be of no value unless combined with pyloric occlusion. In the other cases there will be a rapid return of the symptoms and on examination the patient will present clear evidence of ptosis. There have been two such cases in my series, and in both it was found necessary to operate again and to close the gastro-enterostomy opening.

(B) *There has been Gross Lack of Anatomical Knowledge at the First Operation.*—It occasionally happens that a patient presents a clear past history of a gastric or duodenal ulcer for which an operation has been performed but no relief has followed. If in spite of this the symptoms are clearly defined further operation should be advised. On three occasions I have found that grave errors have been made in performing the previous operation. In two cases a junction of the stomach had been made with the colon instead of with the jejunum; in the third case an anastomosis had been performed with the ileum 12 in. from the ileoc-æcal valve. Under such conditions the symptoms of a fæcal fistula into the stomach might be expected to arise, but in all three of these cases the opening had been so badly made that it was too small to function, and the patients may thus be said to have survived because there were two errors instead of one. Their symptoms were simply those of persistence of the ulcer.

(C) *There is some Obstruction of the Afferent and Efferent Loops.*—This is a condition which is now but rarely seen. It is very probable that the cases of "pernicious vomiting" or "the vicious circle" so frequently described in the older days, were really due to this cause. Four different varieties of the obstruction may be recognized:—

(1) *There is too long a Loop.*—With a long afferent loop there will be a tendency for the intestine to drag upon the stomach at the junction so that the afferent and efferent loops become parallel, and a sharp bend with kinking is formed. This is especially likely to happen with an anterior gastro-enterostomy, if the antecolic method be used. The difficulty can easily be overcome by making use of Sherren's retrocolic method when it is necessary to perform an anterior gastro-enterostomy. In my own series there was only one such case, the patient having been operated upon elsewhere two years before for a perforated duodenal ulcer. Not only was there a distinct loop, but the afferent and efferent loops had become united by adhesions.

(2) *There is too short a Junction.*—If too small an opening be made the attachment will become so narrow that a kink will occur somewhat similar to that occurring in the last group of cases, and hence the afferent and efferent loops will become approximated and the passage will not be clear. This occurred in one of my cases, an operation having been performed at another hospital three years before for a duodenal ulcer. There was a posterior gastro-enterostomy with a small opening; the efferent loop sagged downwards and thus narrowed the entry.

(3) *There is a Constriction by the Opening of the Mesocolon.*—It has always been advocated that the mesocolon be sutured to the junction in the belief that if this step were not carried out a hernia of the small intestine might take place through the opening. This complication is,

however, a rare one, but if the suturing be not performed there is a much more common complication; the ring of the mesocolon will slip downwards and will approximate the afferent and efferent loops so that they will come to lie parallel and will thus be kinked. A somewhat similar complication may arise in fat patients, even if the mesocolon be sutured to the junction. Here the lower leaf of the mesocolon may constrict, and owing to the thickness of this structure the constriction may lie well below the junction and thus approximate the loops. Mayo has well recognized this difficulty, and advocates that in stout patients the mesocolon should be sutured to the posterior wall of the stomach some distance from the junction. Such a complication occurred in one of my own cases. Four weeks after an operation for a duodenal ulcer he began to develop signs of dilatation of the stomach, with marked vomiting; a second operation was performed, and a thickening of the lower leaf of the mesocolon was found which had caused the kinking. The constriction was divided and a lateral anastomosis performed. The patient has now remained free from symptoms for a period of over three years.

(4) *There is Axial Rotation.*—If the jejunum be rotated upon its long axis while the junction is being made symptoms of obstruction will develop almost at once. Such a case has not come under my personal observation but it has been described by Sir Berkeley Moynihan [6].

(D) *The Opening has been badly Planned.*—It would hardly be necessary to-day to mention the use of mechanical devices were it not for the fact that within the last few weeks a so-called new operation of partial gastrectomy has been described, in which it is advocated that a gastro-enterostomy be performed by Murphy's button. It is well realized, however, that such devices are often retained *in situ*, that they leave too narrow an opening, that they often cause hæmorrhage, that they are followed by ulceration and constriction, and that their insertion cannot be carried out more quickly than the safe method of an anastomosis by suture. As late as 1917 two cases of stenosis after the use of the button were described by Lewisohn [3]. The faults which are more likely to be found to-day in the formation of the opening are:—

(1) *The Opening is too Small.*—This is a common fault with those who are unused to performing the operation. In all three cases described above, in which the junction had been made with the colon or ileum, the opening was so small that it failed to act. In one other

case an anterior gastro-enterostomy had been performed elsewhere eight years ago for a pyloric ulcer. The symptoms disappeared but returned again. At the second operation the pyloric ulcer was healed, and therefore the opening had at first probably been large enough, but it was now so small that it would not have admitted an ordinary lead pencil, and there was a second ulcer on the lesser curve. To allow for constriction in the healing the opening should be at least 3 to 3½ in. long when the intestine is held between the clamps—that is to say, its circumference should be from 6 to 7 in.

(2) *The Opening is too High.*—In a small number of cases it is found that ill-defined symptoms follow the operation. These last but a short period and may as a rule be entirely controlled by medical treatment. Some of these will show by the X-rays that the opening is relatively high up on the greater curvature, and it is possible that this may be the cause of the symptoms. It has, however, never been my personal experience to meet with any that have required operation for this cause alone; nor does one find that any symptoms follow in those cases where the opening has purposely been made relatively high up, as in treating an ulcer of the lesser curve or an hour-glass stomach. Trouble is much more likely to follow if the opening be made away from the greater curvature. However the opening is placed, whether it be vertical, retroperistaltic or isoperistaltic, the lower portion of the opening should reach to the greater curvature.

(3) *The Posterior Wall has been united to the Anterior Wall.*—This is never mentioned as one of the difficulties in the operation, but I have always been struck with the ease with which it would be possible to pick up the posterior wall in suturing the two anterior layers, and have always taken care to avoid this pitfall. In one case in which the symptoms returned eighteen months after an operation by another surgeon the X-rays showed that the opening was not working well, and at operation the anterior and posterior walls were found to be united over a considerable area near the centre.

(4) *The Opening is wrongly placed in regard to the Constriction.*—The commonest fault that is made here is that an hour-glass stomach is overlooked when it is associated with pyloric obstruction, and a junction is made between the jejunum and distal pouch; thus, of course, failing to overcome the obstruction in the middle of the stomach. The reverse of this may sometimes happen. With a double constriction an opening is made into the proximal pouch alone. In one of my cases an operation had been performed five years previously, and his symptoms then

returned. It was found at operation that this mistake had been made and there was great distention of the pyloric pouch.

(E) *Gastro-jejunal and Jejunal Ulcers arise.*—We owe our knowledge of the distinction of these two conditions to Paterson [7], but there is still a certain amount of discussion as to the cause of the ulcer.

(1) *Gastro-jejunal Ulcer.*—My own experience strongly supports the view that this form of ulceration is due to the presence of a piece of silk suture. In my own series the three cases that followed a gastro-enterostomy performed by myself all occurred when I was in the habit of using silk sutures for the outer layer, and in every one of them I was able to find the suture at the second operation. A similar suture was found in three out of the four cases in which the first operation had been performed by other surgeons. This view has been supported by Mayo [4], who found the suture in five out of eight cases, and also by Woolsey [9] and Ginsburg [2]. Since I have been wholly using catgut I have never had an example of this condition. That the increase of acid is also a factor is, I think, shown by the fact that in all of my cases the original operation was for a pyloric ulcer, in which condition the free HCl is much higher than with a lesser curve ulcer. The ulcers all show a curious similarity in that they tend to commence either anteriorly or at one extremity; and that they tend to become adherent and to perforate into the colon. I believe that their method of production is as follows: As the first layer of through-and-through catgut suture is approaching its termination the mucosa is hidden from view and slips out of sight so that the through-and-through suture picks up only the muscular and peritoneal coats. When the silk embedding suture passes over this surface it will not be covered by mucosa and a raw surface will be left, in the base of which is an unabsorbable foreign material, and under the influence of excess of acid this gives rise to a chronic ulcer. Such an ulcer may be prevented by taking special care to include the last piece of mucosa in the through-and-through suture and to use a catgut suture for the embedding layer.

(2) *Jejunal Ulcer.*—In my own series I have not yet met with a true jejunal ulcer, and many of the specimens I have seen described as jejunal ulcers are continuous with the gastro-jejunal junction. My belief based upon my present experience is, therefore, that the majority of these ulcers have spread from the gastro-jejunal junction. A true jejunal ulcer appears to be very rare, and it is doubtful if it ever occurs when the anastomosis is functioning correctly. It would also seem doubtful whether such an ulcer might be caused by the trauma due to the use of

clamps for we have very definite knowledge that acute ulcers of the stomach tend to heal rapidly, and, therefore, one would assume, that such a lesion in the surface of the jejunum would also heal if no foreign material were present.

(F) *There is a Hernia through the Mesocolon.*—It is to prevent this complication that the advice is always given that the mesocolon be sutured to the junction. Such a complication seems, however, to be rare, and it has not occurred in my own series. As already stated, I believe that this method of suturing is of more value in preventing slipping of the mesocolon than in preventing such a hernia. Moschcowitz [5] in reporting a case of this complication was only able to find seven others recorded in the literature.

COMPLICATIONS AT PRESENT UNEXPLAINED.

(A) *There is a recurrence of the Ulcer.*—Two groups of this complication may be recognized:—

(1) *Due to Faults of Technique as described above.*—In my own series where I performed the first operation this occurred on one occasion. After excision of the ulcer on the lesser curve where a gastro-enterostomy was not performed symptoms returned after five months. At operation a posterior ulcer was found at the lower part of the resection and in the base of the ulcer a portion of silk was found, so that in this case a recurrence was due to causes similar to those producing a gastro-jejunal ulcer. If a perforated gastric ulcer is sutured and no gastro-enterostomy is performed the ulcer is likely to persist or recur. There were two such cases in my series.

(2) *Occasionally when the Technique seems good an Ulcer may recur.*—I have not had one of these in my own series of first operations, but in four others when the first operation was performed by another surgeon there was a recurrent ulcer. In one the gastro-enterostomy was anterior and the opening was rather too small, but in the other three it was posterior and appeared quite satisfactory. In all four the original ulcer had been a pyloric one and had healed, but a new ulcer had formed at the lesser curve. A somewhat similar case has been reported by Cunning [1] in which the second ulcer was on the posterior surface and at some distance from the gastro-enterostomy opening. The explanation of this re-formation is very difficult to see, but it is possible that the ulcers were present at the first operation and were overlooked, and that like the next group they were not cured by a simple gastro-enterostomy.

(B) *There is a Persistence of the Ulcer.*—It is probable that an ulcer high up on the lesser curve will not be entirely cured by a posterior gastro-enterostomy, and that it will be necessary either to excise the ulcer or to perform a partial gastrectomy. In certain cases the ulcer may be too large and may be too adherent for this to be attempted; under these circumstances the pylorus should be occluded and a gastro-enterostomy performed. In two of my cases this has had to be done and in each of them relief followed but symptoms returned in about eighteen months. The second operation disclosed the fact that the ulcer had become much smaller and was capable of being excised, this step being followed by a complete cure. It is now my custom when the ulcer cannot be removed to tell the patient that his symptoms will probably recur in about eighteen months, but that a second operation will probably lead to a complete cure.

(C) *The Onset of Carcinoma.*—In my experience the percentage of cases in which this has happened is very low, but this may possibly be due to the fact that I excise all lesser curve ulcers. On several occasions a partial gastrectomy has been performed for a pyloric ulcer because one or other detail in the clinical history has suggested the possibility of carcinoma, and on microscopical section a small patch of carcinoma has been seen. It is therefore possible that where carcinoma has apparently supervened after operation it may have been present in the original ulcer. In my own series there were four cases of the onset of carcinoma after operation. In two of these it is very probable that the growth had been present at the first operation, but had been overlooked. In both of the others a carcinoma followed two years after an excision of an ulcer on the lesser curve.

It is often somewhat loosely stated that the results of operative treatment for ulcers of the stomach and duodenum do not give satisfactory results, but apart from such complications which the above figures show to be relatively rare, my experience has been that these patients progress very favourably. It is now my custom to see all cases in a "follow through" department. The equipment of such a department has given me opportunities of keeping well in touch with all my patients and since it has been started I have lost sight of less than 1 per cent. of them. Time will not permit me to analyse the after-results in detail, but my experience has been that some 80 per cent. of patients with gastric or duodenal ulcers are completely cured of their symptoms. Of the remainder, 2·8 per cent. have one of the lesions described above which require operative treatment, while the rest show

TABLE OF CASES OF GASTRIC AND DUODENAL ULCERS. TOTAL NUMBER OF CASES, 335.

	Total	Excision only	Excision and gastro-enterostomy	Gastro-enterostomy only	Gastrectomy	Multiple	Gastric and duodenal	Died	Gastro-jejunal ulcer	Persistent ulcer	Carcinoma	Obstruction of loops
Lesser curve non-adherent	48	2	36	6	3	7	4	1	—	—	2	—
Lesser curve adherent ...	54	—	37	14	2	2	1	3	—	2 (after gastro-enterostomy only; excision later)	1	—
Pyloric; no dilatation ...	42	—	—	37	4	—	—	2	3	—	1	—
Pyloric, with dilatation	18	—	—	18	—	—	—	—	—	—	—	—
Hour-glass	16	2	12	1	1	—	—	2	—	1 (after excision only)	1 (before operation)	—
Lesser curve after gastro-enterostomy	*4*	4	—	—	—	—	—	—	—	—	—	—
Severe hæmatemesis, chronic	2	—	1	1	—	—	—	—	—	—	—	—
Severe hæmatemesis, acute	16	—	—	12	—	—	—	6	—	—	—	—
Recurrence after perforation	*2*	—	1	1	—	—	—	—	—	—	—	—
Perforated gastric ulcers	31	—	—	25	—	—	—	13	—	—	—	—
Duodenal ulcers, chronic	68	—	—	66	—	—	—	4	—	—	—	1
Duodenal ulcers, acute ...	4	—	—	1	—	—	—	1	—	—	—	—
Duodenal severe hæmatemesis	1	—	—	1	—	—	—	—	—	—	—	—
Duodenal, perforated ...	17	—	—	16	—	—	—	5	—	—	—	—
Gastro-jejunal ulcers ...	3, *4*	—	7	—	—	—	—	1	—	—	—	—
Gastro-enterostomy faulty	1, *6* *	—	—	—	—	—	—	—	—	—	—	—
Total ...	338†	8	94	199	10	9	5	38	3	3	5	1

* 1, loop too long; 1, junction too short; 1, constriction of meso-colon; 3, opening made with incorrect piece of intestine; 1, posterior wall united to anterior.

† This figure does not correspond with that of the total number of cases as three patients had more than one complication.

The italic figures indicate that in these cases the primary operation was performed by another surgeon.

but ill-defined attacks of dyspepsia which are easily controlled by short courses of medical treatment. That is to say, that these cases give results as good or better than those to be obtained by practically any other surgical procedure.

REFERENCES.

[1] CUNNING, J. *Lancet*, 1914, i, p. 1108.
[2] GINSBURG, N. *Ann. of Surg.*, June, 1916, p. 732.
[3] LEWISOHN, R. *Surg., Gyn. and Obstet.*, November, 1917, p. 505.
[4] MAYO, W. J. *Boston Med. and Surg. Journ.*, January 29, 1914.
[5] MOSCHCOWITZ, A. V. *Surg., Gyn. and Obstet.*, September, 1915, p. 390.
[6] MOYNIHAN, Sir B. *Brit. Med. Journ.*, 1919, ii, p. 33.
[7] PATERSON, H. J. "The Surgery of the Stomach," 1914.
[8] WALTON, A. J. *Brit. Journ. of Surg.*, 1915, ii, No. 10.
[9] WOOLSEY, G. *Surg., Gyn. and Obstet.*, November, 1917, p. 481.

Mr. H. J. PATERSON (in reply).

I have really no remarks to add to the discussion. I would just say it was in order to ascertain the various opinions of those who might be present that the discussion was initiated, and we have had a very free and helpful interchange of ideas.

I would have defended myself, if Dr. Hurst had not already done so, against the charge of mistaking organic for inorganic acids: the one ought not to be mistaken for the other. No fermentation in the stomach will produce hydrochloric acid. I was very glad Dr. Hurst maintained that the amount of free hydrochloric acid can be influenced by diet, as I was greatly staggered by the opposite opinion of a physician on that point. I was of the opinion that by a purely milk diet one could reduce very materially the free hydrochloric acid present in the gastric contents.

I agree with Dr. Hurst — and I regard it as one of the most important points brought out in this discussion—that, as I said in my opening remarks, gastro-jejunostomy is simply an incident in the treatment of gastric and duodenal ulcer. Unless you give clear instructions to patients about their diet, and watch them and see that they carry them out, it is better—and I am sorry to be dogmatic again —not to do the operation at all. I feel certain that the real crux of the failures in those cases which have remained well for a certain time only, after operation, is the failure to give and to have carried out suitable detailed and definite instructions.

The PRESIDENT.

I congratulate the Section on the interesting facts brought out in the discussion, and I thank those who have taken part in it and have exhibited specimens. For once I am glad to find that the physicians are with the surgeons, even in cases in which operations have failed. Twenty years ago it was a common saying that a good teacher of surgery could interest his class with a chronic ulcer of the leg. It now requires a jejunal ulcer to arouse surgical enthusiasm.

Section of Surgery.

President—Sir John Bland-Sutton, F.R.C.S.

EXHIBITION OF SPECIMENS OF LEATHER-BOTTLE STOMACH AND INNOCENT TUMOURS OF THE ALIMENTARY TRACT.[1]

CATALOGUE OF EXHIBITS.

(I) *From the Museum of the Middlesex Hospital.*

Leather-bottle Stomach.

(1) A stomach, spleen, and part of the transverse colon. The stomach exhibits the condition known as linitis plastica (leather-bottle stomach). The organ is greatly shrunken, and its walls are much thickened. The thickening is uniform from the cardia to the pylorus, at which point it abruptly ceases, the duodenum being thin walled and of normal calibre. All the coats of the stomach participate in this sclerosis. The gastrocolic omentum is also thickened and is so contracted that the transverse colon is drawn tightly up to the greater curvature of the stomach. The transverse colon itself is the seat of similar changes to those in the stomach. The spleen is also tightly apposed to the fundus of the stomach but is not affected in the same way as the stomach and transverse colon. The condition is one of diffuse carcinomatous infiltration of the stomach and transverse colon. (Sir J. Bland-Sutton, "Tumours," 1917.)

Fibroid of Cæcum.

(2) An ovoid tumour which was springing from the wall of the cæcum. A whorled appearance is seen resembling the cut surface of a uterine fibroid. From a woman, aged 48. The cæcal tumour was found during the course of an operation for the removal of an ovarian cyst. There were no symptoms

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[1] At the Annual Meeting of the Section, held on May 5, 1920.

attributable to the cæcal tumour. Microscopically it exactly resembles a "fibroid" of the uterus.

Melanosis of Cæcum.

(3) The cæcum and appendix and part of the ascending colon from a woman, aged 63, showing a condition of pigmentation of the submucous connective tissue probably of hæmatogenous origin. The pigmentation starts abruptly at the ileocæcal valve, and spreads throughout the cæcum and colon and to a less extent into the appendix, but not at all into the ileum.

Polypus of Colon.

(4) A portion of large intestine. The mucous membrane is thickly studded with outgrowths, some forming simple rounded elevations, others stalked processes varying in length from $\frac{1}{4}$ in. to 1 in., with club-shaped ends. In many places the ends are branched, and in others the ends of neighbouring outgrowths are united together so as to form an irregular meshwork. They extend from above a cicatrix which was situated 3 in. from the anus to within a short distance of the ileocæcal valve. From a man, aged 46, who died in the hospital, March, 1863. He had a phagedænic ulcer of the foot, and suffered from diarrhœa and hæmorrhage from the bowels for three years.

(5) A portion of the rectum turned inside out to show multiple papilloma. The mucous membrane is seen to be thickly studded with pedunculated branched papillomata. Some are complex and racemose, others are simpler, terminating in a rounded knob-like extremity. A few are sessile mammillary elevations on the surface of the mucous membrane. One or two submucous hæmorrhages are to be seen. The open mouths of many mucous glands are to be seen. From a man aged 36, upon whom an abdomino-perineal excision of the rectum was performed. His father had died of carcinoma of the rectum while his brother had suffered from a rectal growth. The patient was under the care of Mr. D. Ligat. He survived the operation two years and died of acute pneumonia.

Dermoid of Rectum.

(6) A dermoid cyst attached to the rectum.

Fibroid of Jejunum.

(7) A portion of jejunum showing an intussusception caused by a pedunculated tumour springing from the wall of the gut. Near the neck of the tumour is a small abscess cavity. The growth in section is a fibroma.

(8) Portion of the pelvic colon. A polypus, in shape resembling a large ripe blackberry, hangs by a stalk 6 cm. in length, from the mucous membrane. The stalk is a flattened band of the mucous membrane, and the head of the polypus is crowded with intestinal glands.

(II) *From the Museum of the Royal College of Surgeons.*

(9) Segment of ileum with a tumour shown in section. The tumour is a fibroid and the tissue is whorled. Excised from a man on account of acute volvulus.

(10) Portion of the cæcum and the termination of the ileum. A lipoma grows within the folds of the ileocæcal valve and forms a ring of fat around the opening. Successfully excised from a woman aged 50, who suffered from constipation many years.

(11) Lipoma weighing 2 oz. This tumour was successfully excised from the ascending colon of a man aged 44, who had suffered repeated attacks of acute intestinal obstruction. In the course of the operation a dimple was noticed in the serous coat of the colon over the site of attachment of the tumour.

(III) *From the Museum of the Westminster Hospital.*

Pedunculated Tumour of the Stomach.

(12) From a man, aged 37, who three months before admission had had some abdominal pain and "indigestion." Seventeen days before admission he had severe pain in the back and abdomen, with dizziness. Melæna was noticed next day, and three days after he vomited blood. He next became delirious, and greatly collapsed, in which condition he was admitted. He did not regain consciousness, and died four days after admission. At the post-mortem the stomach was found to contain a pedunculated tumour the size of a golf ball, which was attached to the middle of the posterior wall. On this tumour were three purple-black depressions (from which the hæmorrhage had probably occurred), and the peritoneal coat over area of the tumour indicated a hæmorrhage beneath. The tumour was encapsulated, and histologically consisted of fairly coarse fibrous tissue, in which were a few unstriped muscle fibres and numerous cells of varying shapes (spheroidal, polyhedral, and small multinucleated). *A fibroma*, or, possibly, fibrosarcoma of very low malignancy.

Fibroma of Stomach.

(13) Portion of the fundus of the stomach, with a polypoid mass projecting into the lumen. The growth is sausage shaped, measuring 4 in. in length and about 1½ in. in diameter. It is covered by normal mucosa, except at the summit, which is ulcerated, and dips into a depression. Microscopically it is a soft fibroma, apparently arising in the submucosa, being separated from the mucosa by the muscularis mucosæ. From a woman, aged 59, who for eight months had suffered from pain and vomiting after food, but not from hæmorrhage at any time. In the left epigastrium there was much tenderness, and at times a tumour, movable and moving, with respiration, could be felt in the neighbourhood of the umbilicus.

Papilloma of the Stomach.

(**14**) A small piece of stomach wall, showing the presence of a pedunculated papillomatous growth, the size of a walnut, which projects into the lumen of the organ. The papillary processes are short and thick. There is no ulceration, and very little inflammatory infiltration. Microscopically the growth does not appear to be malignant. From a woman, aged 55, who died in hospital with primary carcinoma of the sigmoid flexure, with deposits in liver and adrenals. All these were examples of columnar-celled carcinoma. Single non-malignant polypi have been found on several occasions in the stomach in cases of malignant disease lower down the alimentary canal.

(**15**) A stomach showing at the pylorus a firm-walled cyst as large as a small hen's egg. The organ is not dilated, nor is the pylorus distorted, showing that no obstruction was caused. From a child, aged 6, who died from perforation of a typhoid ulcer in the ileum. The cyst contained glairy fluid, probably from retention in one of Brunner's glands.

Cyst of the Duodenum.

(**16**) The pylorus with the neighbouring portions of the stomach and duodenum. A thick-walled cyst, the lining mucosa of which is smooth, is seen arising from the first part of the duodenum. From a man, aged 44, who was admitted giving a three months' history of occasional pain and vomiting two hours after food. No hæmatemesis. Gastro-jejunostomy was performed. At the subsequent autopsy many adhesions were found about the portal fissure of the liver, and these probably contributed to cause the duodenal condition. The gall-bladder formed part of the wall of the cavity, which, arising just beyond the pylorus, extended down to and below the pancreas. Microscopically, the wall of the cyst is that of normal duodenal mucosa, and there is no evidence of malignancy.

A Form of Leather-bottle Stomach.

(**17**) The stomach, with part of the œsophagus and duodenum. The wall of the stomach is almost entirely replaced by a firm malignant growth. The cavity has been greatly reduced in size. The histology is that of squamous-celled carcinoma. From a woman, aged 34: History of indigestion for two to three years. At the autopsy a large mass was found to be present in the liver, and there were deposits in the abdominal glands. Microscopically, the growth was much degenerated, but parts were present that left no doubt as to its true structure.

Pedunculated Tumour of the Stomach.

(**18**) From a woman, aged 50, who up to eleven days, prior to admission, had been quite healthy, and had never had any gastric symptoms. She was admitted profoundly collapsed, and stated that the onset had been sudden, with hæmatemesis and melæna. Her condition was considered to be one of gastric ulcer, and was too grave to allow of operative measures being attempted. Death occurred three days after admission. At the post-mortem, 2 in. from the pyloric ring, a little anterior to the greatest curve of the stomach, a sessile spherical tumour, the size of a golf ball, was found. On the summit was a brownish-black depression. The guts contained blood, and the rest of the viscera showed pallor only. Histologically, the tumour was encapsulated, and composed of an admixture of fibrous tissue and unstriped muscle elements. It should be classified as a *fibro-leio-myoma*.

(IV) *From the Museum of the Royal College of Surgeons.*[1]

(19) Submucous fibroma, the shape and size of a kidney, which was encapsuled in the posterior wall of the middle of the stomach: it weighed 200 grm. (7 oz.). A hospital nurse, aged 46, incapacitated on account of dyspepsia and anæmia, presented two movable kidneys and a reniform tumour, freely movable up and down behind the umbilicus. The tumour was shelled out from the submucous layer of the posterior wall after the cavity of the stomach had been laid open through the anterior wall, in the belief that the mass was a hairball. Both kidneys were, in addition, fixed in place, and the patient recommenced work three and a half months afterwards.

(V) *From the Museum of St. Thomas's Hospital.*

(20) Half a coarsely lobulated submucous lipoma removed from the ileum. In the pedicle is a small portion of the proper muscular coat of the bowel, slightly prolapsed by the traction of the tumour. Over the most prominent part of the growth the mucous membrane has been destroyed by ulceration. Male, aged 32. On March 5, 1894, he had a sudden attack of acute pain in the lower part of the abdomen. A rounded movable swelling was felt. Exploratory cœliotomy revealed an intussusception of the ileum, and when this was reduced the tumour was felt within its lumen. The bowel was opened and the tumour was removed.

(21) An oval lipoma removed from the interior of the sigmoid flexure. The chief part of the growth is covered with mucous membrane, a circular defect at the back of the preparation indicating the position of the divided pedicle. On the more prominent part of the tumour the mucous membrane has been destroyed by ulceration. Male, aged 36. He was admitted to St. Thomas's Hospital suffering from abdominal pain, tenesmus, passage of blood *per rectum* and absolute constipation. No vomiting nor abdominal distension. The bowel was opened and the tumour removed by the late Mr. W. Anderson.

Teratoma of Sigmoid Flexure.

(22) An oval tumour removed from the rectum. It has a double pedicle, by which it was attached to the sigmoid flexure, and is composed of fat containing one or two processes of new bone, and is covered with hairy skin. A piece of glass rod has been placed in the cavity of what was probably a small abscess.

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[1] W. G. Spencer, *Proc. Roy. Soc. Med.*, 1909 (Sect. Surg.), ii, p. 313.

(23) A pedunculated adenoma removed from the rectum of a child, aged 6½.

(24) An oval papilloma removed from the rectum of an adult female by Sir George Makins.

(25) A vertical section of a piece of small intestine. Into its interior, at the side of the attached border, there projects a pedunculated tumour, about the size of a walnut. Microscopically, this is a spindle-celled sarcoma, with nuclei of striking size in many of the cells. The character of the cells suggests that a malignant change has taken place in what was originally a myoma. From a woman, aged 45, with a history of abdominal pain and distension for three weeks and vomiting for forty-eight hours. At the operation it was found that the tumour had caused an intussusception of the small bowel. After reduction, a portion of the bowel to which the tumour was attached was resected by Mr. E. C. Stabb.

(26) A vertical section of a small portion of the sigmoid flexure, successfully removed by the late Mr. Bernard Pitts, for the growth of a large papilloma. The muscular wall of the coat, though displaced by traction, is not invaded by any extension of the growth. More than three years later, death occurred from rupture of the intestine during straining. An examination showed a growth, similar to that which had been excised, projecting through a small rent in the bowel. Microscopically, the recurrent growth was also a simple papilloma.

(27) A lowly lobulated oval tumour, of firm fibrous tissue, about 5 in. in chief diameter, which was excised by Mr. Nitch, together with a piece of the small intestine, from the subperitoneal tissue of the free border of which it has grown. From a girl, aged 11, who had suffered for three weeks with abdominal pain, constipation and occasional vomiting, the subacute obstruction being due either to drag or torsion of the bowel. The tumour was felt in the abdomen just below the umbilicus, and was hard and freely movable. At the operation the mesentery was seen to contain caseous lymphatic glands, and there was free fluid in the peritoneal cavity. After resection, the continuity of the bowel was restored by axial anastomosis, and the patient made an uninterrupted recovery.

(VI) *From the Museum of Guy's Hospital.*

Myoma of the Œsophagus.

(28) A portion of an œsophagus, showing an oval tumour projecting into the lumen of the tube about 2 in. above its cardiac end. The tumour measures 1½ in. from above downwards, and about 1 in. from side to side. It is covered internally by mucous membrane, and externally some of the longitudinal fibres of the muscular coat can be traced over it. The cut section

has a fibrous appearance, and histologically the growth consists for the most part of unstriated muscular tissue. See *Transactions of the Pathological Society*, 1875, p. 94.

(29) An œsophagus showing, about 2 in. from its cardiac end, an oval tumour placed transversely between its mucous membrane coats. The tumour measures 1 in. in its longer diameter and about ½ in. in thickness, and projects from the outer wall of the tube. The mucous membrane over it is normal, and histologically the growth consists of unstriated muscular tissue.

Clinical History.—A. C., aged 59, was admitted under Dr. Pavy for diabetes, and died of phthisis.

(30) Polypi of Small Intestine.

Papillomata of the Stomach.

(31) A portion of a stomach showing upon its mucous membane two raised flattened tumours measuring about ¾ in. in diameter. Attached by the side of one of them there is a pedunculated growth ½ in. in length. The surface of these masses bears irregular papillæ, giving them a flocculent appearance. Histologically they consist of hypertrophied mucous membrane, the deeper coats of the stomach being unaffected.

(32) The pyloric half of a stomach showing upon its inner surface numerous sessile wart-like excrescences, most of which are about the size of a split pea. Histologically they show local overgrowth of the mucous membrane, the deeper tissues being unaffected.

Clinical History.—D. S., aged 61, was admitted under Dr. Habershon for jaundice and vomiting, and died from carcinoma of the pancreas.

(33) A portion of a stomach showing upon its mucous membrane a large number of rounded sessile polypi, most of which are less than a line in diameter, while one measures ½ in. across. Histologically they consist of hypertrophied mucous membrane.

Polypus of the Stomach.

(34) A portion of a stomach showing a small polypus attached by a broad pedicle to the mucous membrane about 2 in. from the pylorus.

(35) A portion of a stomach showing a small polypus attached to a prominent ridge of mucous membrane about 1 in. from the pylorus.

Clinical History.—J. C., aged 45, was admitted under Dr. Wilks for chronic bronchitis and emphysema. At the autopsy the mucous membrane of the stomach was healthy.

Villous Tumour of the Stomach.

(36) A stomach showing large masses of soft shaggy growth projecting into its cavity. These masses, which vary in length from ½ in. to 3 in., have a

broad attachment along the greater curvature and anterior wall of the organ about the size of the palm of the hand. Except at the seat of attachment, where the wall is slightly thickened, the coats of the organ appear to be normal. Histologically the tumour consists of mucous membrane supported upon the delicate processes of fibrous tissue. There is no appearance of malignant growth.

Clinical History.—I. D., aged 65, was admitted under Dr. Addison for cirrhosis of the liver, from which she died. There were no special symptoms of gastric disorder.

(37) A portion of a stomach showing upon its posterior surface 4 in. from the pyloric opening, a cauliflower-like mass of growth projecting about ½ in. above the surrounding mucous membrane. The tumour occupies an area about 1½ in. long by ¾ in. in width, and does not extend into the submucous or muscular coats of the organ. Histologically the growth consists of mucous membrane covering the delicate processes of fibrous tissue.

Clinical History.—From a man, aged 49, who suffered for eleven months before his death with anæmia and gradually increasing debility. There was no history of vomiting or hæmorrhage.

Fibromyoma of the Stomach.

(38) A portion of stomach taken from the middle of the greater curvature, showing a rounded tumour, about as large as a pigeon's egg, projecting into the cavity of the viscus. The mucous membrane covering it is healthy. Histologically the tumour consists of unstriped muscle and fibrous tissue.

Diffuse Cylindrical-celled Carcinoma of the Stomach.

(39) A stomach laid open to show its walls thickened by a diffuse deposit of new growth which chiefly occupies the submucous tissue, and infiltrates the subjacent muscle. Upon the lesser curvature there is a large irregular ulcer with sharply-defined edges. The organ is somewhat contracted, and its mucous membrane is smooth, the smaller rugæ being completely obliterated. The neighbouring lymphatic glands contain secondary deposits. Histologically the growth is a cylindrical-celled carcinoma.

Clinical History.—From Mr. W., aged 50, who suffered for some months from constant vomiting, and became greatly emaciated; at the autopsy no other secondary deposits were found.

Diffuse Carcinoma of the Stomach.

(40) A contracted stomach measuring 8 in. along its greater curvature and 2½ in. transversely at its widest part. The walls of the organ are universally thickened, in some places to the extent of ½ in., by infiltration of growth in the submucous tissue and by hypertrophy of the muscular coat. The lining of the stomach is slightly ulcerated, and externally there are numerous adhesions to the neighbouring structures. Histologically all the coats of the organ are

seen to be infiltrated by a growth having the characters of a spheroidal-celled carcinoma.

Clinical History.—See *Transactions of the Pathological Society*, xxxvii, p. 236.

(41) A stomach measuring 7 in. along its greater curvature and 2 in. transversely at its widest part. Its walls are ⅜ in. thick, and on section show a white deposit in the submucous tissue. The mucous membrane is smooth and free from ulceration. The omenta are puckered and contain secondary deposits in their lymphatic glands. The portion of colon adherent near the pylorus is considerably narrowed. Histologically all the coats of the organ are seen to be infiltrated by spheroidal-celled carcinoma.

Clinical History.—From a man, aged 77, who suffered from pain after taking food, vomiting and progressive emaciation. A tumour was felt in the epigastric region nine months before his death. At the autopsy the other viscera were healthy.

Papilloma of the Duodenum.

(42) A portion of a duodenum mounted to show a cauliflower-like mass of growth having a broad attachment to the mucous membrane around and below the papilla of the common bile-duct, into which a yellow rod has been inserted. The mass is about 1½ in. long, ¾ in. broad, and projects ½ in. above the surrounding mucous membrane. Close to the growth at its upper part there is a small shallow pouch in the wall of the duodenum.

Clinical History.—E. D., aged 49, was admitted under Dr. Goodhart for jaundice, with pyrexia and hæmorrhage from the intestines. She died three months from the commencement of her illness. At the autopsy two chronic ulcers were found in the rectum, and there were numerous small abscesses in the liver. The common bile-duct was not obstructed.

Polypus of the Jejunum.

(43) A portion of the jejunum showing a tumour about the size of a haricot bean, situated upon one of the valvulæ conniventes. Histologically the polypus consists of a collection of lymphoid cells in the submucous tissue.

Polypus of the Ileum.

(44) A portion of an ileum laid open to show a polypus covered with mucous membrane, the core of which is composed of adipose tissue. It appears to be produced by the inversion of a small diverticulum which has drawn in with its mesentery.

Filiform Polypi of the Ileum.

(45) A portion of the lower end of an ileum showing upon its mucous surface numerous thread-like polypi processes, some of which are nearly ½ in.

in length. Histological examination shows that they are covered with normal mucous membrane.

Clinical History.—D. D., aged 44, was admitted under Mr. Cock, and died from pyæmia following upon a comminuted fracture of the femur.

Polypus of the Colon.

(46) A portion of the sigmoid flexure of a colon, showing a pedunculated polypus attached to its mucous membrane by a stalk about 1½ in. in length. The polypus is oval in shape, measuring about 1 in. in length and ⅝ in. across; the mucous membrane covering it is traversed by irregular fissures. Histologically it consists of a central core of fibrous and muscular tissue, covered with branched villous processes of mucous membrane.

Clinical History.—M. M., aged 69, was admitted under Dr. Perry for acute peritonitis, from which she died twelve hours later. At the autopsy the peritonitis was found to be caused by suppuration of the gall-bladder.

Lymphadenoma of the Jejunum.

(47) A portion of the jejunum mounted to show its mucous surface studded with nodules, varying from ⅛ in. to ⅓ in. in diameter. The surface of some of the nodules is slightly ulcerated. At the back of the preparation is mounted a portion of the ileum in which the growths appear as raised plaques occupying Peyer's patches.

Clinical History.—G. B., aged 48, was admitted under Dr. Pye-Smith for pleural effusion with the physical signs of pressure on the left bronchus. At the autopsy lymphadenomatous growths were found in the tonsils and stomach, and in the mediastinal glands.

Lymphadenoma of the Ileum.

(48) A portion of an ileum laid open to show a circumscribed mass of growth projecting from the mucous membrane opposite to the attachment of the mesentery and almost filling the lumen of the bowel. The growth appears to have its origin in a Peyer's patch. Histological examination of similar deposits in the same intestine shows them to consist of collections of uniform lymphoid cells which do not invade the muscular coat.

Clinical History.—A. MacG., aged 50, was admitted under Dr. Goodhart for emaciation with general enlargement of the lymphatic glands. There was no leucocytosis nor decrease in the number of the red blood corpuscles. She died about six months after the onset of her illness. At the autopsy the spleen was greatly enlarged, and contained a diffuse deposit of soft white growth. The mediastinal and mesenteric glands were swollen, and there was hypertrophy of the lymphoid tissue in the stomach and in the intestinal canal.

Lymphadenoma of the Cæcum.

(49) A cæcum which is considerably dilated and has been laid open to exhibit a mass of growth, about the size of a man's fist, attached to its wall,

round the opening of the ileum, indicated by a blue rod. The growth is soft and lobulated, and its surface presents patches of superficial ulceration. The mucous membrane lining the cæcum and the commencement of the ascending colon is much thickened and marked by transverse rugæ, having the appearance of enormous valvulæ conniventes. Under the microscope the new formation is seen to be due to an infiltration of the mucous and submucous tissues, with uniform small round cells, which do not invade the muscular coat of the intestine.

Polypus of the Rectum.

(50) A globular polypus about ½ in. in diameter with a small portion of a rectum to the interior of which it is attached. The surface is smooth and covered by normal mucous membrane. Histologically the polypus consists of fibro-myomatous tissue.

Clinical History.—G. F., aged 40, was admitted under Dr. Mahomed for heart disease from which he died. At the autopsy the polypus was found to be situated 3 in. from the anus.

Papilloma of the Rectum.

(51) A portion of a rectum to the mucous surface of which is attached a pedunculated polypus about the size of a small cherry. The polypus is covered with delicate papillæ; its pedicle is broad and smooth. Histologically the tumour has the structure of a simple papilloma.

Clinical History.—E. J., aged 40, was admitted under Dr. Babington, in 1847, and died from suppurative peritonitis. At the autopsy there was a circumscribed phlegmon in the wall of the stomach.

Villous Tumour of the Rectum.

(52) A portion of the mucous membrane of a rectum the inner surface of which is covered by a flattened mass of villous growth. Below is mounted a globular tumour of similar appearance and structure. Histologically the growth is a papilloma.

Clinical History.—From Mr. T., aged 58, who had suffered for seven months from rectal tenesmus and a discharge of blood-stained mucus, symptoms which were relieved by the removal of the tumour from the rectum 4 in. above the anus. The disease gradually returned, and seven years later the whole rectum was found to be filled with villous growth producing intestinal obstruction. Left lumbar colotomy was performed, and the condition was discovered to extend as high as the descending colon. The patient survived the operation six years, and eventually died from lung disease at the age of 71.

(53) Three portions of a large sessile tumour removed from the rectum by operation. The masses are made up of closely-set polypoid outgrowths from the mucous membrane giving to the surface a cauliflower-like appearance. Histologically the growth is a papilloma showing no evidence of malignancy.

Clinical History.—T. M., aged 73, was admitted under Mr. Symonds in 1888 for obstinate constipation and a profuse discharge of clear mucoid fluid from the rectum. These symptoms had existed for eighteen months. The tumour was situated 3½ in. from the anus. After its removal the patient made a good recovery.

Pedunculated Polypus of the Rectum.

(54) Found post mortem in a patient who died from lymphadenoma involving the inguinal, retroperitoneal and mesenteric glands.

Papillomata of the Gall-bladder.

(55) A portion of the liver, with the gall-bladder laid open, to show its mucous membrane thickly beset by papillomatous growths, which are small and sessile towards the fundus, whilst towards the neck they are larger and pedunculated.

Clinical History.—See New Sydenham Society's "Atlas of Pathology," Fasc. 5.

Warty Gall-bladder.

(56) The fundus of a gall-bladder everted to show its mucous membrane covered by numerous slender and short papillary processes, which, under the microscope, are seen to consist of connective tissue covered by epithelium.

(57) Fibromyoma of Intestine.

Lipoma of the Stomach.

(58) Found post mortem in a man, aged 65, who died from pneumonia.

Pedunculated Lipoma of the Small Intestine.

(59) *Clinical History.*—A woman, aged 39; sudden abdominal pain five weeks before admission, then at intervals only, with obstinate constipation. Later, persistent vomiting, and finally melæna. On admission, small hard tumour felt to right of umbilicus on pelvic examination.

Operation.—Intussusception of the small intestine, high up, found and easily reduced, when tumour could be felt in jejunum; 12 in. of latter resected and end-to-end anastomosis. Good recovery.

(VII) *From the Museum of Charing Cross Hospital.*

Subperitoneal Lipoma of Small Intestine.

(60) A portion of ileum (inflamed and bearing a small amount of lymph), with a pedunculated tumour attached. The pedicle of the growth springs partly from the mesentery and partly from the gut itself. Some distortion of the gut has resulted.

Microscopically.—It consists of large fat cells of uniform size with very small amount of connective tissue stroma.

Polypus of the Gut.

(61) This was found when examining the intestines of a boy who died from other causes. It had never caused any symptoms during life, and was situated about the middle of the ileum. It has a long and somewhat twisted pedicle, and its structure is glandular in appearance.

Diffuse Scirrhus Carcinoma of Pylorus and Body of Stomach.

(62) Shows complete stomach and portion of duodenum laid open. The viscus is much contracted and appeared before it was opened as a thickened tube. Cavity, 1·45 cm. long (5¾ in.); its broadest part is 5·5 cm. (2¼ in.). The wall near the pylorus is 1·4 cm. thick and thence tapers to 0·5 cm The strict limitation of the growth by the pylorus is marked. The growth appears chiefly to affect the mucosa and submucosa, the muscular wall being greatly hypertrophied, though infiltrated. A few points of ulceration exist. The mucosa generally is catarrhal, and near the antrum pylori is vascular and congested.

Microscopically.—The general thickening of the coats is shown to be due to massive infiltration of mucous and submucous coats, with numerous round cells of inflammatory origin, hiding the normal structure. Large spheroidal cells (cancer cells) are interspersed throughout these cells; they are not accumulated into columns, but towards the fundus they lie in the chinks and clefts between dense fibrous strands (scirrhus). The muscular coats are infiltrated by cancer cells and the bundles of muscle fibres separated by fibrous tissue in great amount. The serous and subserous coats show marked thickening from fibrosis and cellular exudation.

A. R., aged 49, male, painter. General health good. Moderate drinker. Never had syphilis. Four months ago continuous slight pain across back and at same time stools were streaked with blood; bowels constipated. Pain in stomach after food. A month later unable to retain solid food; lived on slops. Great loss of weight. Obstinate constipation. On admission: Looks ill and thin. Lost 2½ st. Pain and tenderness over stomach. Vomiting when solids taken. Vomit contains no HCl. Excess of lactic acid. Laparotomy *nil.*

Gastric Polypus.

(63) The pyloric valve, with adjacent parts of stomach and duodenum. Lying about 1 in. from the valve, on the stomach side, is a lobulated polyp, with a thick pedicle connected with a long radicle, running downwards in the specimen. It is uncertain whether the growth is a true papilloma, or whether it is an outgrowth from the mucous membrane of an inflammatory kind, such as is sometimes found in chronic gastritis. At the upper part of the specimen, near the pylorus, a small nodule seems to indicate the formation of a second polyp in an early stage.

(VIII) *From St. Mark's Hospital.*

Multiple Adenomata of Colon.

(**64**) The specimen, removed by operation, consists of the terminal 4 in. of the ileum with the cæcum and the entire colon to the point where it was divided in the distal third of the pelvic colon, its length exclusive of the small intestine being 29 in. The gut has been opened longitudinally in order to display the mucous surface throughout its length. The cæcum and ascending colon are normal; beyond this the mucous membrane presents a number of small sessile polypi. At a point 12 in. from the ileocæcal valve the first distinctly pedunculated polyp is seen, and these recur at irregular intervals throughout the remaining extent of the specimen. In all there are nine large polypi, the largest being about 1 in. in diameter; in shape they are round or oval, their surface is irregular and of a darker colour than the mucous membrane of the colon generally; their peduncles are 1 in. or slightly more in length. The mucous membrane in between these polypi is irregular and presents large numbers of small, rounded, sessile elevations. The peritoneal aspect of the colon is normal.

The patient, a spinster, aged 24, was admitted into St. Mark's Hospital on March 13, 1918, with a history of recurrent attacks of abdominal pain and diarrhœa since the age of 16. As a rule the bowels were open five to six times daily; slime was constantly present in the stools; bleeding variable. She had lost weight, and had become thin and anæmic; amenorrhœa was present. On examination with the sigmoidoscope numerous polypi were seen as high as the sigmoidoscope could reach; they were red and cherry-like, of variable sizes, they bled readily when touched. Operation was performed by Mr. Lockhart-Mummery on March 18, 1918, under gas and ether, with local infiltration of the abdominal wall with novocaine. The abdomen was opened by a 6-in. incision to the left of the mid-line, splitting the rectus. The colon was examined and numerous movable stalked polypi were felt throughout its length. The ileum was divided near the cæcum, and its proximal end anastomosed to the distal third of the pelvic colon. The entire colon was then excised. Further operation was performed on April 22, and the rectum cleared of polypi through the sigmoidoscope. The patient made a good recovery and was discharged on May 17, 1918. Her condition remains very satisfactory; she has put on weight, and there has been no recurrence of the polypi in the rectum or the small remaining portion of the pelvic colon.

(IX) *From University College Hospital Museum.*

(**65**) Part of a myoma of the small intestine. The tumour weighed 7 lb. and was 10 in. in length. From a man, aged 40. Admitted into Derby Infirmary with acute intestinal obstruction (third day). The abdomen was distended, free fluid present and a large tumour palpable in the left hypochondrium and loin. Laparotomy: The hilum of the tumour was attached to an acutely kinked loop of small intestine. Portion of bowel with tumour (which was free from adhesions) resected.

(66) Large villous tumour of rectum. Removed by Mr. Quain from the rectum of a woman, aged 67. It was attached by a fibrous pedicle 2 in. broad, 3 in. above the anus.

(67) A lobulated villous tumour of the rectum, with pedicle 4·5 cm. broad. From a woman, aged 59, under Mr. Heath; she began to suffer with "piles" eight years previously; a lump sometimes became prolapsed and later there was a slimy discharge, with attacks of diarrhœa.

(68) Section of a rectum with an extensive cavernous angioma; reaching from a point 2 cm. above the anus upwards for 8·5 cm. From a man, aged 45. Since boyhood he had occasionally suffered from slight rectal hæmorrhage. Eight months before admission an attack of diarrhœa occurred with considerable loss of blood at each stool; this was followed by constipation, with occasional hæmorrhages and frequent desire to go to stool. Rectal examination revealed three shallow ulcers, from which blood continuously oozed. The hæmorrhage could not be arrested and the patient died from anæmia.

(69) Carcinoma and papilloma of rectum. The papilloma measures 9 cm. in breadth and is separated from the carcinoma by 2 cm. of healthy mucous membrane. From a woman, aged 68, with symptoms of two years' duration—emaciation, pain and mucous discharge.

(70) Lower end of ileum and cæcum of a female infant, aged three months. The lower end of the ileum is partly obstructed by a cyst connected with its wall, about 3 cm. in diameter. The cyst contained mucus and its wall is composed of mucous membrane, with submucous and muscular layers similar to those of the intestine. The child vomited from birth and was constipated, these symptoms gradually increasing in severity. There was no abdominal distension and no visible peristalsis. A mass, which could be indented with the finger, was felt at the pelvic brim. The constipation and vomiting (varying from three to eight days) were relieved by enemata, but the vomiting recurred after a few days and the patient died from emaciation and exhaustion.

(71) Leather-bottle stomach. (Scirrhus carcinoma.) There is no history.

(72) Stomach greatly contracted from scirrhus carcinoma. From a woman, aged 57. Clinically the case closely resembled œsophageal obstruction; regurgitation of solid food, liquids could be taken slowly and with difficulty. Great pain at lower part of sternum, on swallowing. A full sized bougie passed readily into the stomach. Death from emaciation and exhaustion.

Gastric Cancer.

(73) *Histological Appearance.*—Carcinoma with extreme lack of differentiation of cells. The growth is very cellular, almost sarcomatous in appearance and infiltrates through the inner muscular coat, spreading between the layers of the outer muscular fibres.

Abstract of Clinical Notes.—Severe abdominal pain coming on immediately after food, relieved for a short time by vomiting. Laparotomy had been performed elsewhere about six months before admission, when a large lump was said to have been found on the anterior wall of the stomach. Never vomited blood. Abdominal examination revealed a tumour of the size of a coco-nut filling the costal angle: surface smooth, consistence firm. The inferior margin of the tumour reaches to within ½ in. of the umbilicus. The mass moves with respiration and is not adherent to the abdominal wall.

Stomach infiltrated with Cancer.

(74) *P.M.*—Stomach, small, hard, contracted. Walls much thickened by growth especially in the neighbourhood of the pylorus, where there is a circular induration of indefinite extent and a superficial ulceration for almost half of the stomach. Disease has spread into the great omentum which is represented by a hard, irregular, shrivelled mass. No further spread of the disease either to the lymphatic glands or viscera.

Histologically, adenocarcinoma.

Abstract of Clinical Notes.—Pain after food for some years. Much worse for last seven months so that patient had to give up solid food. Exploratory incision showed "leather bottle" stomach, omental mass and much ascitic fluid.

(X) *From the Museum of the Prince of Wales's Hospital.*

Leather Bottle Stomach.

(75) Stomach, small, hard, and contracted. Large, saddle-shaped ulcer (2 in. by 2½ in.) on lesser curvature, quite shallow, and apparently not extending beyond submucous layer. No evidence of malignancy. Intense fibrous and round-celled infiltration affecting all coats.

Clinical History.—Ten years ago operation for gall-stones. For one year pain in epigastrium, flatulence, vomiting, and loss of weight. The portion of stomach shown was resected and gastro-jejunostomy performed by anterior colic (Polya) method and implantation of cut end to side.

CYRIL NITCH, M.S. } *Hon.*
HERBERT J. PATERSON, M.C. } *Secs.*

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Case of Multiple Exostoses.[1]

By W. Rowley Bristow, F.R.C.S.

The patient is a girl, aged 16. The exostoses involve the regions of the shoulders, wrists, hips and ankles. There is deficient growth at the epiphysis of one ulna, only half of the epiphysis of this ulna being present and the head of the radius being displaced.

Mr. Laming Evans: I think Mr. Bristow is wrong in saying that an inequality in length of parallel long bones is common in these cases. In a paper in *La Revue d'Orthopédie* only a small proportion of the cases quoted showed this condition. Possibly, however, we are now discovering it in cases in which it was formerly missed. Dislocation of the head of the radius is also uncommon. More often a radius which is longer than the ulna extends downwards, leaving the lower end of the ulna away from the wrist joint. Unless the exostoses interfere with the patient's work, they should be left alone. In one case I have removed an exostosis from the inner side of the knee in order to enable the patient to ride.

Congenital Malformation of the Right Femur.

By W. Rowley Bristow, F.R.C.S.

This child is aged 5 months, and presents a congenital deformity of the right femur. The knee and hip joints were flexed at birth, but are being manipulated by the mother and are straightening. As soon as the child is old enough to walk, I propose to put on a walking caliper splint, controlling the flexion deformity. Later, when muscular control is established, he will be fitted with a high patten, and should be able to get about quite well.

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[1] At a meeting of the Sub-section, held December 2, 1919.

The X-ray suggests the possibility of an intra-uterine fracture which has united, but the appearance is probably due to bending of the bone.

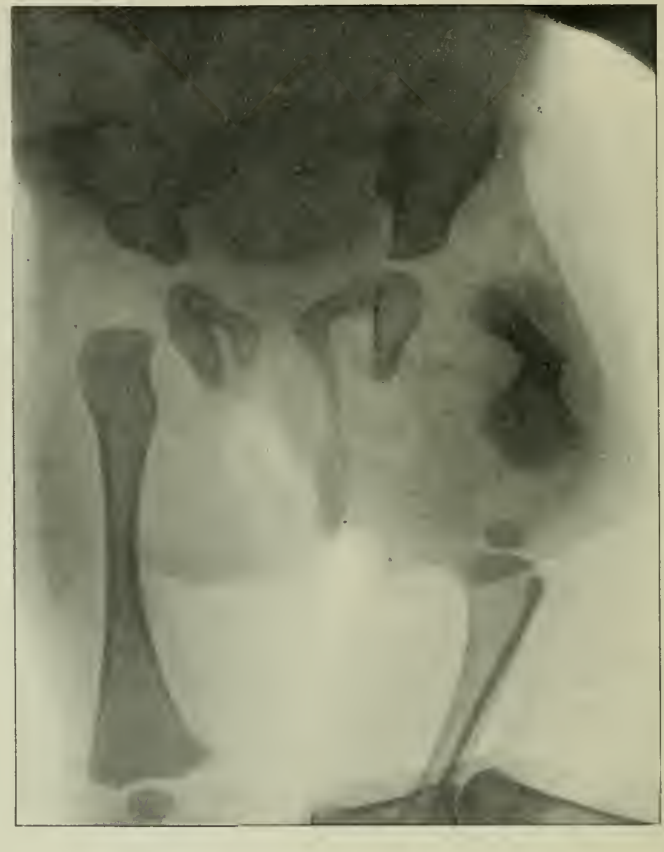

Fig. 1.

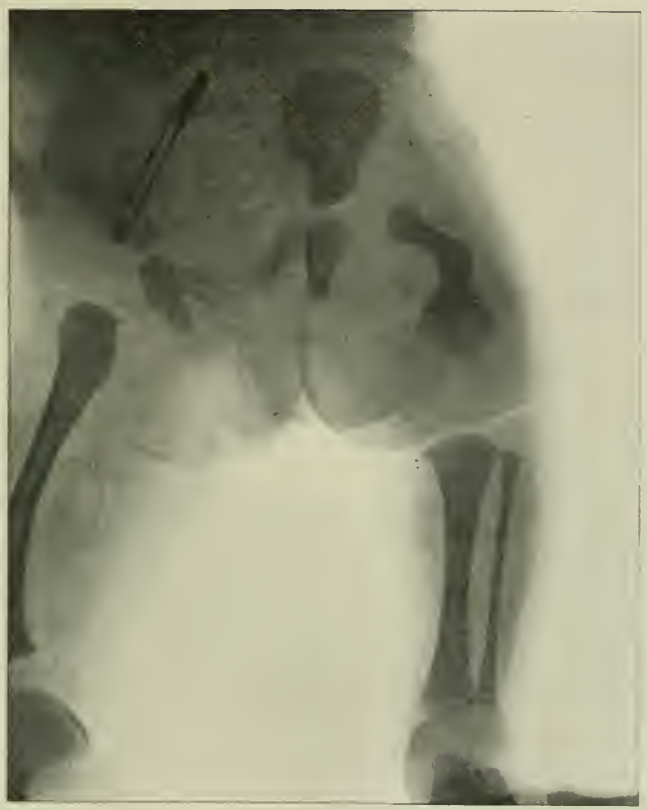

FIG. 2.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Coxa Vara in a Child aged 9 Months.[1]

By R. C. Elmslie, M.S., F.R.C.S.

This boy, aged 9 months, has a deformity of the left femur. The mother states that there was a "cross birth"—one leg was brought down, it is not known which, the left arm was fractured and the left leg was black and blue, but no fracture in it was diagnosed. I saw the child first in December, 1919, the condition being then much as it is now; the femur is bowed forwards and outwards in its upper part, and abduction at the hip is very limited; there is half-an-inch of real shortening. X-ray examination showed that the neck of the femur forms a right angle with the shaft and the latter is convex outwards immediately below the great trochanter; the actual neck of the femur and the epiphysis of the head of the bone are normal. The appearances indicate that there has been a fracture through the region of the trochanters. The pathological anatomy differs from that of the form of coxa vara usually seen in young children; the appearances might explain certain rare cases of coxa vara with a well formed neck which is at right angles to the shaft. I propose to treat the case by an osteotomy through the great trochanter with abduction of the lower fragment into line with the upper.

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[1] At a meeting of the Sub-section, held January 6, 1920.

Osteitis Deformans.

By E. Laming Evans, F.R.C.S.

This patient, a woman aged 48, came under my care at the Royal National Orthopædic Hospital in 1919. In 1910 she knocked her left leg against a form: a swelling appeared accompanied by a slight aching pain; the swelling has gradually increased in size. In 1918 it increased rapidly with exacerbation of the pain. At that time she was admitted into a general hospital in London. The Wassermann test was carried out twice and was negative. After six weeks' observation and rest an exploratory incision was made into the tibia; since that time, however, the pain has continued to increase. There is now uniform thickening of the upper two-thirds of the shaft of the left tibia with well marked anterior curvature; the tibia is half-an-inch longer than that on the right side. X-ray examination shows a uniform absence of definition of cortical and cancellous bone in the upper third with obliteration of the medullary cavity. The middle third shows in addition a suggestion of cystic formation with sequestra. For the last fifteen months there has been pain in the back of the head and the neck with tenderness in the occipital region so that the patient avoids lying on the back of the head. She thinks her head is increased in size; at the present time the occipito-frontal circumference is 21 in. X-ray examination of the skull shows thickening of the occipital and parietal bones.

I regard this as a case of osteitis deformans which I thought worth bringing to the notice of the Section for the following reasons:—

(1) The definite traumatic origin.

(2) The limitation of the disease after nine years to a single long bone.

(3) The anterior curvature of the affected tibia, the more usual curvature being antero-external.

(4) The actual lengthening of the affected bone.

(5) The interval of eight years between the commencement of the disease and the involvement of the second region, namely the occipito-parietal.

Mr. Elmslie: I agree that this case is one of Paget's disease; I should have classed all Mr. Evans's special points as phenomena which commonly occur in this disease. The only appearance which raises any difficulty is the suggestion in the X-ray of the presence of a cavity containing a sequestrum. In the Museum at St. Bartholomew's Hospital there are two femora described by Sir Anthony Bowlby in which cavities containing sequestra exist but in which the general appearance of the bone is that seen in osteitis deformans. It has always been a question whether these bones should be classed as instances of Paget's disease or as chronic osteomyelitis. In this case of Mr. Evans's, however, I think the changes in the skull render the diagnosis certain.

Fracture of the Carpal Scaphoid.

By E. LAMING EVANS, F.R.C.S.

THIS man is aged 39. He injured his wrist in 1917 when starting a motor car: he was pushing down the handle when the engine back-fired and as the handle was not released he received the thrust of the back-fire through the wrist-joint. He has only done a little light work since that date and the disability seems to be quite out of proportion to the physical signs. There is now a slight swelling over the carpus with tenderness on pressure over the scaphoid, the grip is fair except in certain positions of extension when the hand becomes powerless. The power of extension of the wrist is poor, and extreme movements in all directions are restricted. X-ray examination shows a partially united fracture through the neck of the scaphoid with arthritic changes between this bone and the os magnum. The fracture would appear to have been due to a crushing between the down thrust of the forearm and the up thrust of the starting handle. If at the time of the back-fire the handle was held with the hand deviated towards the ulnar side, the head of the os magnum would be directed against the neck of the scaphoid and, acting as a wedge against the weakest part of the latter bone, would produce a fracture. If on the other hand the hand was deviated towards the radial side, the head of the os magnum would press against the semilunar and might produce a dislocation or sub-luxation of this bone.

Mr. C. MAX PAGE: I certainly think that this injury is often of the nature of a crush fracture; I have recently had two cases in which the scaphoid was broken as the result of a back-fire. When the pain and disability persist, removal of the fragments by operation is the best course and in my experience gives satisfactory results; after operation the wrist should be kept supported in an extended position.

Dupuytren's Contraction.

By E. LAMING EVANS, F.R.C.S.

THE patient is a pensioner, aged 32. The ring finger of the right hand is affected and the condition appears to have followed a traumatic arthritis of the first interphalangeal joint caused by the fall of the roof of a dug-out in 1915. The case is of interest both because of the youth of the patient and because of the possible traumatic causation.

Mr. C. MAX PAGE : I have seen a good number of cases of Dupuytren's contraction treated with fibrolysin both by local injection and by intramuscular injection. It has seemed to me that these injections have no influence on existing scar tissue in these cases, and I am very doubtful of their value in the prevention of recurrence of the deformity. I believe that a recurrence of contracture after operation can only be prevented by the wearing of a splint at night for a period of a year or more.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Ankylosis of both Hip Joints.[1]

By R. C. Elmslie, M.S., F.R.C.S.

F. E. G., a private in the Royal Defence Corps, whilst acting as guard at Trent in January, 1917, was seized with acute pain in both hips. The pain came on quite suddenly and necessitated the man's removal to a military hospital on a stretcher; there his case was diagnosed as rheumatism and he remained fifteen weeks in bed. After this he received bath treatment at Buxton. He was finally discharged from the Army on July 30, 1917. He was admitted on January 2, 1920, to the Special Surgical Hospital of the Ministry of Pensions at Shepherd's Bush. He was then walking extremely badly and still suffering from considerable pain. The hips were fixed each in a slightly flexed position and no mobility could be detected; attempts to force movement produced pain. The X-rays show much enlargement and irregularity of the heads of the femora with an absence of the joint line, but without clear evidence of bony ankylosis. As the man was almost entirely unable to walk, I first had him treated by re-education. This resulted in rapid improvement up to a certain point and at the same time encouraged the man to think that he might improve further. He walks now from the knees with the characteristic gait of a double ankylosis of the hip. I think there can be no doubt that the condition was a subacute infective arthritis which has left a close fibrous, or possibly bony, ankylosis. The important question at the present time is that of surgical treatment. The man's disability is very great, and in my opinion an excision of one hip followed possibly later by the same operation on the second hip is justifiable; the alternative is an arthroplasty. I feel less favourably inclined to this, first because it is a more severe operation and the man is 51 years of age, and secondly because in my experience it is not unusual to get a recurrence of ankylosis after an arthroplastic operation.

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[1] At a meeting of the Sub-section, held February 3, 1920.

DISCUSSION.

The PRESIDENT: Whatever one does in these cases of arthritis the cause is not removed and therefore treatment is never very satisfactory. I am inclined to agree that excision is the best treatment in this case and that after an arthroplasty re-ankylosis is frequent. The man's present condition is miserable and whatever is done for him can hardly make him worse.

Mr. LAMING EVANS: Our experience of excision is chiefly based on the result of excision of the hip for tubercle. I have more than once been struck by the stability and usefulness of the hip-joint after excision; possibly the opposition in London to this operation arose because it was devised in Manchester and introduced to London thence. In Manchester I have seen a case of tubercular hip in an adult excised without hesitation by Mr. Platt. In this case I should excise freely, displace the neck upwards and get a false joint. Any inconvenience that the man suffers from having a weak joint cannot be greater than that which he suffers at present.

Mr. ELMSLIE (in reply): Much of the prejudice against excision of the hip in London is the result of the teaching of the late Professor Howard Marsh. I know that weak false joints are common as a result of excision of the hip in children, but I believe that much of the disability is due to inefficient after-treatment, no attempt being made to keep the hip abducted or to support it when the patient begins to walk. I now make it a regular routine to excise the head of the femur in cases of ununited intracapsular fracture when the patient is not too old. I consider that an excised hip is better than one which is chronically painful. In this case I shall now certainly excise one hip.

Congenital Absence of the Ulna.

By A. S. BLUNDELL BANKART, M.C.

THIS child is 10 weeks old, one of twins. The right ulna is absent except for a minute portion at the elbow. The hand is slightly deflected to the ulnar side; there is a dimple in the skin over the middle of the radius. The right elbow-joint is ankylosed. In addition to the elbow deformity there is a congenital contracture in the region of the shoulder-joint, the whole upper extremity being abducted and rotated inwards; the whole limb is smaller than the left.

DISCUSSION.

The PRESIDENT: This case is interesting because in my experience a congenital absence of the ulna is extremely rare. How does Mr. Bankart propose to treat the child?

Mr. BANKART (in reply): I do not propose to do anything for some years, but to wait and see how the deformity progresses.

The Use of Pneumatic Pads in the Treatment of Ununited Fractures of the Humerus.

By C. MAX PAGE, F.R.C.S.

THE principle of the use of a slowly acting force to reduce a malposition or deformity is an important one in orthopædics. Such a force is most often used in the form of a fixed splint. When some degree of reduction has been thus obtained a readjustment of the splint is carried out, so that the force which has diminished or ceased to act is brought to bear again; that is to say, a readjustment or often a replacement of the apparatus is necessary in order to "recharge the recoil spring." It appears to me that in some cases the use of pneumatic pressure could effectively add to the reducing force and increase its range of action.

I first used pneumatic pads in 1913, in conjunction with corrective plaster jackets applied according to the Abbott method. The air pad replaced the felt packing between the jacket and the prominences it was desired to reduce. This application of pads was certainly effective, and considerably increased the amount of reduction which could be effectively carried out by one jacket.

The pads were made from sections of motor inner tubes of a diameter of 2½ to 4½ in. Convenient lengths were from 3 to 9 in. The ends of the tubes were vulcanized, and an ordinary bicycle tyre valve was set in at a convenient position. The tube was enclosed in a fabric case of such a size as to limit its undue expansion when inflated. Pads made in this way by a competent tyre mechanic retain pressure up to 20 to 30 lb. to the square inch, in the same degree as a bicycle tyre. These pads when inflated became cylindrical in form and so exert pressure evenly along the line of their axis. Clearly, however, pressure can be distributed as required for any special purpose by placing the pads between two plaster casings.

I have recently employed these pads for the treatment of certain ununited fractures of the humerus; the aim being to exert a graduated compression at the seat of fracture along the long axis of the bone. I believe that delayed union or non-union in this bone is often the result of over-extension by the weight of the limb, there being an absence of any pressure along the long axis of the bone. Pressure of this kind is undoubtedly of value in stimulating union in fractures of the tibia and femur, and there seems to be no reason why the method should not be equally effectual when applied to the upper extremity.

The method of application is shown in the two cases that I bring forward. A plaster case is applied over the shoulder, arm and forearm of the affected side, usually with the arm well abducted and with the elbow flexed to a right angle. A small pneumatic pad is placed under the plaster beneath the elbow, which is covered with a felt pad. When the plaster is hard the pad is inflated to the degree necessary to push the lower fragment of the humerus upwards in the axis of the bone against the upper fragment, the casing as a whole maintaining the lateral stability of the arm. I have not as yet had sufficient experience of the final results to define the limitations of the method, but I think that it affords a simple method of carrying out a principle of treatment which has long proved effectual in the stimulation of delayed union in the lower limb.

DISCUSSION.

The PRESIDENT: I think this may prove a very useful method. These pads have been used before, but the difficulty has been to get surgical instrument makers to make them satisfactorily. Mr. Page has evidently done the right thing in going direct to bicycle tyre makers for his supply.

Mr. ELMSLIE: We have all found the difficulty of supporting the lower fragment of a fractured humerus in plaster. There is too commonly an idea that extension is required as a routine in fracture of the humerus; very often the exact opposite is the case and upward pressure is wanted. Looking at the pads as used by Mr. Page, I should have thought that they were scarcely thick enough, they should be capable of an extension of 1½ in. It would be interesting to see an X-ray photograph taken in the plaster before and after inflation of the pad.

Mr. Laming Evans: Have any pressure sores been produced? And does Mr. Page use only one shape of pad or as he several different shapes?

Mr. Page (in reply): Without doubt a sufficient range of movement can be obtained by inflation of the pad. The diameter of the pad is 2 in. and it can lift fully 1½ in. I have not had any pressure sores in these cases, but I have seen such sores when using similar pads in plaster jackets for the correction of scoliosis. I think that with reasonable precautions, by the use of felt padding, and by the avoidance of excessive inflation of the tube, sores can be prevented. It should be remembered that a pad can be inflated so as to exert pressure of 30 lb. to the square inch, under which circumstances it becomes almost as hard and resistent as a bicycle tyre. So far I have only used one pattern of pad.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Two Cases of Thickening of the Femur.[1]

By E. Laming Evans, F.R.C.S.

Case I.—M. M., pensioner, aged 34, has had pain in left hip, thigh and knee which commenced in September, 1916, and which has been permanent ever since, without intermission. He has been treated by hot air, electricity and baths, both in London and at Buxton, for eighteen months. Six injections of salvarsan have been given on the chance that the case was syphilitic, although a Wassermann test has always proved negative. He complains of continuous pain in the left thigh, during both day and night, for which slight relief is afforded by very large doses of aspirin. The left thigh shows ¾ in. atrophy (18¼ in. against 19 in.). A slight thickening of the left femur can be felt at the junction of the upper and middle thirds. Local heat, redness or swelling of soft parts are absent. The X-ray shows an osteosclerosis of the shaft of the femur, involving the whole circumference of the shaft, but more marked upon the median segment and extending through the middle and upper thirds as far as the lesser trochanter. The outlines of the sclerosed bone are smooth and the internal structure is well defined. The medullary canal is not encroached upon and there is no evidence of necrosis nor of chronic abscess. The treatment suggested is exposure of the femur on its median surface and a longitudinal linear osteotomy exposing the medullary cavity through the length of the sclerosed area.

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[1] At a meeting of the Sub-section, held March 2, 1920.

Case II.—R. T. P., aged 43, pensioner, noticed pain in the left thigh in February, 1919; and swelling, which started just above the left knee and has gradually increased in circumference and extended upwards. Old standing scars in the region of the right iliac bone suggest an osteomyelitis of the iliac bone in infancy. The right hip-joint is freely movable. On January 23, 1920, when he came under observation, he said he had lost 6 st. in the preceding twelve months. The left thigh showed a hard fusiform enlargement in the lower three-quarter, involving the femur. The left inguinal glands were enlarged but discrete. An X-ray shows a fusiform periosteal thickening of the middle third of the femur with spicules of bone projecting obliquely from the shaft. On February 13, 1920, an incision on the inner side of the middle of the thigh exposed a cavity containing semi-solid gelatinous material. At the bottom of the cavity the periosteum was thickened and studded with bony spicules. An incision on the outer side of the thigh exposed a new growth formation $\frac{3}{4}$ in. thick attached to and growing from the periosteum. A section from this growth is exposed. A report on the semi-solid material states that a few streptococci and staphylococci were found, but the specimen was almost sterile. The tissue cells present were almost entirely lymphocytes, together with a few endothelial cells. The specimen appears to be an inflammatory exudate.

DISCUSSION.

The PRESIDENT: The remarkable thing about the first patient is that he alleges that he has severe pain, but shows no evidence of it; probably this is a case of osteosclerosis. The best chance of improvement appears to be by cutting down upon the bone and drilling a series of holes into it, or cutting a linear incision into it with a saw.

Mr. ELMSLIE: In the second case there are three points which make me disagree with the diagnosis of sarcoma: (1) In the microscopic section there is no evidence of the presence of sarcomatous tissue, but only signs of infiltration and chronic inflammation; (2) the skiagram shows a definite curvature of the femur; and (3) in the X-ray there is an appearance suggestive of the presence of a long central sequestrum. I think that probably this is a case of chronic osteomyelitis, and that for some reason the periosteal bone has taken on a spiky form which is often associated with a sarcoma. I believe that if Mr. Evans had cut more deeply into the bone he would have found inflammatory tissue.

Mr. WARREN LOW: I quite agree that the case is probably one of quiet necrosis, the infection presumably being of low virulence. This seems most probable because the patient has had an osteomyelitis elsewhere. The X-ray does not quite suggest a sarcoma; the spicules are not as directly at right angles to the bone as they often are in sarcoma.

Case of Achondroplasia.

By W. T. GORDON PUGH, M.D.

THE patient is a girl, aged 7, whose height is only 35½ in., about the height of a child aged 3½. She presents the usual characteristics—the stunted arms and legs, contrasting with the trunk of normal size, the short fat "trident" hands, with digits all nearly the same length and showing the deviation of the ring from the middle finger, and the dorsolumbar kyphosis, closely resembling that seen in rickets. The clavicles,

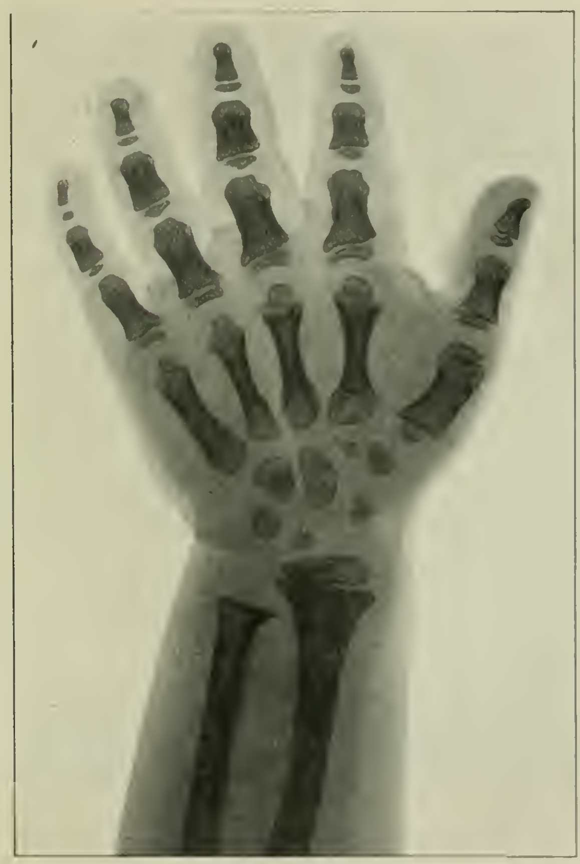


FIG. 1.

Achondroplasia. Child, aged 7.

sternum and ribs are, as usual, unaffected. The child is bright and fairly intelligent. The skin, hair and thyroid gland appear normal, and the Wassermann reaction is negative. The stunted bones of the limbs have broad ends, the shafts are not abnormally bent, and there is normal movement of all the joints except the elbows. The latter joints cannot be fully extended by about 30°, and there is cubitus valgus, the deviation outward of the forearms being about 35°. The measurements compared with those of a normal child of the same age are :—

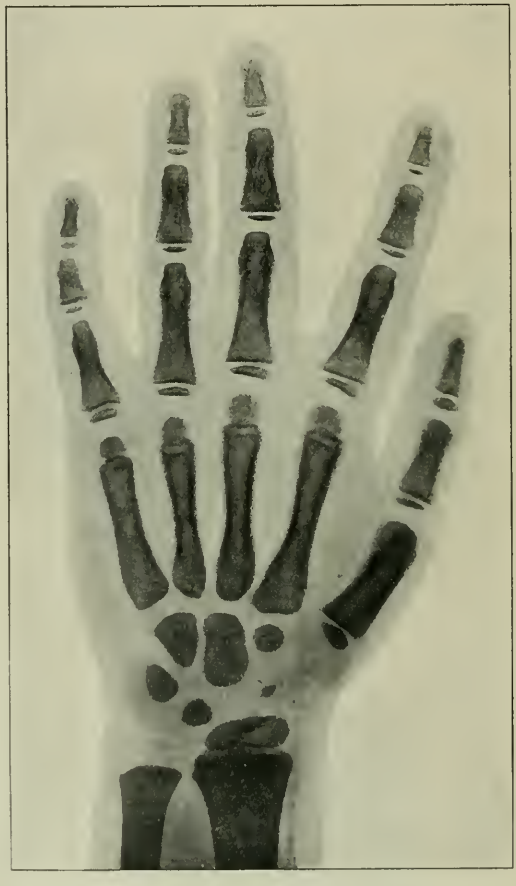


Fig. 2.

Normal child, aged 7, for comparison.

	Inches		Inches
Height	35½	Normal	43
Base of skull to infragluteal fold	19½	,,	19½
Infragluteal fold to sole	10	,,	19½
Acromion to tip of fingers	13	,,	19½
Humerus	5	,,	7¾
Ulna	5	,,	6¼
Femur	6	,,	10½
Tibia	5¼	,,	9¼
Between anterior superior spines	6	,,	6¼
Girth of skull	21	,,	20

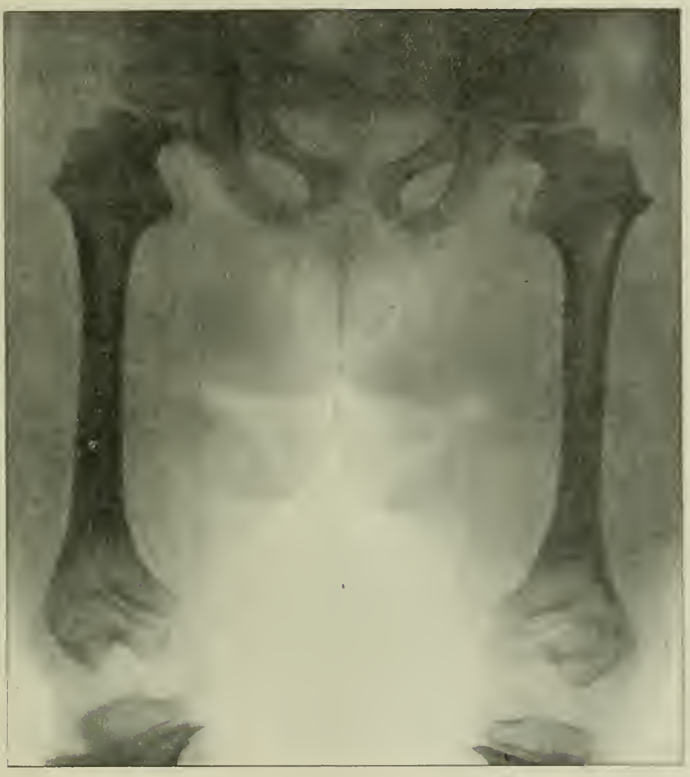


FIG. 3.

Achondroplasia. Child, aged 7.

X-ray photographs show that the bone in the epiphyses is less than normal, whilst the diaphyses show a slight cupping, this indicating that the periosteal bone has not been completed by the addition of endochondral bone, which the epiphysis normally deposits at the ends of the diaphysis.

The ætiology of the condition appears to be still unsettled. Some think it is simply a biological variation similar to that seen in certain breeds of dogs. It has been suggested that it may be due to under-secretion by the pituitary, thyroid, and thymus, and over-secretion by the intestinal glands. Jänsen, of Leiden, eight years ago, produced evidence that this disturbance in the development of the skeleton may result from pressure upon the fœtus at a very early period, from the third to the sixth week, by a amnion either too small or over-distended with fluid. As to treatment, he suggests that having regard to the fact that the pituitary gland has been found reduced in size in severe cases, the prolonged administration of pituitary extract in small doses may possibly stimulate growth, although it will never cure achondroplasia. I do not know whether this has been tried. Recently Professor Keith has declared that, although experiments upon the thyroid have failed to produce achondroplasia, and microscopic examination has revealed no changes in the thyroid gland, these cases may safely be grouped as growth disturbances due mainly to alterations in some of the functions of the thyroid gland. It is generally agreed, however, that thyroid medication has no influence on the growth in achondroplasia.

Exostosis of the Radius.

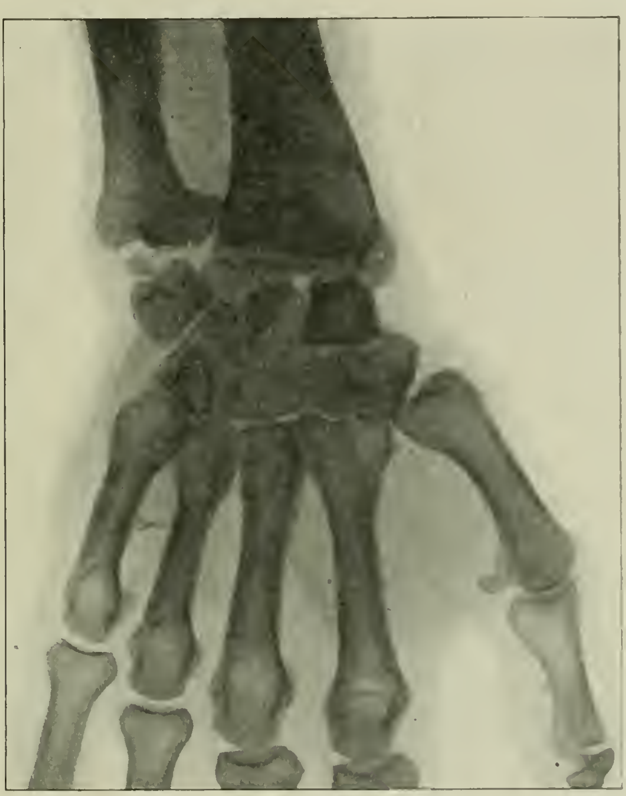
By W. T. Gordon Pugh, F.R.C.S.

In this boy, aged 15, there is an exostosis, about 1½ in. wide and raised about ½ in., arising from the lower end of the radius just above the epiphyseal line. The boy says that he has noticed it about two years, and he thinks that it has grown recently, especially since he knocked it a few weeks ago. It gives him no trouble at present, but as he wishes to enter the Navy it will probably be advisable to remove it.

Mid-carpal Fracture Dislocation.

By R. C. Elmslie, M.S., F.R.C.S.

T. B., aged 38, had an accident to his left hand in January, 1916. He was in the hold of a ship: a heavy sling swung against him, threatening to pin him against the side of the hold; he protected himself by holding out his hands and knee. The sling forced the left wrist into a hyperextended position. The injury was treated in a military hospital as a fracture. The hand recovered to a certain extent, but the condition has for a long time been stationary. The styloid processes are at the normal level; on the posterior surface of the wrist the ends of the forearm bones are less prominent than on the right side, and the posterior surface of the carpus is more prominent. The wrist is ordinarily held in a straight line with the forearm, and only about 30° flexion and no extension is possible from this, and no abduction or adduction is possible.



Midcarpal fracture dislocation.

The X-ray shows a fracture of the scaphoid; one half of the scaphoid and the semilunar have remained in their proper apposition with the radius; the remainder of the carpus is dislocated backwards; the styloid processes of both radius and ulna have been fractured. I have seen three exactly similar injuries to the carpus within a period of a few months. I think this midcarpal fracture dislocation requires recognition as a definite type of injury, resulting in certain cases from forcible hyperextension of the wrist. I propose in this case to remove the semilunar and the proximal half of the scaphoid.

DISCUSSION.

The PRESIDENT: The man has at present a very stiff hand. I think that the operation proposed will probably improve him, and in any case it is not likely to make him worse; I therefore agree that it is advisable.

Mr. P. B. ROTH: I should like to know whether any attempt has been made to improve the condition by forcing movement under an anæsthetic; if not, I think this could be done before the operation is carried out.

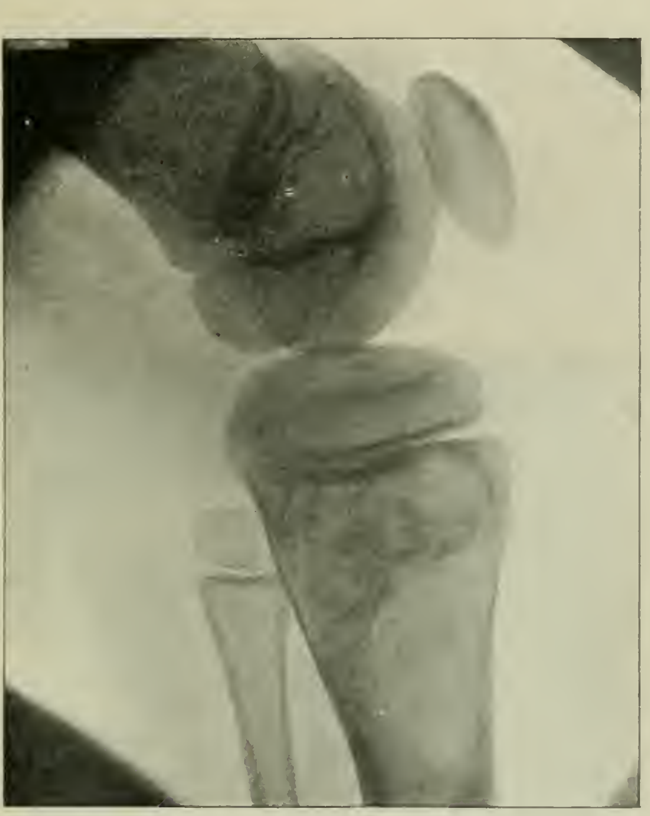
Mr. LAMING EVANS: I do not quite understand why Mr. Elmslie proposes to remove only half the scaphoid. I think a better joint would be left if the whole bone were removed.

Cyst of the Head of the Tibia.

By R. C. ELMSLIE, M.S., F.R.C.S.

THIS boy is aged 10. About a year ago he had a fall, striking the left knee on the ground; he was able to walk home, but the same night a considerable swelling appeared, involving the whole of the knee and accompanied by much pain. He was not seen by a doctor till a week later and he was then treated only by a bandage; he was not confined to bed at all. After the application of the bandage the swelling decreased, but it has never disappeared completely; there has been some pain ever since, but no difficulty in walking except when the boy has walked for a considerable time; he is otherwise healthy and has always been so. At the present time there is a hard hemispherical swelling about 2 in. in diameter situated at the inner side of the head of the tibia. The skin moves freely over it, and there are no signs of inflammation; there is no evidence of implication of the knee-joint. There is a slight degree of knock-knee. The X-ray shows a clear cystic condition, involving the upper end of the shaft of the tibia for a distance of about 2 in., extending to the inner surface and up to the inner half of the epiphyseal line; there is a small portion of healthy tibia left at the outer side.

I believe this to be a case of simple cyst of the tibia, and I propose to treat it by opening the cavity, curetting it thoroughly and crushing in part of the wall.



Cyst of the head of the tibia.

DISCUSSION.

Mr. P. B. ROTH: What is the prognosis after an operation of curetting and crushing in the wall? Does the cyst always heal, and are there no complications?

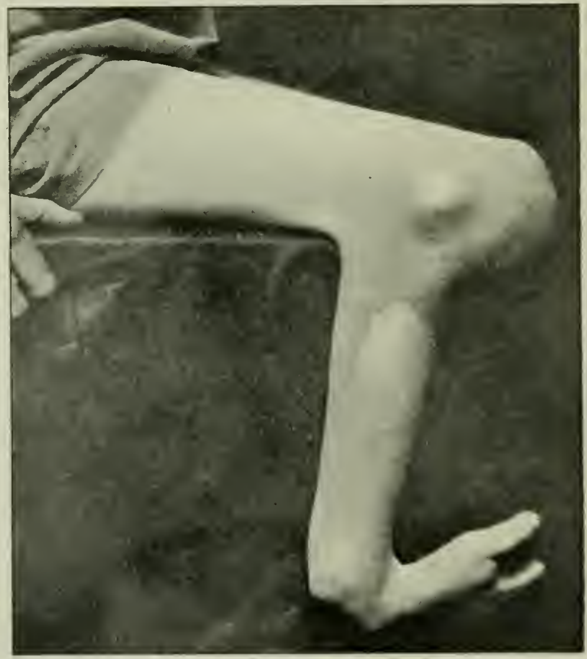
Mr. H. A. T. FAIRBANK: I am not quite clear as to Mr. Elmslie's reasons for considering this to be a cyst and not a myeloma.

Mr. ELMSLIE (in reply): In cases of cyst of the femur, humerus and tibia upon which I have operated, healing has taken place satisfactorily; the bone around has sclerosed and become strong enough for use, but a slight cystic appearance has remained. In no case was there further growth or extension or recurrence. My feeling that this is a cyst is based entirely upon analogy with other cases; I agree that it is possible that I am wrong, and that I shall find on operation that there is myeloma.

Congenital Absence of the Tibiæ.

By R. C. Elmslie, M.S., F.R.C.S.

The patient is a girl aged 13 years 8 months. The deformity is congenital, and no treatment has so far been carried out. The tibia is absent in both the lower limbs, the patella and the condyles of the femur being apparently normal. The head of the fibula on either side is large and articulates with the outer side of the external condyle. The feet are in a state of severe talipes varus, on the right there are four



Congenital absence of the tibiæ. Photograph of the right lower limb.

toes with a deep cleft between the first and second, as in a lobster-claw deformity. On the left side the great toe is absent.

The child gets about well on the knees. I propose to amputate at the knee on each side, and will present a further report on the anatomy of the parts removed at a later meeting.

The PRESIDENT: This is a very remarkable case. I have only seen about six cases of absence of the tibia, and never one with so much displacement of the fibula. I agree that there is nothing to be done short of amputation; if it were not for the shortening the girl would do very well without artificial limbs.

Birth Palsy with Dislocation of the Head of the Radius.

By R. C. ELMSLIE, M.S., F.R.C.S.

F. S., A GIRL aged 15½, has had a deformity of her right arm since birth. No history of the birth or early condition is available. No treatment appears to have been carried out. The arm is held a little away from the side with the elbow flexed and the forearm supinated. Movements of abduction and rotation at the shoulder are restricted. Full extension of the elbow is impossible, and pronation of the forearm is also absent. Active use is present in all the muscles of the limb except the pronator radii teres, but the deltoid, triceps and extensors of the wrists and fingers are weak. The muscles all react to a faradic current, including the pronator radii teres.

An X-ray photograph shows a forward subluxation of the head of the radius and a great prominence in front of the tuberosity.

The chief interest of the case consists in the supination deformity and in the presence of the subluxation of the head of the radius. I do not think that excision of the head of the radius is advisable, as it will probably weaken the arm; the girl gets very good use out of the hand as it is. I propose to limit treatment to manipulation and exercises for the joints and electrical treatment of the weakened muscles.

DISCUSSION.

The PRESIDENT: I have never seen a case of fixed supination deformity. There is a little passive pronation possible, and this appears to be limited by the prominence of the bicipital tuberosity. I agree that operative treatment is inadvisable.

Mr. H. A. T. FAIRBANK: I have a case now under treatment in which there is a limitation of pronation, but this is due to previous treatment, the forearm having been held in a supinated position, and having become stiff in the new position; it is improving. I have seen several cases of subluxation of the head of the radius, but most of them have had limitation of supination.

Mr. LAMING EVANS: Is there a more active response to faradic stimulation of the biceps in this case than in other cases of birth palsy? It is interesting to note that subluxation occurs in these cases both in the shoulder and in the radius; presumably this was due to retraction of muscles which recovered early.

Case for Diagnosis.

By PAUL BERNARD ROTH, F.R.C.S.

PATIENT, a boy, aged 4, with waddling gait. He had never walked properly, but always with the toes turned out, rocking from side to side with each step. He had been taken previously to two hospitals, at one of which he was treated for a time with long external splints, but these had not been able to keep the legs from twisting. He had suffered from rickets, as evidenced by slightly bowed tibiæ and enlarged lower radial epiphyses.

The gait was characteristic of double congenital dislocation of the hip-joints, but the radiogram by Dr. W. Ironside Bruce showed no abnormality of outline of bones to suggest this or any other deformity. When the patient was lying supine, passive abduction of each thigh was limited to 45°. The thighs were kept rotated out 80°, but could be untwisted so as to make the toes point straight up, by using moderate force.

(Since writing the above it has been suggested by Dr. Martin Berry that in all probability this is a case of *abnormal torsion* of the femoral shaft.)

Mr. LAMING EVANS: There may be a waddling gait in any case of weakness or deficiency of the pelvi-trochanteric muscles. I should look upon this case as one of weakness occurring in a child suffering from rickets.

Dupuytren's Contracture in a Young Man following Injury (shown before and after Operation).[1]

By PAUL BERNARD ROTH, F.R.C.S.

S. S., AGED 31, was wounded in January, 1916, near Armentières, with a rifle grenade splinter in the ring finger of the left hand. There was a small flesh wound situated, as can be seen by the scar on the palmar aspect of the ring finger, at the junction of the middle and distal phalanges. After a month it was completely healed, though there seems to have been some suppuration, and patient thought nothing more about it and returned to duty. He was treated the whole time at an "advanced dressing station."

In December, 1916, patient "began to feel something tight pulling down his finger, and it became bent towards the palm. This got worse gradually and when shown on November 4, 1919, the ring finger was flexed 60° at its metacarpo-phalangeal joint, the interphalangeal joint not being affected. A very prominent ridge, due to the contracted palmar fascia, was seen in the palm, leading up to the base of the ring finger: to this ridge the skin was adherent, as shown by puckered dimples and transverse ridges at several places.

Note.—The little fingers of both hands had been contracted ever since the patient could remember, but had never given any trouble.

REMARKS.

I suggest that the points of interest about this case are: (1) The manner of causation; (2) the youth of the patient; and (3) the treatment to be adopted.

(1) There is no doubt this is a true case of Dupuytren's contracture. The cause of the condition is not definitely known: but in this case it is probable that organisms of suppuration have gained entry to the prolongation of the fascia on the ring finger, spread down it to the palm and have set up a septic fibrositis in it.

(2) The youth of the patient—he was aged 28 when the contraction commenced—is certainly worthy of special note. In all descriptions of

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[1] This case was shown to the Members of the Sub-section at their Meeting on December 2, 1919, before operation. See *Proceedings*, 1919-20, xiii (Sect. Surg., Sub.-sect. Orthop.), p. 113.

the condition the writers have insisted on the advanced age of the patients. Thus Noble Smith recorded seventy cases of this condition, all of which occurred in elderly people (quoted by Bradford and Lovett).

(3) In the treatment of these cases I prefer to adopt Adams's operation performed in the manner he described, and followed by bandaging the finger in complete extension to a straight splint.

Operation was performed on November 7. Under chloroform anæsthesia the skin of the palm was punctured with a fine tenotome in eight or nine places just to one side of prominent parts of the tense fascia. At each puncture the tenotome was introduced with the flat of its blade almost parallel with the surface of the palm and it was gently insinuated between the skin above and the prominent band of fascia beneath. The blade was then rotated 90° until its edge was at right-angles to the fascia and the fascia was then divided by cutting down into the palm. Traction was simultaneously made on the flexed finger, and as each part was divided, the finger gradually straightened until it lay completely extended. A straight splint was applied and kept on for a week: after this it was removed during the day, but applied for a further two weeks at night.

The operation has resulted in a complete cure, with full power of extension and flexion.

The PRESIDENT: Many of us saw this case a few months ago and we must congratulate Mr. Roth on the treatment. Possibly, as the case may be one due to sepsis, the prognosis may be better and relapse less likely, for the septic element having subsided there cannot be the tendency to progress that is present in a true Dupuytren's contraction.

Section of Surgery.

SUB-SECTION OF ORTHOPÆDICS.

President—Mr. E. Muirhead Little, F.R.C.S.

Case of Genu Varum of Late Onset.[1]

By C. Jennings Marshall, F.R.C.S.

J. W., a youth, aged 18, applied at the out-patient department of King's College Hospital for relief of bandy legs. Occupation, hotel porter. Nothing abnormal was noticed before a year ago, and there was little change till six months back when the condition rapidly became worse till the present time. Throughout this period no other departure from general health has been noticed. There is no history of previous illness. The parents, both of them of average stature, while confirming the above history in every respect, state that the patient is distinctly inferior to his brothers in height and in general build. To the best of their knowledge the patient suffered from no illness during his childhood.

The patient is a sallow, greasy-skinned youth some 5 ft. 4 in. in height. The lower limbs show severe genu varum, the knees being 5 in. apart, with the malleoli in contact. Curvature has taken place at both knees, more so on the left side. In each instance the femur and tibia share in the deformity, the tibia showing the greater change. Palpation reveals just above the inner condyles of the femora a small expansion of the bone at the level of the epiphyseal cartilage. There is no such change in the tibiæ. Examination of the bony parts elsewhere shows: the lower ends of the radius and ulna seem expanded; the radial styloid is lower than usual; no other notable bony abnormalities are to be found; the trochanters exhibit normal relationships. Visceral findings: Severe gingivitis; throat and tonsils negative; heart and lungs normal. Abdomen: The spleen is slightly tender and descends to two finger-breadths below the ribs on inspiration; nothing else of note. Examination of glands and joints yields negative results. Urine: No albumin, no sugar. Wassermann reaction negative. Blood examination: Red cells, 5,100,000; colour index, 0·95; leucocytes, 21,000; polymorphs, 85 per cent.

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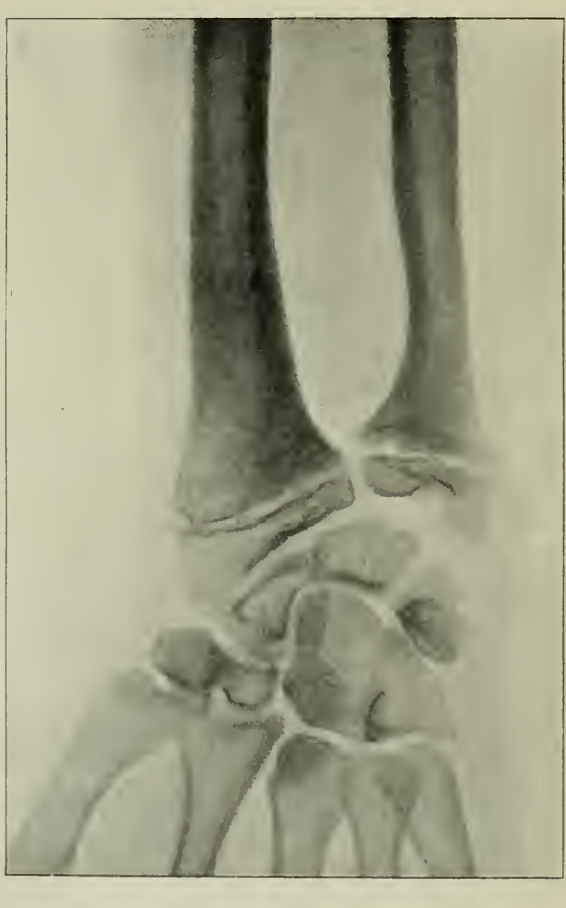
[1] At the Annual General Meeting of the Sub-section, held May 4, 1920.



Radiograms show changes in the bones concerned that are fairly typical of rickets. The epiphyseal interval is increased and the adjoining parts of the diaphyses and epiphyses show rarefaction with a billowy sort of appearance. In the case of the femora the softened tissue projects on the inner side, as it were squeezed out. Both in the femora and in the tibiæ these changes affect only the inner aspect of the epiphyseal region. On the outer sides fusion of epiphysis and diaphysis seems already to have taken place. At the wrists identical changes are seen at the epiphyseal lines in their entirety. Apparently, there is delayed growth of the ulnæ, the line lying much higher than that of the radius. In addition, the line in the radius has a curious obliquity directed downwards and to the radial side. The hip regions present no pathological change.

The feature of the case chiefly meriting attention is the curious selective action of the pathological process at the epiphyses in the knee region. Accepting the process as being essentially the same as rickets, we have to decide whether the case is one of late rickets or of continued rickets. If it is a case of continued rickets, why has fusion occurred already on one side of the epiphyses, two years in advance of the usual date? I can find no information as to whether the two sides fuse simultaneously in the normal individual; it seems a point worth investigating, but in any case it is difficult to see how such fusion could occur in face of a pathological process of the extent now present in the inner epiphyseal zones.

The question of the production of the deformity, given fusion of the outer zone and greater pressure on a wide band of soft bone at the inner zone, seems to present no great difficulties.

The history would appear to indicate a somewhat deficient growth of bone in general. We have physical signs of a toxæmia—a large spleen, and a pasty, greasy skin. The gingivitis may be the source—the therapeutic test should decide this question for us. We do not know its duration however; from the history of the deformity and from the peculiarities at the knees already pointed out, it would appear to be recent. It would be an advantage to know whether in addition the case is not complicated by some glandular insufficiency.

There is no evidence for placing the case in the category of "nephritis associated with deformities at the knees," described in the *Lancet* of January 3, 1920.

The treatment to be adopted will be as follows: (1) Attention to general hygiene and dental toilet. (2) Thyroid feeding for a while. (3) Gradual correction of the deformity by screw pressure on metal plates embedded in plaster cases gripping thighs and legs. (4) After a suitable period of recumbency, sufficient to procure correction, the wearing of knee splints with moulded thigh and calf pieces, and irons to the ankles.

DISCUSSION.

The PRESIDENT: I quite agree that this interesting case resembles rickets, but the condition is not typical of this or of any other definite disease. The most important question is, what treatment should be carried out?

Mr. R. C. ELMSLIE: I suppose we must call this late rickets, but in most of the cases that have been described under that name the changes have been more characteristic of rickets than are those seen in this boy. The bone changes which we call "rickety" are apparently not always due to the same cause; thus in renal insufficiency in children and also in cases of intestinal infantilism we see epiphyseal changes and deformity of the long bone indistinguishable from those of true rickets. In this case the changes are also similar to those of rickets and here chronic sepsis is the cause. The indications for treatment are, evidently, first to combat the septic condition and secondly to vary the diet, giving particularly such food as we know from our experience of rickets will assist ossification.

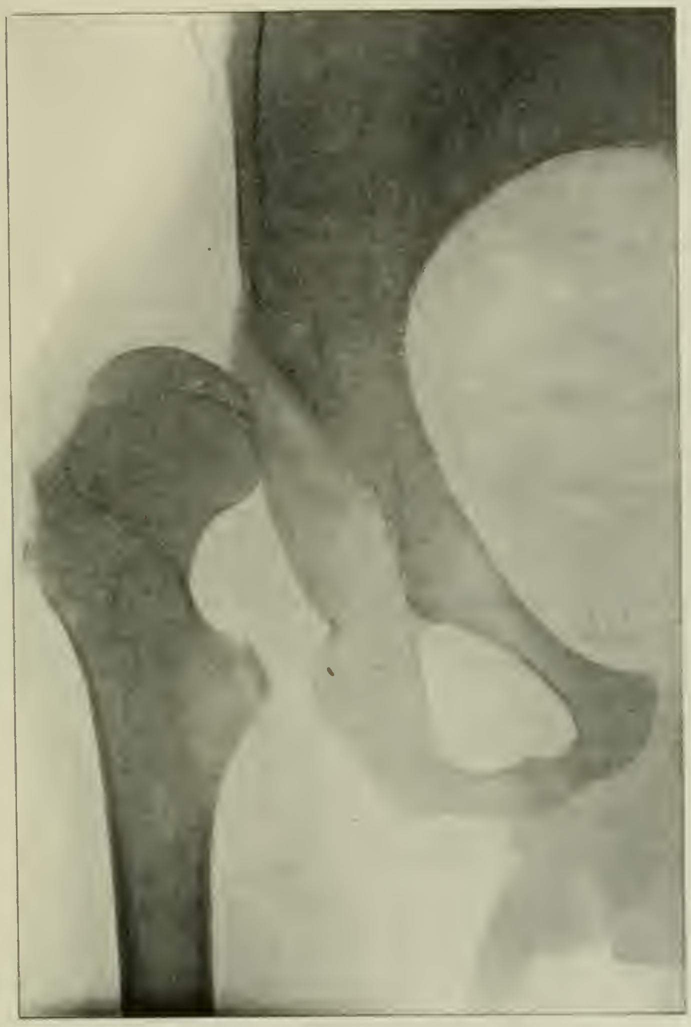
Mr. MARSHALL (in reply): I intend to treat the case by combating the septic condition and by dieting; I have also been trying thyroid extract. I propose to take him into hospital, and acting on the assumption that if the bones will bend out of shape, they can be bent back into shape—to attempt to correct the deformity by pressure.

Case of Paralytic Dislocation of the Hip.

By R. C. ELMSLIE, F.R.C.S.

THE patient is a boy, aged 15. He had infantile paralysis as a baby. The right lower limb is now affected. The limb is 3½ in. short. The muscles around the hip are fairly strong; all groups seem to be active. The thigh muscles are active, but below the knee the tibialis anticus is missing, the rest of the anterior tibial group and the peronei being active. The calf muscles are absent, with the exception of the long flexors of the toes. There is talipes calcaneo-valgus. There is a scar on the outer side of the foot; what operation has been performed I do not know. The hip is dislocated, the head of the femur being palpable at the level of the anterior superior iliac spine, the femoral neck is apparently much anteverted. An X-ray photograph shows that the femoral head is well formed, but that the acetabulum is shallow and the upper margin much flattened.

True paralytic dislocation of the hip is in my experience very rare, if it ever exists. One can understand a dislocation occurring in a case of severe paralysis with some persistent adductors and flexors and no gluteal muscles. But though dislocations are seen in such cases, they are generally incomplete. In this case there is a complete dislocation in a comparatively mild paralysis. I have seen one such case before in which the paralysis and the completeness of the dislocation were very similar. It is possible that the dislocation is not really paralytic, but should rather be called a dislocation in a paralysed limb.



Paralytic dislocation of the hip.

I should like to hear opinions as to the possibility of treatment, whether it would be possible to reduce the dislocation with any prospect of retention, or whether an extension instrument should be worn.

DISCUSSION.

The PRESIDENT: I do not think that Mr. Elmslie need alter his opinion as to the occurrence of complete paralytic dislocation in this case, as it is quite possible that this is really a congenital dislocation in a limb in which an attack of poliomyelitis has subsequently occurred. In the cases of paralytic dislocation that I have seen there has been a much more extensive paralysis. I hope that we shall hear some suggestion as to treatment.

Mr. FAIRBANK: I have seen a case of paralytic dislocation recently, and in this the limb was flail and useless. In Mr. Elmslie's case I feel certain that the limb is so good that an effort might be made to reduce the dislocation. I have read that an operation is being done in America in which a chisel is driven into the ilium above the acetabular margin and wedges of bone inserted, with a view to forming a shelf which will prevent the head of the femur from slipping upward. I do not quite gather on what ground Mr. Elmslie based his idea that there is great anteversion of the neck of the femur.

Mr. ELMSLIE (in reply): I judge of the anteversion by the fact that the head of the femur is felt to lie almost directly in front of the great trochanter and that in order to make the femoral neck point inwards, the limb must be rotated so that the patella is directed towards the opposite knee. The operation Mr. Fairbank suggests is one which I have myself suggested for cases of congenital dislocation in which the acetabulum is insufficiently deep to retain the head of the femur. It is quite possible that the President is right and that this is really a case of congenital dislocation.

Case of Bony Ankylosis of Left Knee.

By PAUL BERNARD ROTH, F.R.C.S.

H. A., AGED 43, housekeeper. One night in May, 1919, her left knee began to swell after some kneeling; it swelled up very much and was very painful and hot. She went to bed and was treated by her doctor, who applied cold dressings; it got better, but not well. So as she could not get a bed in a hospital she went into the Chelsea Infirmary, where she was six months in bed, with Scott's dressing and splints applied to the knee. No operation of any kind was done.

She came to hospital on March 23, 1920, to know if anything could be done for her stiff left knee and for her swollen right ankle. The condition of the ankle has been troubling her since she left the infirmary; it swells up every day and hurts her, but at night the swelling subsides and by next morning is all right again.

Family history *nil*; husband, Lance-Corporal Royal Engineers; one child, aged 8. Wassermann reaction negative.

On examination, there is complete bony ankylosis of left knee, which has fortunately occurred in the extended position. There is slight heat and swelling of right ankle-joint; pupils are unequal; pyorrhœa + +.

Treatment: As X-ray of right ankle is suggestive of gonorrhœa, treatment by gonorrhœal vaccine has been begun.

Case of Bony Ankylosis of Right Ankle-joint Treated by Cuneiform Osteotomy.

By Paul Bernard Roth, F.R.C.S.

E. M., aged 42. After having given birth to six healthy children, all of whom are alive and well, there was one premature birth at six to seven months, followed by two miscarriages. For the second miscarriage she entered the infirmary, and while there, there was some ulceration round her right ankle and an abscess in the left buttock which was opened by the infirmary doctor. As the right foot had become twisted, the doctor said he would put it right for her, and he applied a splint for six weeks; but the treatment did not improve the foot, which remained stiff and dropped.

When seen in May, 1919, there was complete bony ankylosis of right ankle-joint, with foot fully plantar flexed (confirmed by radiogram, unfortunately lost). As the patient was unable to walk owing to the position of the foot, an anterior cuneiform osteotomy was done in October, 1919, and the foot brought up to a right angle. Bony ankylosis has again formed in this position, and patient is able to get about very comfortably, regarding herself as completely cured.

Case of Bilateral Congenital Elevation of the Shoulder.

By H. A. T. Fairbank, F.R.C.S.

C. E., aged 10. The mother only noticed that the girl could not raise the right arm properly two months ago. Both scapulæ lie at an abnormally high level, the right being higher than the left. Both are rather small scapulæ, and that on the right has the vertebral border tilted up. On both sides the movements of these bones are much restricted, the vertebral borders hardly moving at all away from the spine, yet the left arm can be raised to the vertical, while the right only moves to a position a little beyond right-angle abduction. The spinal column is flattened in the upper dorsal region. There is a slight long dorsal curve to right. There is a pigmented umbilicated spot, the size of a sixpence over the third or fourth dorsal spine. A complete electrical examination of the scapular muscles has not yet been made.

A radiogram shows thirteen ribs on each side, the highest being well formed, and apparently a normal first rib on each side. The fifth, sixth, and seventh dorsal bodies, reckoning all ribs as true ribs, seem to be flattened and crowded together. Considering the malposition of the scapulæ and the extra ribs, the vertebral deformity, by no means clearly seen in the radiogram, is considered to be congenital, and not the result of disease.

It is not suggested that any treatment is necessary.

DISCUSSION.

The President: I quite agree with Mr. Fairbank that in this case operative treatment does not seem likely to promise improvement. In cases in which the scapula is tethered to the spine by an intermediate mass of bone it is much more likely to be useful.

Mr. Bankart: The extremely good function in this case, particularly on the left side, is a commentary on operation in such cases. It is quite a new fact to me that it is possible to move the shoulder-joint itself through an angle of 180°. Watch this girl carefully and it can be seen that she moves the left scapula a little, then fixes it and elevates the arm from the shoulder, the head of the humerus becoming subluxated downwards in the latter part of the movement. It is interesting to work out what muscles carry out this movement; apparently it is the deltoid and the clavicular part of the pectoralis major which are elevating the shoulder-joint beyond the right angle.

Mr. ELMSLIE: This case seems to me to show how we should treat congenital elevation of the scapula, that is by exercising the shoulder so as to get a maximum of movement in the shoulder-joint itself. The appearances are very different to those of the ordinary bilateral elevation of the scapula; there is usually a very short broad neck with some angular deformity of the spine at the cervico-dorsal junction. The obvious external deformity in this case is much less than that commonly seen.

Mr. FAIRBANK (in reply): I will get the shoulder-joint on the left side X-rayed in various positions in order to show the subluxation which evidently takes place when the limb is elevated.

A Note on Abnormal Torsion of the Femoral Shaft.

By PAUL BERNARD ROTH, F.R.C.S.

AT a recent meeting of the Section (*Proceedings*, 1920, xiii, Subsect. Orth., p. 226) I showed for diagnosis, and again show to-day, a small child with waddling gait of unknown origin. It was afterwards suggested by Dr. Martin Berry that this was a case of abnormal torsion of the femoral shafts. As this was a condition unknown to me, I decided to gain some information about it, and applied to the Librarian for the latest treatise dealing with the Femur. He referred me to Professor Karl Pearson's newly published work, "A Study of the Long Bones of the English Skeleton," and by a happy co-incidence "Part I, the Femur," arrived next day. From this exhaustive study I was able to gather the following particulars about torsion of the shaft, which will, I think, be of interest to the members of this Section.

(1) *Normal Torsion of the Shaft.*—There exists in the normal femur a torsion or twist of the shaft: if the upper end be regarded as fixed, the torsion consists of a rotation inwards of the lower end. This torsion affects the whole of the shaft. It is much more marked in the left femur than the right (*see* Part I, p. 132), and slightly more marked in the female than the male. Thus in 735 femora measured, of which 359 were right and 376 left, the average angle of torsion for the former was 21·8°, and for the latter 27·7°: of the 359 right, of which 208 were male and 151 female, the average angle for the former was 21·7° and for the latter 22·0°: of the 376 left, of which 211 were male and 165 female, the average angle for the former was 26·7° and for the latter 28·7°. The following table may help to make these figures clear:—

	Right		Left	
	Cases	Average angle	Cases	Average angle
Male ...	208 ...	21·7°	211 ...	26·7°
Female ...	151 ...	22·0°	165 ...	28·7°
	359	21·8°	376	27·7°

The largest angle recorded was 69·6°.

(2) *Measurement of the Torsion.*—To measure the amount of torsion, a torsiometer was devised by Professor Pearson, which was clamped on to the shaft as shown in fig. 1A and fig. 1B, reproduced from Plate XXI.

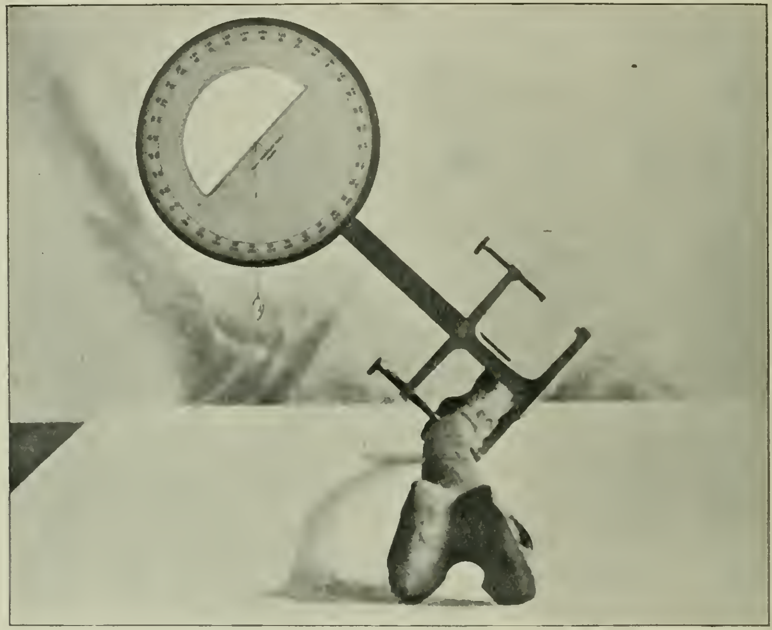


FIG. 1A.

(Reproduced from Plate XXI by kind permission of Professor Karl Pearson.)

In applying the torsiometer the screw points are fixed at the terminals of the "platymeric antero-posterior diameter" (q.v.). This instrument can only be used of course on the detached femur: for measuring the torsion in the living subject, *see* paragraph (4).

(3) *Abnormal Torsion of the Shaft.*—The torsion already described with the lower end of the femur rotated inwards—or if we care to regard it so, with the upper end of the femur rotated outwards—is known as positive torsion. When the torsion is found in the opposite

direction, with the lower end of the femur rotated outwards—or with the upper end of the femur rotated inwards—it is known as negative torsion. Whether an increased amount of positive torsion can give rise to symptoms I do not propose to discuss, but I do suggest that marked negative torsion certainly does so, and I offer my case of the small child with waddling gait as an example of the condition. In the 735 femora examined, negative torsion was present in thirteen cases, that is, in 1·76 per cent. The amount of it in the thirteen cases was respectively 2·2°,

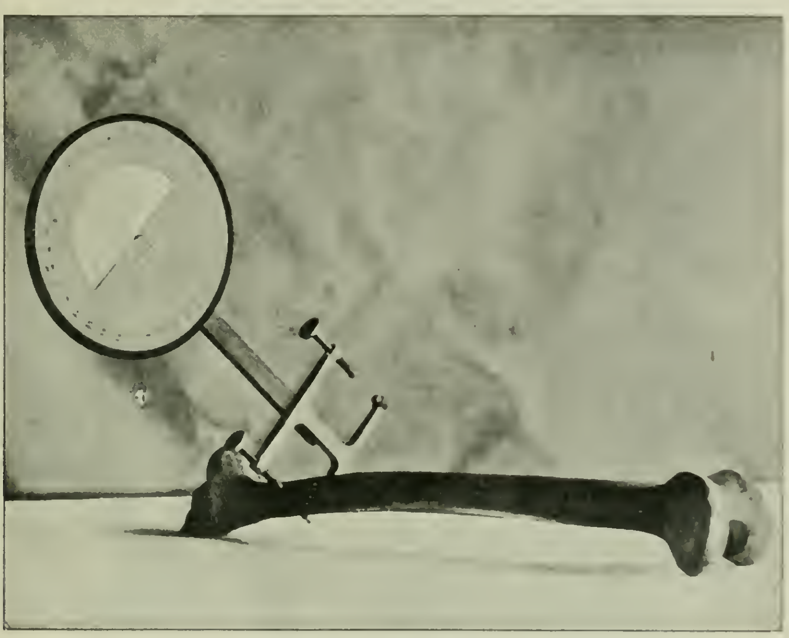


FIG. 1B.

(Reproduced from Plate XXI by kind permission of Professor Karl Pearson.)

3·9°, 5·0°, 5·9°, 6·0°, 6·1°, 7·0°, 7·3°, 7·5°, 10·2°, 10·5°, 10·6°, and 31·0°, giving an average negative angle of 8·7°. In six of the cases, including the three most marked, there was deformity of the shaft suggestive of rickets. Fig. 2, reproduced from Plate IV of Professor Pearson's work, shows varying degrees of negative torsion compared with positive torsion.

(4) *The Recognition of Abnormal (Negative) Torsion in the Living Subject.*—The features of the case shown are (i) presence of rickets; (ii) waddling gait; (iii) rotation of the thighs outwards, so that when the child walks the two feet enclose an angle of 90°; (iv) no demonstrable

radiographic abnormality; (v) when lying supine on a flat table with feet touching and toes pointing directly upwards (standard position), the great trochanter on each side occupies a more anterior position than usual. This is because the deformity causes the lower end of the femur with the leg and foot to be rotated outwards: when this rotation is

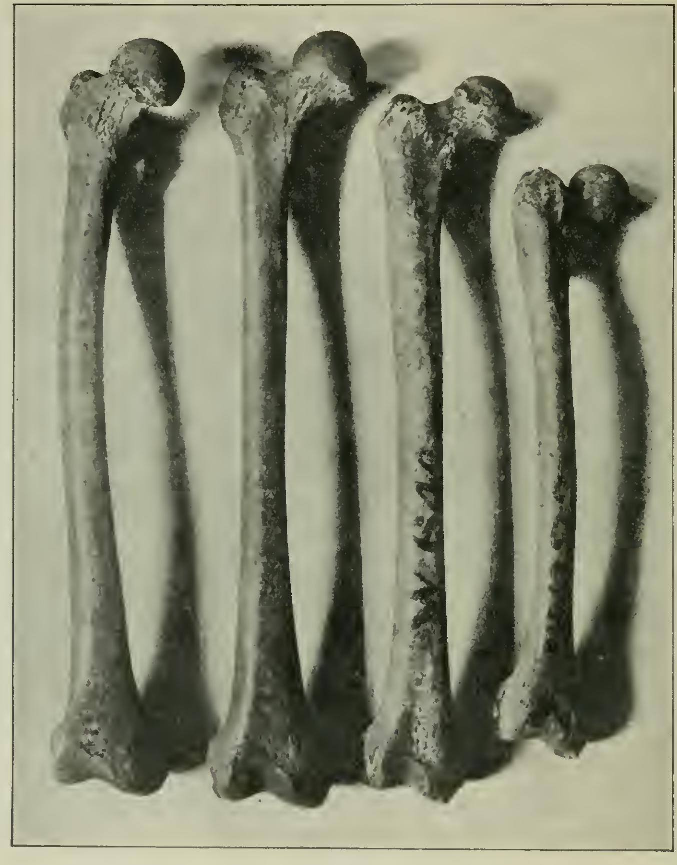


Fig. 2.

(Reproduced from Plate IV by kind permission of Professor Karl Pearson).

"Torsion of the femur. Right femora. When the plate is held vertically the bones are in the standard vertical plane, although not adjusted to the same standard (infra-condylar) horizontal plane. Starting on the left with large position torsion, we find the great trochanter is in contact with the standard vertical plane; we then pass through a case of moderate positive to one of slight negative torsion and conclude with a case of large negative torsion in which the head of the femur provides the third point of contact with the standard vertical plane. Note the disappearance of the small trochanter from the anterior aspect as the torsion becomes negative."

passively corrected by bringing the feet together, the whole femur rotates inwards, carrying the great trochanter forwards.

Note.—The normal position for the great trochanter with the child lying supine on a flat table in the standard position is exactly midway between the surface of the table and the upper surface of the thigh. In an older subject, it may be possible to demonstrate the condition radiographically; thus the small trochanter ossifies and casts a shadow at 12 or 13 years, and in the standard position described, if negative torsion be present, the trochanter will be twisted back out of sight. It is not likely that we shall ever be able to measure the amount of negative torsion in so many degrees: but shall have to be content with describing it as slight, moderate, or severe.

(5) *Treatment.*—The child must be taught to walk with its feet pointing straight forwards. If after several years of instruction and supervision the waddling gait persists, there appears no alternative to an osteotomy of the femur, followed by sufficient rotation inwards of the lower fragment to correct the deformity, and fixation in plaster.

(6) *Remarks.*—An interesting point for me has been the discovery by Professor Pearson that the left femur is normally much more twisted in than the right. One would expect from this that the right foot would be much more "turned out" than the left, but I know of no observations confirming this. It has, however, been suggested to me that this may be why a blindfolded person set to walk straight across an open space walks in a circle. The cause of the condition I take to be rickety softening of the bones: when one lies on one's back, there is always unconscious rotation out of the thighs due to gravity; in a rickety child this rotation affects the growing architecture of the bones.

(7) *Conclusions.*—To sum up: (1) There is normally a *positive* torsion of the shaft of the femur, this being much more marked on the left side than on the right. (2) Among 735 femora examined by Professor Pearson were thirteen bones in which the torsion was *negative.* The condition of negative or abnormal torsion therefore undoubtedly exists anatomically. (3) The condition exists clinically: no other hypothesis will satisfactorily explain the symptoms in the case I have shown to-day.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. J. P. Lockhart-Mummery, F.R.C.S.

Case of Excision of the Rectum, Four Years after Operation.[1]

By J. P. Lockhart-Mummery, F.R.C.S.

A. S., a clerk, aged 48, was admitted to St. Mark's Hospital in September, 1916. There was a history of bleeding and diarrhœa for six months, with loss of weight and pain in the sacrum and down the left sciatic nerve.

On examination there was a carcinomatous growth situated at the upper end of the rectum, fixed posteriorly, and causing considerable stricture. There were no signs of secondary deposits. Preliminary colostomy was performed through the left rectus muscle and a fortnight later the rectum was removed under a spinal anæsthesia by Mr. Lockhart-Mummery's technique.

The patient made an excellent recovery from the operation and is now in first-rate health and able to live an ordinary life.

The case is shown to demonstrate the small amount of disability resulting from this operation.

Case of Two Carcinomatous Growths of the Colon in the same Patient.

By J. P. Lockhart-Mummery, F.R.C.S.

C. J. S., aged 55, was admitted to St. Mark's Hospital on November 19 last. He had suffered with constipation since the previous July, alternated with attacks of diarrhœa and the passage of blood, poor appetite, and he had been losing weight. These symptoms had gradually increased up to the time when he was seen.

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[1] At a meeting of the Sub-section, held March 10, 1920.

On examination *per rectum* there was a large growth in the upper part of the rectum, the upper part of which was out of reach of the finger and appeared to involve the entire circumference of the bowel. The abdomen was slightly distended, but there was no evidence of secondary deposits. As the patient was suffering from a good deal of obstruction it was decided to perform a colostomy. On November 24 the abdomen was opened through the left rectus muscle. On opening the abdomen it was discovered that there was a second carcinomatous growth in the descending colon, just below the splenic flexure. This growth was of the scirrhus type. The rest of the abdomen was free from any secondary deposits. A transverse colostomy was performed.

The patient has done very well so far and was discharged a month after the operation. He is now attending the hospital for injections of copper colloids.

There are numerous instances of two growths occurring in the colon of the same patient, but they are sufficiently rare to make it desirable that all such cases should be reported. Mr. Littlewood[1] reported the case of a woman aged 52 with cancer in the splenic flexure and also in the rectum. He also reported the case of a man aged 69 with a growth in the ascending colon and another in the rectum. Mr. G. Simpson[2] reported a case in which there was a growth in the cæcum and another at the hepatic flexure. I operated on a lady some years ago for a growth in the rectum, and a fortnight later she developed intestinal obstruction due to another growth in the transverse colon. A patient of mine, aged 46, at St. Mark's Hospital, had no less than three adenocarcinomata in the descending colon and sigmoid flexure.

Case of Stricture of Rectum, probably Syphilitic.

By H. Graeme Anderson, F.R.C.S.

Patient, a female, aged 53, married.

History: Indefinite onset about fifteen years ago; constipation; discharge from the rectum of blood and mucus. Nine years ago had an operation at Guy's—and she subsequently passed bougies for three or four years. This became gradually more painful and she stopped it. Since then she has slowly lost weight, has passed blood and mucus, has had loss of sphincteric control, with occasional diarrhœa. Had no difficulties with labour. Two children born alive and healthy, one born dead, and one miscarriage. No operation for hæmorrhoids.

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[1] *Lancet*, January 11, 1907.

[2] *Brit. Med. Jour.*, December 7, 1907.

General condition: A thin woman, who looks ill and suffering from septic absorption. Abdomen distended; no free fluid. Rectum: Atonic condition of sphincters; 3 in. from anal margin there is a funnel-shaped stricture. This hardly admits the finger tip, and the ordinary sigmoidoscope will not pass. The walls are very hard and fibrous. There is copious pinkish purulent discharge. Impossible to say how far up the stricture extends. The surface is irregular and ulcerated. There are multiple ulcers of the left leg of nine years' duration. She had alopecia, sore throat, and ulcers on tongue and gums; old scars on knee, and left fauces; tongue smooth and glazed. Wassermann reaction positive.

Operation: Colostomy performed through left rectus muscle.

A Short Communication on a Case of Abdominal-perineal Excision of the Rectum, followed by Intestinal Obstruction and Gangrene of the Terminal Portion of the Colon.

By W. B. Gabriel, M.S., F.R.C.S.

The following are brief notes on a case which was in St. Mark's Hospital last year under the care of Mr. Aslett Baldwin, who has kindly allowed me to describe the case. The patient, a man aged 57, had a carcinoma involving the upper third of the rectum for which an abdomino-perineal resection was done on May 28, 1919. The operation was performed in the usual manner: the pelvic floor was carefully repaired with catgut sutures and the proximal end of the pelvic colon was brought out through a small incision in the left iliac region. A rubber catheter was tied into the colon and saline administered after the operation. Next day his condition was quite satisfactory.

On May 30, however, part of the terminal portion of the colon outside the abdomen was found to be gangrenous and was cut away; there was no evident cause for this; the finger entered the colostomy satisfactorily. The abdomen was slightly distended, but was flaccid and movement on respiration was good. Calomel, 5 gr., in split doses, was given orally, and in the evening a turpentine enema was administered through the colostomy and was repeated four hours later.

Next morning, May 31, the abdomen was more distended, there had been no result from the enemata, and the patient was hiccoughing. The gangrene of the colon had extended. At 2.30 p.m. Mr. Baldwin explored the abdomen under spinal anæsthesia. The median wound was opened up and a coil of greatly distended small gut presented; the contents of this were evacuated by means of a cannula. There was no obstruction in the region of the pelvic floor and it was then demonstrated that the obstruction was caused by the strangulation of a loop of small intestine which had passed up on the left side round the termination of the colon, between it and the parietes. The colon was compressed and the pressure on the sigmoid vessels accounted for the gangrene of the colon. The loop of small intestine involved was greatly congested: it extended up into the left hypochondrium and could not be reduced. The terminal gangrenous part of the colon was therefore excised and its stump reduced into the abdomen, the loop of small gut thus being freed. The blind end of the pelvic colon was closed with purse-string sutures and a loop of the distended transverse colon was brought out through the median incision; this incision was then rapidly closed and a drainage tube passed through the lateral wound. Finally the transverse colostomy was opened and a large bore Paul's tube tied in. At 5 p.m. a considerable amount of fluid fæcal material had drained away, but his general condition became progressively worse in spite of salines and cardiac stimulants and he died at 7 p.m.

This variety of obstruction has been noted previously by others. It would seem to be a possibility in every case in which a terminal colostomy is performed in this way, leaving an internal foramen in the left iliac region which is bounded internally by the colon as it passes forwards to the exterior. It may prove to be worth while as a routine measure to close this artificial and potentially dangerous aperture by inserting a few interrupted sutures between the mobile portion of the pelvic colon retained and the parietes, or by some other method of colopexy. This would add very little to the length of the operation, it should not interfere with the efficiency of the colostomy, and it should eliminate the risk of this unfortunate complication.

Case of Primary Chancre of Anus.

By C. H. Shorney Webb, M.S., F.R.C.S.

H. B., aged 25, a gardener, has been in the Army but never "went sick." No history of previous venereal disease. Three weeks and four days ago first noticed pain and a little swelling which he likened to a boil. No sore throat nor rash. He is now suffering from the condition seen, in addition to enlargement of the nearest lymphatic gland. Examination of material from a smear under the dark-ground illumination showed innumerable *Spirochætæ pallidæ*.

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Section of Therapeutics and Pharmacology.

President—Dr. H. H. Dale, C.B.E., M.D., F.R.S.

DISCUSSION ON THE TREATMENT AND MANAGEMENT OF DISEASES DUE TO DIETETIC DEFICIENCIES.[1]

Professor F. G. Hopkins, F.R.S.

Rightly or wrongly I have been held qualified to open this discussion. I appreciate the honour, but I recognize that in speaking to an audience rich for the most part in the clinical experience of which I have so little, my remarks ought to be limited in scope.

We are to discuss the treatment and management of diseases due to a deficiency in the diet, and not to deficiencies in the body itself. It is clear, however, that if a disease can be proved to be due to a definite and specific deficiency in the food consumed, its essential treatment and management, except in connexion with secondary issues, are narrowed down to a single procedure—the supply of what was deficient. Much of our discussion, then, must, it seems to me, still turn upon the question: What diseases, if any, have been proved to be due to a specific and ascertained defect in the diet? In the present state of knowledge and opinion concerning these diseases, or supposed diseases, I do not think that reference to ætiology can be kept out of the discussion of treatment.

The vitamine hypothesis has been passing through a stage which is usually illustrated in the history of any scientific hypothesis; the early stage, namely, during which what is new and unaccustomed in the hypothesis arouses perhaps undue enthusiasm in some, and uncritical opposition on the part of others. It has now, I believe, to enter upon the second stage, when sound and considered criticism will be applied to it. Its claims and limitations will have to be decided by yet further experiments and observations, aided by discussions such as this, in which laboratory and clinical experience come together.

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[1] At a meeting of the Section, held November 18, 1919.

It is unnecessary and would clearly be out of place for me to discuss the stages by which the hypothesis reached its present position. I would only comment upon the circumstance, which seems to me astonishing, that the facts concerning the ætiology of scurvy, i.e., facts which have been familiar for centuries, did not long ago lead to the conception at which we have now arrived. At this or that period the spirit of the time has blinded the observer; fashions in thought have led him astray. At one time the syphilitic taint was suspected in scurvy, at another an infective origin was taken almost for granted. Those who remained impressed with the obvious importance of the diet were content to think in terms of the known. Antiscorbutics were held, e.g., to be potassium salts or citric acid and related substances. Yet it really wanted very little discriminative study, given at last in the past few years, to show that the antiscorbutic effects of fresh food are due to substances of a quite specific type. It needed, however, additional facts from other sources to crystallize the conception of vitamines and deficiency diseases.

Scurvy indeed would seem to constitute a remarkable example of what is meant by a deficiency disease. Yet in illustration of what I have said about the approach of a period of criticism we may recall that one who has done as much as any other to establish the vitamine hypothesis, E. V. McCollum, has denied the right of scurvy to the title. He claimed, at least for a time, that the disease as seen in guinea-pigs, and presumably in other subjects, is due to intestinal stasis. The treatment of scurvy would call therefore for attention to the gut rather than for the administration of antiscorbutics, except in so far as the latter are aperient in action. But McCollum has, I understand, recanted, and how anyone with experience of laboratory experiments or of clinical cases could doubt the quite specific effect of antiscorbutics is hard to see. Certainly those familiar with the quantitative work done at the Lister Institute and especially the production of the disease and its prevention or cure in the monkey must be convinced or doubt their senses. I feel very sure that we shall find from later contributions to the discussion that experience with the troops during the war has brought abundant confirmation of the belief that scurvy is due to a specific food deficiency and that its rational treatment involves little more than control of the diet. One important point will, I think, be dealt with by later speakers and with more confidence than was possible only a few years ago.

Thanks to quantitative experiment it is now possible to classify materials in respect of their antiscorbutic content and indicate their relative curative potency.

The facts relating to infantile scurvy are more difficult to interpret. Remembering that, to judge from the experiments reported from the Lister Institute, milk contains no excess of the antiscorbutic factor, and in view of the instability of that factor towards heat, it is difficult to understand why, when children are fed so much upon milk which has been heated, scurvy is relatively so rare in infants. Possibly the child at birth starts with a certain store of the antiscorbutic vitamine in its tissues just as it starts with a store of iron.

I propose to say little about beri-beri. In support however of my suggestion that the vitamine hypothesis must now expect to encounter criticism and that its exponents may have to face difficulties, I would like to report that several officers of the Japanese Medical Service have recently expressed to me doubt as to whether the absence of the water-soluble vitamine is the sole determinant of the disease. According to them the improvement in the army diet has, at any rate, not had such unequivocal success in removing the disease as was seen when a similar improvement was made years ago in the food of the navy. I think myself that the mass of evidence for the relation between typical beri-beri and the dietetic deficiency is so great and so conclusive that it cannot be seriously shaken; but the Japanese experience, assuming that my informants have correctly expressed it, would seem to show that other factors may have to be taken into account.

In considering rickets as a deficiency disease we have to deal with facts perhaps less well established and with views less generally accepted. But the national importance of the disease makes the study of its ætiology of quite exceptional interest. If it be really shown that the prime and essential factor in its causation is a dietetic deficiency—whatever other factors may be accessory—its prevention becomes the clearest of issues. I for one, having seen much of Dr. Edward Mellanby's experimental work at first hand, am convinced that the influence of the vitamine associated with fats is an all important factor in the prevention of rickets, though the relation between the disease and the absence of the specific dietetic factors are certainly less simple than in the case of, say, scurvy. In experimental studies the results seem clear enough. In the majority of controlled observations variation in one factor and one only—the nature of the fat in the diet—may decide the issue between rickets and no rickets. There are, I think, quite certainly contributing factors, but, to judge from the experimental results, they are of minor importance as a whole.

It is not sophistry but common sense for those who believe in the importance of a dietetic factor in the causation of a disease to point out that in exceptional cases the disease may occur even when the diet has not the specific deficiency. The specific substance may, for instance, in a given case, be ill absorbed. The more I see of the action of vitamines the greater seems to me the probability that their function, or the function of some of them, is concerned in some way with the stimulation of internal secretions. If this be true the independent functional failure of an organ may induce the same symptoms as the absence of a normal external stimulus to its activity.

Again it must be remembered that, however real and specific the influence of a single dietetic factor may be, the response of the animal to variations in its amount cannot be independent of the balance of the diet as a whole, while the quantitative demand for the specific substance will vary with the rate of metabolism. Even the most prolonged fasts do not seem to have been associated with scurvy or with other specific symptoms of deficiency.

We have to think of considerations such as these before deciding that exceptional cases, in which, say, the occurrence of rickets seems to be more or less independent of the fats in the diet, can be held to disprove the relation. It is of course necessary that they should be exceptional. Speaking as an experimentalist I would express the conviction that there is more sound evidence for the dependence of rickets upon a deficiency in a specific factor associated with fats than for any other theory of its origin. If the evidence be accepted the essential basis of treatment is at once determined.

Last month a view was advanced in a lecture delivered here[1] which would once more (for the view has been advanced before) bring rickets under the head of disorders which arise from acidosis. Acidosis was claimed to be the result of all chronic conditions of malnutrition, whatever their kind or origin, and rickets is the final outcome. The author of this view, Dr. Eric Pritchard, has had great clinical experience, but he will forgive me if I say that some of the hypotheses used to support his views run counter to one's knowledge and instincts as a biological chemist. In any case the blood of rickety patients ought to be more thoroughly examined before acidosis can be accredited with so prominent a rôle in the ætiology of the disease.

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[1] E. Pritchard, "Causation and Treatment of Rickets" (Lecture delivered before the Fellowship of Medicine), *Brit. Med. Journ.*, 1919, ii, p. 627.

Whatever totality of factors may ultimately be proven to play a part in the ætiology of rickets it would, I think, be lamentable if the importance of fats were allowed to retreat once more from the forefront of our conceptions.

Of the peculiar eye trouble, the so-called xerophthalmia which appears in animals fed experimentally upon diets deficient in the fat-soluble vitamine I would speak with caution. In my experience it is extremely difficult to decide as to how far infection is concerned with the condition. McCollum looks upon it as a specific deficiency disease, and it is certainly striking to observe the rapid cure when proper fats are restored to the diet. Since Bloch has described the occurrence in more than fifty children in Copenhagen of what would certainly appear to have been a similar condition, due apparently to a similar cause, it becomes necessary to give it serious study. It must be very rare in this country but its occurrence suggests that in ophthalmic practice inquiries into diet should not be neglected in obscure cases.

In the case of pellagra we have a disease, which, if due to a dietetic deficiency at all, would seem to follow upon the lack of something other than vitamines. The evidence gathered during the war will be of the utmost importance in deciding upon this point.

Professor Boyd.

My contribution to this discussion has to do with the subject of pellagra. Numerous cases of this disease occurred amongst Turkish prisoners of war in Egypt and a committee was appointed to inquire into and report on its origin and treatment. As collaborators we had Professor Wilson, of Cairo, a physiologist; Professor Ferguson, of Cairo, a pathologist; Captain Roaf, bio-chemist; Dr. Woodcock, protozoologist; Captain Campbell and Captain Paton, bacteriologists.

It is unnecessary for me to enter into the clinical symptoms of the disease, it will suffice to say that all the patients showed erythema, digestive disturbances, with entire absence of hydrochloric acid in the stomach secretion, low blood-pressure, nervous phenomena and depression which is such a pronounced feature in pellagra. My object is to show the grounds for our conclusion that pellagra is a deficiency disease. We arrived at that view in part by a process of exclusion.

Defective sanitation, presence in the water supply of colloidal alumina and silicon have been cited as causes of pellagra, but investigation showed the sanitation to be satisfactory and the water supply excellent.

Metabolic experiments showed that there is a deficiency in the gastric acid secretion and consequent deficiency in pancreatic secretion. Food assimilation was imperfect in consequence, and a large destruction of protein occurred in the bowel through the action of micro-organisms tolerated by the defective acid secretion in the stomach. It was therefore not surprising to find a large amount of indican in the urine in early cases of pellagra. The quantity increased as the disease became more severe and diminished as it improved. There was much protein loss in diarrhœa.

No specific organism was found. Fifty bloods were examined for spirochætes, protozoa, ultramicroscopic organisms, &c., but nothing was found. Blood cultures were sterile. There was no increase of mononuclear cells so commonly found in protozoal infection. There was no fever in uncomplicated cases; no organisms were found in the urine in spite of numerous examinations. The cerebro-spinal fluid was normal. The stools of 220 patients were examined; these stools were always fœtid, they contained flagellates and most of them contained eggs of ascarides but no specific organisms were found.

Metabolic experiments showed a loss by the bowel in apparently healthy prisoners of 33 per cent. of protein and 19 per cent. fat instead of an anticipated 23 per cent. and 10 per cent. In pellagra patients the losses were 35 per cent. and 28 per cent. respectively. Apparently then these prisoners, though seemingly healthy, were in a pre-pellagrous state.

All proteins have not the same nutritive value. Some of them lack certain amino-acids and have a low "biological value." Thus if 30 grm. of meat protein suffice to maintain a man of 70 kilos in nitrogenous equilibrium, 108 grm. of maize protein are required to effect the same result. The "biological value" of maize protein is thus 0·29. It was found that pellagra was associated with a deficiency of biological protein for the needs of the individual. The energy value and total protein content of the dietary might vary within wide limits and a high calorie value did not compensate for a low biological value of the protein. Hard labour necessitates an increase in the biological protein.

Increase in the useful protein of the dietary was followed rapidly by a decline in the occurrence of pellagra cases. Patients on hospital diet to which an addition of extra beans was made improved more rapidly than others receiving an addition of butter, though the calorie values of the additions were the same.

We may conclude that a deficiency of biological protein is an essential factor in the causation of pellagra. Other factors such as digestive disturbances, and flagellate and helminthic infections are but contributory in that they increase the protein losses and are added causes of malnutrition. The association of pellagra with the consumption of maize is not due to the maize, nor to any toxin contained in it, but to the low biological value of maize protein.

(Professor Boyd showed charts and diagrams in illustration of his address.)

Dr. W. H. Willcox, C.B., C.M.G.

In this paper I propose to deal with the treatment and management of scurvy and beri-beri as they affect an army in the field. Among the many fascinating and interesting medical problems which presented themselves during the campaign in Mesopotamia those connected with scurvy and beri-beri occupied the foremost place. The deficiency diseases of scurvy and beri-beri occurred to a greater extent in Mesopotamia than in any other theatre of the War. The enormous wastage caused by scurvy in that campaign can be realized from the statistics available: thus 11,445 cases occurred in the last six months of 1916. These cases were almost all evacuated to India, since they were quite unfit to undergo the hardships of active service in the field.

The careful investigations made in connexion with these diseases in Mesopotamia and the results of the management—that is, the scientific rationing of the troops—have most conclusively established that both scurvy and beri-beri are essentially deficiency diseases and are due to a deficiency in the respective vitamines in the dietary associated with their causation.

From the beginning of May, 1916, until the end of January, 1919, as Senior Consulting Physician to the Mesopotamian Expeditionary Force, I made a special study of scurvy and beri-beri from their clinical aspects and from the point of view of prevention and treatment. I would like to take this opportunity of expressing my thanks to the Directors of the Medical Service, Major-General Sir Francis Treherne, K.C.M.G., and Major-General A. P. Blenkinsop, C.B., C.M.G., for the help and encouragement always given me in this work.

In order to understand clearly the problems which presented themselves in the Mesopotamian campaign in connexion with the management of scurvy and beri-beri it is necessary to review thoroughly the circumstances which effected the rationing of the troops in so far as they related to the occurrence of scurvy and beri-beri.

The Mesopotamian campaign commenced on November 6, 1914, when troops under General Delamain landed at Fao, and on November 22, 1914, Basrah was occupied, General Sir Arthur Barrett being in command. The Force pushed its way up the Tigris and after a series of brilliant military successes Kut was captured on September 28, 1915. From the beginning of the operations until 1916 the Mesopotamian Force was under the control of the Indian Government and the system of rationing troops was based on the Field Service Scale of Rations laid down in "War Establishments, India," pages 10 and 11: Table I, British rations; Table II, Indian rations.

TABLE I.

(A) *British Troops. Field Ration.*

(1) Bread	1 lb.	(6) Sugar	2½ oz.
(2) Fresh meat	1 lb.	(7) Salt	½ oz.
(3) Bacon	3 oz.	(8) Pepper	$\frac{1}{36}$ oz.
(4) Potatoes	1 lb.	(9) Fuel	3 lb.
(5) Tea	1 oz.		

TABLE II.

(B) *Indian Troops and Followers. Field Ration.*

(1) Atta	1½ lb.	(8) Ginger	⅓ oz.
(2) Fresh meat	4 oz.	(9) Chillies	⅙ oz.
(3) Dhall	4 oz.	(10) Turmeric	⅙ oz.
(4) Ghi	2 oz.	(11) Garlic	⅙ oz.
(5) Gur	1 oz.	(12) Salt	½ oz.
(6) Potatoes	2 oz.	(13) Fuel	1½ lb.
(7) Tea	⅓ oz.		

It may be stated now that scurvy in Mesopotamia was practically limited to Indian troops, only a few isolated cases occurring amongst British troops, the causation of which could be traced to inadequate dietary owing to some other diseases occurring in the individual patient. Beri-beri, on the other hand, was limited to British troops, practically no cases occurring amongst Indians. Some cases occurred amongst the Chinese Labour Corps at Basrah in 1918, and also in the crews on board ships arriving at Basrah. Both of these epidemics were traceable to polished rice as the ætiological factor.

A review of the ration scales shows that as regards deficiency disease the ration for British troops was fairly satisfactory. Attention will be called later to the deficiency as regards beri-beri under the conditions of service at the Front. For Indian troops the field service ration was very deficient as regards antiscorbutic vitamines, the only antiscorbutics being 2 oz. potatoes and 4 oz. fresh meat. This deficiency became much accentuated owing to the conditions of service at the Front. The great cause of the wastage in the Indian troops of the Mesopotamian Force up to the end of 1916 was scurvy. To understand this thoroughly it is necessary to call attention to the system of rationing of Indian troops in India prior to 1917. An allowance was made to each soldier equivalent to the cost of the items in the field service ration. He bought his ration from the bunniah or food contractor of his unit. The Indian has the instinct of saving money for his family very firmly implanted in his mind and under the old system of rationing Indian troops in India there was no guarantee that the Indian soldier bought and consumed even the field service ration. He no doubt often starved himself to save a few annas. As a consequence of this system the Indian troops arriving from India in Mesopotamia were found in many cases to contain a considerable percentage of men anæmic and debilitated and suffering from pyorrhœa and a ready prey to scurvy if put under hardships as regards the restrictions in diet necessitated by service in the field. They had little "balance in the bank" against the onset of the deficiency disease scurvy and their latent period would be short were dietetic hardships imposed on them.

My colleague, the late Colonel H. G. Melville, I.M.S., paid special attention to the examination of Indian troops arriving from India during the latter half of 1916, and he found at this time a considerable percentage of men on arrival to be anæmic, debilitated and suffering from pyorrhœa. These men were not actually suffering from scurvy but they were in a condition to be strongly predisposed to it.

The British soldier, on the other hand, came out to Mesopotamia well nourished and with a good "balance in his bank" against deficiency disease, and if he developed this his latent period would be a long one.

A glance at the map of Mesopotamia shows the military situation up to February 23, 1917, when the Tigris was crossed at Shumram and the Turks retreated towards Baghdad. From December 11, 1915, up to this date the operations of the Mesopotamian Force were directed towards the relief of Kut and the advance onwards to Baghdad. Basrah and Amara were centres of local production of fresh fruit and vegetables and the incidence of scurvy was very slight amongst the troops stationed there. Beyond Amara there were no centres of local production of fruit or vegetables and troops had to rely entirely on the river transport of supplies for these articles.

Attempts were made by the Army to grow vegetables for the troops, but it was impossible to do this in sufficient quantity since in Mesopotamia irrigation is essential for cultivation and a scheme for growing vegetables for the Army on a large scale would have taken a very long time to carry out. Also the art of growing fruit and vegetables successfully in Mesopotamia demanded a long and intimate experience of local conditions, and it was found that this could be much more economically and satisfactorily carried out by the native Arabs under our supervision, the produce being purchased from them by the Army.

During 1915 and 1916 the river transport was taxed to its utmost in conveying troops and supplies and in carrying sick and wounded downstream, and no special provision for the transport of fresh fruit or vegetables or fresh meat was available until towards the end of 1916. The climatic conditions of Mesopotamia were such that vegetables and fruit sent by river from Basrah arrived generally in a damaged condition unsuitable for issue as rations. It is thus obvious that the troops in the front area from December, 1915, to February, 1917, obtained few supplies of fresh vegetables or fruit; this explains the fact that up to February, 1917, there was an enormous incidence of scurvy amongst Indian troops in the front area in Mesopotamia.

ALTERATIONS OF RATIONS.

The incidence and causation of deficiency diseases in Mesopotamia was thoroughly appreciated and steps were taken to put the ration scales on a scientific basis as regards deficiency disease. The Army Commander, Sir Percy Lake, in 1916, with this object sanctioned the ration scales of July 4, 1916, which were a great improvement as regards the protection from deficiency diseases.

TABLE III.—RATION, JULY 4, 1916.

(A) *British Troops. Field Ration.*

(1) Bread	1 lb.	(10) Condensed milk ...	2 oz.
(2) Fresh meat	¾ lb.	(11) Salt	½ oz.
(3) Bacon	3 oz.	(12) Pepper	1/36 oz.
(4) Potatoes	1 lb.	(13) Wood	3 lb.
(5) Tea	1 oz.	(14) Oatmeal	4 oz.
(6) Sugar	2½ oz.	and Condensed milk	2 oz.
(7) Cheese	3 oz.	(15) Tobacco, weekly ...	2 oz.
(8) Rice	3 oz.	(16) Matches, weekly ...	2 boxes
(9) Jam	3 oz.		

Extras.

(1) Chocolate	1 oz.	(4) Dates	4 oz.
and Bread ...	4 oz.	(5) Fresh fruit	4 oz.
(2) Lime juice	½ fl. oz.	(6) Dry lentils	2 oz.
and Sugar... ...	½ oz.	Curry powder ...	⅛ oz.
(3) Rum	4 fl. oz.	(7) Limes	3 per man

TABLE III—(*continued*).

(B) *Indian Troops. Field Ration.*

(1) Atta	1½ lb.	(9) Matches, weekly ...	2 boxes
(2) Fresh meat	4 oz.	(9a) Condensed milk ...	2 oz.
(3) Dhall	4 oz.	(10) Tea	⅓ oz.
(4) Ghi	2 oz.	(11) Ginger	⅓ oz.
(5) Gur	2 oz.	(12) Chillies	⅙ oz.
(6) Potatoes	2 oz.	(13) Turmeric	⅙ oz.
(7) Fresh fruit	2 oz.	(14) Garlic	⅙ oz.
(8) Tobacco, weekly ...	2 oz.	(15) Salt	½ oz.
(Substitutes same as per British troops)		(16) Fuel	1½ lb.

Extras.

(1) Atta	½ lb.	(4) Fresh vegetables ...	4 oz.
(2) Ghi...	1 oz.	(5) Fresh fruit	4 oz.
or Gur	2 oz.	(6) Tamarind	2 oz.
(3) Fresh meat	2 oz.	(7) Rum (25 per cent. under proof)	2 fl. oz.

Later the rations were further improved and the Army Commander, General Sir Stanley Maude, who took a very great interest in the prevention of scurvy and beri-beri, sanctioned the ration scales of October 31, 1916.

TABLE IV.—RATIONS, OCTOBER 31, 1916.

British Troops.

Daily.

	lb.	oz.
(1) Bread (or Biscuit, 12 oz. when bread not available)	1	0
(2) Fresh meat (or preserved, 12 oz., when fresh not available) ...	1	0
(2A) Pickles, when preserved meat is issued		1
(3) Bacon		3
(4) Potatoes or fresh vegetables (or dried vegetables, 3 oz., when fresh not available)		12
(5) Tea		¾
(6) Cheese (not in summer)		3
(7) Sugar		3
(8) Jam or golden syrup		3
(9) Tinned milk		2
(10) Salt		½
(11) Fresh fruit (or tinned fruit, 2 oz., or dried fruit, 1 oz., when fresh fruit not available)		2
(12) Soup or Oxo (not in summer)		2
(13) Fuel (coal 1 lb., wood 1 lb.)	2	0

Weekly.

	lb.	oz.
(14) Pepper		¼
(15) Mustard		¼
(16) Tobacco (or cigarettes, No. 40, or sweets, 4 oz.)		2
(17) Matches ... ... (boxes)		2

Twice weekly.

	lb.	oz.
(18) Marmite, Monday and Thursday (not in summer)		¼

TABLE IV—(*continued*).

	lb.	oz.
Thrice weekly.		
(19) Oatmeal (Monday, Wednesday and Friday)		3
(20) Tinned milk (Monday, Wednesday and Friday)		1
(21) Rice (Tuesday, Thursday and Saturday)		2
(22) Curry powder (Tuesday, Thursday and Saturday)		⅛
(23) Butter (Monday, Wednesday and Friday), not in summer ...		2
(24) Lime juice (Tuesday, Thursday and Saturday), not in winter ...		½
Indian Troops.		
Daily.		
(1) Atta or rice	1	8
(2) Fresh meat (or gur, 2 oz,, when fresh meat not available) ...		6
(3) Dhall		4
(4) Gur		2
(5) Ghi		2
(6) Potatoes or fresh vegetables (dried vegetables, 2 oz., when fresh not available)		6
(7) Fresh fruit (tinned fruit, 2 oz., or dried fruit, 1 oz., when fresh fruit not available)		2
(8) Tea		⅓
(9) Milk, tinned		2
(10) Condiments (ginger, chillies, garlic, turmeric)		¾
(11) Salt		½
(12) Tamarind or coccum		2
(13) Fuel (wood)	2	0
Thrice weekly.		
(14) Ghi (Monday, Wednesday, Friday)		2
(15) Lime juice (Tuesday, Thursday, Saturday), not in winter ...		½ fl. oz.
Weekly.		
(16) Tobacco (Sundays) or cigarettes, No. 40, or sweets, 4 oz. ...		2
(17) Matches ... ... boxes		2

REVIEWING THE RATION SCALES OF OCTOBER 31, 1916.

As regards protection against scurvy the Indian ration contained: Potatoes or fresh vegetables, e.g., onions, 6 oz.; fresh fruit (limes, &c.), 2 oz.; fresh meat, 6 oz.; tamarind or coccum, 2 oz.; lime juice, ½ oz. three times a week. The only difficulty was the conveyance of this ration to the troops. The fierce heat of Mesopotamia rendered the carriage of perishable articles such as vegetables, fruit and fresh meat up the 300-mile journey on the Tigris an impossibility without special provision as regards transport. Up to the latter part of 1916 there was an inadequacy of transport both on river and land to cope with this great problem. Towards the end of 1916 the transport became much improved and refrigerator barges with cold storage chambers for fresh meat arrived, which were of great value. Major-General Sir G. MacMunn, K C.B., Inspector-General of Communications, took a great interest in the problem of conveying antiscorbutics to the troops on the front and devised crates for carrying fresh vegetables and fruit, which were of great value in protecting them from damage during transport.

Fresh Meat.

The evidence from the Mesopotamian campaign proved that this is of important antiscorbutic value. During 1916 when the troops at the Front, both British and Indian, were unable to get fresh vegetables or fruit for long periods the only protection that the British had above that of the Indians was the fresh meat allowance—1 lb. daily. They were unable to get this, but usually two or three issues were obtained from local supplies of Arab sheep, and on other days tinned beef would be issued. The Indian troops did not care for meat and usually had only one or two rations of 6 oz. per week. On other days owing to their caste prejudices tinned meat could not be issued.

Further evidence of the value of fresh meat was obtained by investigations carried out in 1916 by Nos. 9 and 10 Indian General Hospitals by Major Marjoribanks, I.M.S., on Indian patients suffering from scurvy. Two parallel groups of scurvy cases were treated on identical lines as regards diet: one group of patients was given a daily ration of fresh meat juice, and these improved much more rapidly than the other patients. I saw these patients several times in consultation and can testify to the striking antiscorbutic value of raw meat juice. The climatic condition of Mesopotamia and the risk of parasitic infection prohibited the use of raw meat juice in the treatment and prophylaxis of scurvy. In the treatment of cases of scurvy in hospital the beneficial effect of a liberal allowance of fresh meat was very striking.

Tamarind in all probability has antiscorbutic properties. It was taken by the Indian troops either as a chutney with stewed meat, or as an infusion with sugar, which formed a palatable acid drink. Coccum is a sort of dried plum, which appeared to have no antiscorbutic value. It has had a reputation in India as an antiscorbutic, but it was not liked by the Indian troops and was little used in Mesopotamia.

Lime Juice.

The ration lime juice up to the end of 1916 had no antiscorbutic value and had no beneficial effect on patients suffering from scurvy. It usually arrived in Mesopotamia after a long journey overseas, and was probably six months or more old before issued. At my suggestion in August, 1916, fresh lime juice was prepared in India from fresh limes, a small quantity of alcohol (5 per cent.) and salicylic acid (2 gr. to the pint), being added as a preservative. This was sent to Mesopotamia in special casks with the date of preparation marked on it. This gave better results as regards antiscorbutic properties and was used in the treatment of patients suffering from scurvy as well as for issue to troops.

Lime-juice as a prophylactic against scurvy is of uncertain value, since it is difficult to ensure its delivery to an army in the field within three months of its preparation, after which time much of its antiscorbutic value is lost. Undoubtedly when fresh it has important antiscorbutic properties, which I saw demonstrated on many occasions in patients suffering from scurvy. After the occupation of Baghdad lime juice was prepared from limes and bitter oranges obtained locally and preservative was added as above described. This was issued to troops with as little delay as possible, and had an undoubted value as an antiscorbutic.

The recent work of Miss Chick and Miss Hume, showing the superior value of lemon juice to lime juice is of great value and interest.

During the deficiency of fresh vegetables and fruit in 1916, in consequence of the great incidence of scurvy in Indian troops, an order was issued that Indian troops should have the first call on the fresh vegetables and fruit available.

BERI-BERI IN RELATION TO THE RATION SCALES.

Indian.—This ration is highly protective as regards antiscorbutic vitamines. Atta was the usual issue to Indian troops, although rice was an alternative. Atta is a wheat flour containing the germ and some of the aleurone layer of the grain, so that it is rich in anti-beri-beri vitamines. Dhall contains the coat and germ of the lentil and is rich in antiscorbutic vitamines.

British.—In 1915 over 300 cases of beri-beri occurred amongst British troops. The Indian Field Service British ration was protective against beri-beri but owing to the inadequacy of transport and the long distances from the base of the troops at the front, fresh meat and vegetables were not available so that the basis of the ration was tinned beef, bread or biscuits, jam and tea. The British bread and biscuits were made of white flour from which the germ and aleurone layer had been removed, thus depriving them entirely of anti-beri-beri vitamines. Hence the British soldier as regards his vitamine ration became liable to beri-beri and in many cases fell a victim to this disease. In the July, 1916, ration, oatmeal, 4 oz., and dhall, 2 oz., were added to the British ration to supplement the British vitamine deficiency. In October, 1916, marmite, ¼ oz., three times weekly was added to the ration.

Marmite.

Beri-beri cases occurred in the Dardanelles. I saw practically all of these cases, and, at the suggestion of Colonel C. J. Martin, advised the treatment with yeast, which gave satisfactory results. In February, 1916 (vide *Lancet*, March 11, 1916), I suggested to the Sanitary Committee of the War Office the advisability of an extract of yeast being issued to the troops as a prophylactic against beri-beri. Experiments were instituted at the direction of General Sir William Horrocks, K.C.M.G., and carried out by Professor Starling, F.R.S., Dr. S. M. Copeman, and their co-workers. These showed that extract of yeast was a prophylactic against beri-beri, and a preparation known as marmite was issued to British troops in Mesopotamia in October, 1916. Marmite can be mixed with warm water and taken like bovril or be added to a stew after cooking. Marmite was undoubtedly of great value as a prophylactic against beri-beri in Mesopotamia, and it was a valuable remedy in the treatment of cases that occurred. It kept well in Mesopotamia, and no difficulty was experienced in its issue as a ration to the troops.

In February, 1917, at my request, experiments were carried out at Amara to determine the practicability of issuing bread to British troops made with a certain proportion of atta in the flour. Loaves were made with flour containing 100 per cent., 75 per cent., 50 per cent. and 25 per cent. respectively of atta. In all cases the bread was very palatable but the addition of atta caused some difficulty in the rising of the dough so that the bread was somewhat heavier. Bread made with 25 per cent. of atta was quite as palatable, and differed little from ordinary bread except in the slightly brownish colour. For a trial the bread was issued to several units at Amara with satisfactory results, and later on a general issue of this bread on three days a week to the Army was sanctioned by the G.O.C. in C.

The issue of bread to British troops rich in anti-beri-beri vitamines was undoubtedly an important factor in the reduction of beri-beri in British troops.

Rice.

This article of diet as a causal factor of beri-beri amongst the troops did not attract much attention because, as already mentioned, in its place, atta was almost entirely issued to Indian troops. However, in 1918, a Chinese labour corps arrived at Basrah, and the men of this unit refused to eat any rice except the polished variety, and in consequence a number of beri-beri cases occurred amongst them. The difficulty was overcome by the issue of atta in place of a portion of the rice ration and afterwards the outbreak of beri-beri subsided.

After the occupation of Basrah, on March 11, 1917, local supplies of fresh vegetables and fruit and fresh meat became available in ample quantity. The purchase of supplies was undertaken by the Department of Local Resources, the director of which (General Dickson, C.M.G., C.B.E.) took the greatest pains in ensuring the issue of these articles to the troops throughout the advanced area.

It was owing to the adequate supply of fresh vegetables, fruit and fresh meat that deficiency diseases were stamped out from the Mesopotamian Expeditionary Force after March, 1917.

STATISTICS.

Scurvy in Indian troops and beri-beri in British troops were very prevalent up to July 1, 1916, but no correct statistics were available up to this date. From July 1, 1916, scurvy and beri-beri were made notifiable diseases in the Army. From July 1 to December 31, 1916, 11,445 cases of scurvy occurred in Indian troops. In 1917 2,199 cases occurred, and in 1918 only 825 cases although the force had largely increased in 1917 and 1918.

TABLE V.—SCURVY AND BERI-BERI.

Total figures, July 1 to December 31.

	1916 Last six months	1917	1918
Scurvy—			
Indians	11,445 ...	2,199 ...	825
Beri-beri—			
British	104 ...	84 ...	51

Previous to the end of February, 1916, *335* cases of beri-beri were recorded as occurring in the Mesopotamian Expeditionary Force.

As regards beri-beri, records are available showing that 335 cases occurred up to the end of February, 1916. In the last six months of 1916, 104 cases occurred in British troops. In 1917 only eighty-four cases occurred, and in 1918 only fifty-one cases occurred amongst British troops.

The attached curve shows for 1917 and 1918 the incidence of scurvy, the maximum being in May and June. The incidental rates are the admissions to hospital per mille of Indian strength.

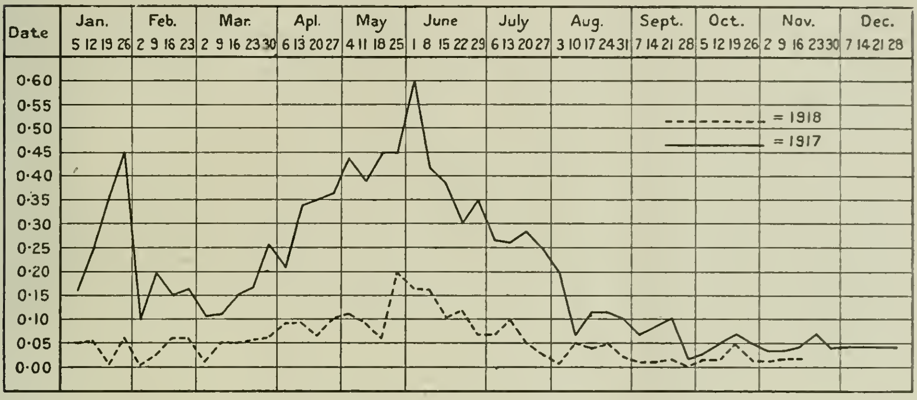

TABLE VI.

ÆTIOLOGY.

Undoubtedly the prime causes of scurvy and beri-beri are vitamine deficiencies; other factors, however, play a part as predisposing causes.

Climatic influences produced the maximum incidence of scurvy in the hot months of May, June and July, while beri-beri attained its maximum in November and December. Mental depression is mentioned by Osler as a predisposing cause in scurvy. In Mesopotamia the depressing influences associated with the campaign in 1916 were undoubtedly factors in the causation of the high incidence.

Intercurrent Diseases.

Patients suffering from diseases such as dysentery, the enteric group, and epidemic jaundice, which required careful dieting, would rapidly develop scurvy unless care were taken that antiscorbutics formed a part of the dietary. It was remarkable how quickly scorbutic symptoms would develop in Mesopotamia in patients on a dietary devoid of antiscorbutics. I have seen several cases develop typical scorbutic gums within six weeks of admission to hospital. It became a rule, therefore, to add to the diet of all patients in hospital a sufficiency of antiscorbutics—e.g., lemons, limes, oranges, &c., in order to prevent the development of scurvy.

In the Dardanelles campaign I called attention to the occurrence of beri-beri in patients recovering from an attack of epidemic jaundice. The cause of the beri-beri was undoubtedly the absence of anti-beri-beri vitamines in the dietary for the jaundice. A few cases of scurvy occurred in British troops in men with very carious teeth who avoided eating any fresh fruit or vegetables on account of the difficulty of mastication. I saw one case in a man who had an intense dislike to fruit or vegetables of any kind.

"Starvation" *per se* does not cause scurvy or beri-beri. In May, 1916, I examined a large number of sick who had been in Kut from December 11, 1915, to April 29, 1916, during the period of its siege. Many of these showed signs of acute starvation, but the signs of beri-beri and scurvy were not usually manifest in them.

TREATMENT OF SCURVY AND BERI-BERI.

In order to obtain effective control of these diseases, in June, 1917, special hospitals were established in the centres Baghdad, Amara, and Basrah for scurvy and beri-beri—an Indian hospital for scurvy and a British hospital for beri-beri. A special medical officer was attached for duty in each of these hospitals, and special registers were kept in which all the scurvy and beri-beri cases were fully recorded. In this way the occurrence of scurvy or beri-beri in any unit was at once recognized, and also uniformity of diagnosis and treatment was ensured. It was ordered that all the cases were to be seen by the consulting physician of the respective areas, who would advise as regards management, treatment, and disposal.

Scurvy.

The symptoms of scurvy exhibited by the individual cases called for special lines of treatment, thus anæmia and debility were the early symptoms.

Gum Changes.—The hyperplastic condition of the gums, with characteristic red buds between the teeth often shown best on the buccal aspect, was the most common symptom. In some cases the gums were so swollen as to have an appearance resembling a new growth. *Pyorrhœa*, which is quite a distinct disease from scurvy, often accompanied it, and no doubt by its presence tended to accentuate the symptoms of scurvy.

Palate Changes.—Petechiæ, or patches of hæmorrhage, occurred in the soft and hard palate, and if old had a pigmented appearance, also a characteristic dark crimson or purplish area of discoloration commonly extended from the inner aspect of the molar teeth upwards towards the palate. This sign was very common, and a very valuable early indication of scurvy. The palate itself was very pale owing to the marked anæmia present.

Muscle Hæmorrhages.—These were very common (about 30 per cent.), and usually occurred in the calf or thigh muscles or around the popliteal spaces or anterior tibial region, forming a hard brawny swelling, hot to the touch and tender on pressure.

Œdema.—Scorbutic œdema of a firm brawny type occurred in the legs and feet and around the tendo Achillis; this œdema was of quite a different type from cardiac or renal dropsy, and often showed tenderness on pressure. It was too firm to pit readily. *Subperiosteal swellings*, e.g., along the ulna or tibia, were fairly common. *Effusion* into the joints of a hæmorrhagic type sometimes occurred.

Skin.—In Indians skin manifestations were very difficult to determine owing to the natural pigmentation of the skin. In white races purpuric patches are very common, and a general earthy or bluish appearance of the skin is frequently present.

Subjunctival hæmorrhages occurred in a few cases. *Hæmorrhagic pleural effusion* was seen by me in six cases. Some cases showed hæmorrhage from the bowel, which required careful differentiation from dysentery.

Dilatation of the heart was usually present in severe cases, and was an important indication of the period of rest in bed. Hæmic murmurs usually accompanied the dilatation.

Captain A. J. Stevenson, M.C., R.A.M.C., was appointed M.O. in charge of the scurvy wards in the special scurvy hospital in Baghdad, and he did valuable work. The following scheme of treatment for scurvy cases in Indians was drawn up by us:—

(1) *Rest in bed* was essential as long as the anæmia or cardiac dilatation persisted, also the presence of muscle hæmorrhage, œdema, periosteal or joint hæmorrhages, or other severe hæmorrhagic symptoms demanded complete rest in bed.

(2) *Local Treatment of the Mouth and Gums.*—A solution of ½ per cent. salicylic acid in alcohol was applied to the gums twice daily,

and also a mouth-wash of alum and carbolic acid was used frequently. Dental treatment, such as scraping of the teeth to remove tartar, and removal of carious teeth was carried out.

(3) *Anæmia.*—A mixture of iron and arsenic was given for this in addition to the other measures.

(4) *Diet.*—6 a.m., tea and biscuits, and 2 oz. fruit; 8 a.m., ½ oz. fresh lime juice; 10.30 a.m., chapatie or rice with 8 oz. vegetables and two pints fresh milk; 12 noon, ½ oz. fresh lime juice; 2 p.m., 10 oz. fresh fruit; 7 p.m., 14 oz. meat, 8 oz. vegetables. Tomatoes, cucumbers and onions were given raw. Other vegetables were boiled for twenty minutes.

The accompanying tables show the selection of fruit and vegetables obtainable in the Baghdad area :—

TABLE VII.—VEGETABLES OBTAINABLE IN THE BAGHDAD AREA.

A.		*B.*	
Summer planting commencing from the month of February.		Winter planting commencing from the month of September.	
Onions.	Tomatoes.	Cabbage.	Turnips.
French beans.	Cucumber.	Beans.	Radish.
Haricot beans.	Pumpkin.	Spinach.	Cauliflower.
Brinjals.	Melons.	Beetroot.	Lettuce.
Lady's finger.	Water melons.	Carrots.	Purslain.

TABLE VIII.—FRESH FRUITS OBTAINABLE IN THE BAGHDAD AREA.

C.		*D.*	
Fruit produced during the summer locally.		Fruit produced during the winter locally.	
Apricots.	Figs.	Oranges.	Limes.
Apples.	Dates.	Tangerine oranges.	Quince.
Peaches.	Pears.	Sour oranges.	Pomegranates.
Plums.	Mulberry.	Lemons.	Citron.
Grapes.			

One of the most effectual remedies for scurvy is raw potato salad. It is made by cutting raw potatoes into very fine slices and adding slices of onion and a little vinegar. Captain A. L. Shepherd, I.M.S., at my suggestion tested this in 1916 on cases in the scurvy camp at the front area. He found that it gave the best results of any of the antiscorbutics he was then using.

(5) *Physical exercises* were given for twenty minutes twice a day to those patients sufficiently well to undergo them. In a few isolated cases the muscle hæmorrhages suppurated. These cases required surgical treatment—incision, &c. The cases of scorbutic hæmothorax required treatment by aspiration in addition to the other treatment of scurvy.

(6) *Germinated Dhall.*—The discovery in 1917 by Miss Chick and Miss Hume of the value of this as an antiscorbutic has resulted in supplying a very valuable antiscorbutic prophylactic to forces in the field where no fresh vegetables or fruits are available. It was unfortunate that this discovery was not made during the trying periods of 1915 and 1916 in Mesopotamia, when antiscorbutics were unavailable for the troops at the front. After May, 1917, germinated dhall was used in outlying districts in Mesopotamia as a ration when fresh vegetables or fruit could not be supplied. The remedy was also employed in hospitals for the treatment of scurvy cases, but it had no advantage over the use of fresh fruit and vegetables, and was less palatable. The great value of germinated dhall is that it can be used as a good substitute for fresh fruit and vegetables when these are not available. It is not meant to take the place of these.

Beri-beri.

In a country like Mesopotamia, where toxic influences causing multiple neuritis were prevalent, the most careful examination was necessary to exclude such cases of multiple neuritis as: (*a*) dysentery, (*b*) enteric or paratyphoid fever, (*c*) effects of heat, (*d*) malaria, (*e*) sandfly fever, (*f*) diphtheria, since these not uncommonly caused symptoms of multiple neuritis like beri-beri. The early signs of beri-beri were those of cardiac weakness accompanied by symptoms of multiple neuritis, the disease being associated with vitamine deficiency as regards diet.

A valuable early diagnostic test for troops consisted in the squatting test which I described in the *Lancet*, March 11, 1916. In this paper the treatment for beri-beri cases was carefully dealt with. In Mesopotamia the cases were treated on similar lines to those described, the only difference being that marmite was used in place of yeast, it being given three times a day.

CONCLUSION.

The experiences of the War, especially in distant countries such as Mesopotamia, called for special attention to the scientific rationing of troops. The old idea of sufficiency of calories, or of proteins, fat and carbohydrates is quite inadequate to meet the needs.

A dietary to be satisfactory must contain proteins, fat and carbohydrates in the proper amount and also must satisfy the calorie requirements. In addition it is essential that it should be adequate as regards vitamine content, e.g., it must be protective against scurvy, beri-beri and other deficiency diseases.

Too often financial considerations predominate, and I cannot let this opportunity pass without calling attention to the action of the Indian Government in the matter of the rationing of the Indian soldiers serving in India. On February 17, 1917, a ration was sanctioned by the Indian Government for Indian troops in India. This ration repeats the field service ration which had been responsible for the enormous outbreak of scurvy in the Indian troops in Mesopotamia, and indeed it is inferior as regards antiscorbutic value for it entirely lacks any fresh meat allowance. Under a ration of this kind scurvy is likely to be prevalent amongst Indian troops in India, and if at any time they are called upon for active service there is bound to be a great wastage owing to the incidence of scurvy. This method of rationing may appear economical to those without special knowledge, but to those who have studied the subject it is a false economy. It seems incomprehensible that after the tragic experiences of Mesopotamia the same mistakes should be repeated.

Since the rationing of troops or of a civil population is carried out usually by the laity not possessing special medical knowledge, the education of the lay mind in the scientific principles which form the basis of a sound dietary is of the utmost importance. For this purpose in Mesopotamia the "Official Memoranda" on scurvy and beri-beri which had been carefully drawn up were circulated not only to all the medical units but to the O.C.s of all combatant units in the Force. The education of the fighting forces in the principles of rationing as regards protection from the deficiency diseases scurvy and beri-beri was of great value, for the Commanding Officers took the greatest care to see that their troops received those articles of their dietary which were essential for their protection.

Dr. MACKENZIE WALLIS.

My remarks this evening refer more particularly to beri-beri. Experiments carried out in India upon the food value of the ground-nut, known also as the monkey nut, pea nut or Arachis nut, led to the fact that it was remarkably rich in anti-beri-beri vitamine. In view of this both bread and biscuits were made from the nut in the following way: The thin red skin was first removed from the nut, and the arachis oil then expressed under high pressure without any heating. The resulting oil press cake when ground into a fine white flour, contained 44 per cent. of protein, and 5 per cent. of oil. This flour prepared in this particular way was used for the manufacture of bread and biscuits. After numerous trials a nutritious and palatable biscuit was obtained, and distributed to criminal tribe settlements, and subsequently to Indian troops. The biscuits were also used for the prevention and treatment of beri-beri. Bread made from the same flour was distributed to British troops, and favourably reported upon as to its nutritive value and palatability. The value of the biscuits as regards their anti-beri-beri vitamine content was proved by the experiments on birds. Very small quantities of the biscuit protected the animals against beri-beri when fed on a diet of polished rice which was known to produce beri-beri in control animals.

The use of such a biscuit as an emergency ration for troops has been strongly advocated from the point of view not only of its high vitamine content but also for its protein content and extreme cheapness compared to any other biscuit. The flour was used successfully in the preparation of a substitute for the German product "nutrose" and proved to be far more efficacious. For the preparation of bacteriological culture media the ground-nut flour yields a medium equal to meat in nutritive value, besides abundant vitamine essential for the proper growth of certain micro-organisms.

Dr. Roaf.

In considering the treatment of deficiency diseases there seem to me to be three indications. The first and obvious one is to make good the deficiency. With that end in view, as Professor Hopkins said, we must know what is the nature of the deficiency. The second indication is to see that the substance in regard to which there is a deficiency is properly absorbed. One might take, as an example, the fat-soluble vitamine. Possibly that would not be absorbed if the absorption of fats were deficient. The third indication is, to minimize the expenditure of any substance for which the deficient material is required, that is to say, we must understand the pathology of the disease if we are to prevent wastage of some rare and needed material.

I shall discuss pellagra only. I was associated with Professor Boyd in dealing with Turkish prisoners of war, and we considered that their deficiency was due in some way to a deficiency in the biological value of protein. The probable deficiency in pellagra is that of some aromatic amino-acids, though that has not been definitely proved. One must not lose sight of the fact that the countries in which maize is largely used are also countries in which there is more sunlight than there is in more northerly countries, and strong sunlight may be required to bring out the characteristic skin symptoms by which a diagnosis is helped. With regard to the aromatic amino-acids, it seems possible that tryptophane is the one which is deficient or lacking. It is interesting to note that the people who get pellagra suffer from indicanuria. That was pointed out to me by Professor Wilson. He said that if you examine the ordinary population few of those people have indicanuria, but the majority of pellagra patients have it. The association is probably this: In a country where the diet is somewhat deficient in tryptophane, those who suffer from bacterial putrefaction of tryptophane are more likely to have pellagra than those who do not suffer from a destruction of tryptophane. The experiments which showed that the pellagrous person was losing 35 per cent. of protein in his fæces were done by analysing the urine and the fæces. The pellagra patients were suffering from indicanuria, but none of the other patients who were examined had indicanuria, therefore the total loss to the body is greater than is shown by the analysis, because a certain amount of nitrogen in the urine is there as indoxyl-sulphate, the tryptophane having been destroyed in the alimentary canal.

Slight cases of pellagra always get well in hospital if a proper diet is given them: the treatment of pellagra is a matter of diet. This is found to be the case in Egypt, in America, and wherever the disease occurs. But there are also some severe cases, and the patients do not always get well: they may remain in hospital a long time, possibly six months, and then they die; or they may be in hospital six months and then suddenly begin to improve. If we examine the tissues of a case post mortem, we find the tissues are greatly wasted, and the walls of the intestines are extremely thin—as thin as paper even—so that they are semi-translucent. Perhaps the reason severe cases remain so long without improvement is, that there is a greatly reduced absorption from the walls of the intestine. Hence it is important to treat the condition of the intestines on general principles.

As has already been said, pellagrous patients suffer from a deficiency or absence of free hydrochloric acid, and I think one may well try giving "pre-digested" food to help on the organs of digestion which are failing; we must also treat such complications as dysentery, worms, and so on.

Now as to the indication for economizing the expenditure of the substance which is deficient, we do not know actually what is the deficiency in pellagra, but we have some evidence as to what is the underlying pathological factor. The symptoms of pellagra, corresponding to those of Addison's disease, point to the implication of the sympathetic nervous system. Unfortunately, the pathology of that system is as yet practically unknown. I have examined the sympathetic nervous system from certain cases of pellagra, also that system from certain Turkish prisoners of war who did not have pellagra. I found a distinct change in the ganglion cells of the pellagra patients: there is a marked degeneration—namely, plasmolysis. The cell may be apparently absent, that is to say it has shrunk to one side. That is a point upon which I hope to comment in a future publication, and if it can be substantiated it will be of some use in dealing with such a disease as pellagra. Thus in dealing with the disease, our object will be to spare the sympathetic nervous system as much as possible. Strains of the body which throw extra work on the sympathetic system, such as sweating, must be avoided, as that is due to activity of the sympathetic. It has some bearing on the fact mentioned by Dr. Boyd, that pellagra was largely found among the prisoners who worked. We found that some of the men could live on a certain diet when not working, but as soon as they worked they showed a tendency to develop pellagra. We had many cases of pellagra which started when the men were put on to work.

Miss HUME (representing Dr. HARRIETTE CHICK).

With regard to the comparative value of raw foodstuffs in preventing beri-beri and scurvy:—

(1) *To prevent Beri-beri in Armies.*—Marmite and wheatgerm are to be recommended, as being three or four times more potent than the next most potent foodstuffs known—egg yolk and dried peas.

(2) *To prevent Scurvy in Armies.*—Preserved lemon juice and germinated peas or beans are recommended. Raw lemon juice is four times as potent as raw lime juice, and retains its antiscorbutic vitamine well. The development of the antiscorbutic vitamine in seeds on germination was discovered by Dr. Valentine Fürst, of Christiania, and has been

confirmed by Dr. Chick and her co-workers. The discovery has been applied at Archangel, and by Dr. Wiltshire in the Balkans.

(3) *To prevent Scurvy in Infants.*—Any milk other than raw milk will be poorer in antiscorbutic vitamine, and a slight degree of deficiency may be produced in consequence; other antiscorbutics should therefore be given—i.e., orange juice, swede juice, or tomato juice; but carrot juice, meat or grape juice are much less concentrated. Orange juice is one hundred times more potent than raw milk. Even freshly dried milk may have lost half its antiscorbutic value.

Dr. MARION DELF.

The part of the work on this subject at the Lister Institute which was assigned to me was, primarily, that which related to the effects of temperature on the antiscorbutic vitamines. Other work had previously been done by Dr. Chick and Miss Hume, which, I think, remains the principal quantitative work on the antineuritic vitamine—i.e., that which protects against beri-beri. They were able to show that this vitamine is stable for a period of two hours when heated to 100° C., and that it also retains a good part of the stability when heated at even higher temperatures for periods of time varying from half-an-hour to one hour. There is very little other evidence of a satisfactory quantative nature as to the effect of heat on the anti-beri-beri vitamine, but there was an account published last year which suggested that this substance is destroyed if heated in an alkaline medium. This is interesting in connexion with some work which we may be hearing of later, I think, in connexion with antiscorbutic vitamine.

With regard to the effect of temperature on the fat-soluble accessory factor, the evidence is very conflicting, and, in view of the late hour, it will not be worth entering into. This divergence appears, to me at any rate, largely explicable by the fact that the fat-soluble vitamine has not been dealt with quantitatively with sufficient care in the unheated condition. It is impossible to estimate a temperature effect until we have some quantitative measure for the minimum protective dose in the raw condition.

More is known of the effect of heat on the antiscorbutic vitamine, both in the green leaf (cabbage) and in expressed juice of fruit (orange) and vegetables (swede, cabbage). In all these cases, it has been necessary first to find the minimal protective value of the raw substance when added to a nutritious but otherwise scorbutic diet. Young guinea-pigs were used as experimental animals.

TABLE I.

Substance	Treatment	Time (minutes)	Temperature °C.	Limiting protective ration	Percentage destruction	Author
Green cabbage	Raw ...	—	—	1 grm.	—	Delf, 1918
	Simmered ...	60	60	5	80	,, ,,
	Steamed ...	20	100	> 5	>80	,, ,,
	,, ...	20	90	5	80	,, ,,
	,, ...	120	130	<15	—	,, ,,
	Canned ... (2 weeks old)	60	90-100	> 7·5	>80	Campbell and Chick, 1919
Runner Beans	Raw ...	—	—	5	—	,, ,, ,,
	Canned ... (3 months old)	140	100	>20	>75	,, ,, ,,
Germinating lentils	Raw ...	—	—	5	—	Chick and Delf, 1919
	Boiled ...	15	100	>12	>60	,, ,, ,,

TABLE II.

Juice	Treatment	Time (minutes)	Temperature °C.	Limiting protective dose	Percentage destruction	Author
Filtered:						
Cabbage ...	Raw ...	—	—	<1·5 c.c.	—	Delf (unpublished experiments)
	Heated ...	60	100	>5 c.c.	>70	,, ,, ,,
Swede ...	Raw ...	—	—	2·5	—	Chick and Rhodes
	Heated ...	60	100	<5	<50	Delf (unpublished experiments)
	,, ...	60	130	>5<10	>50<75	
Fresh:						
Orange ...	Raw ...	—	—	1·5	—	Chick, Davey, and others
	Heated ...	60	100	1·5	—	Delf (unpublished experiments)
	,, ...	60	130	>2<3	>15<50	

In Table I the minimal daily protective amounts of three antiscorbutic substances are compared, in the raw state and after heating. In every case the minimum ration is much greater in the case of heated materials indicating a corresponding diminution in antiscorbutic value. In the case of the canned material (which was prepared at the Lister Institute), there was probably also some deterioration due to time of storage. When effects of different temperatures are compared, we find that the *time* of heating has more importance than the temperature used. Heating cabbage for one hour at 60° C. has about the same effect on its antiscorbutic properties as heating for twenty minutes at 100° C., but the softening effect of heating at the higher temperature for the shorter time is very much greater. The hay-box method of cooking is thus unsuitable for any substances of antiscorbutic value (fruit and vegetables).

In Table II the minimal protective doses of freshly expressed juices in the raw and heated condition are shown. It will be seen that cabbage juice loses about four-fifths of its value after being heated for one hour at 100° C. Swede juice is more stable at 100° C., but when heated under pressure at about 130° C. for one hour it loses more than half its value. Orange juice is still more stable at both these temperatures, even when the juice is nearly neutralized before heating.

Dr. A. Harden.

I would draw attention to the experiments of Dr. Robison and myself,[1] which show the possibility of evaporating fruit juices to dryness and preserving them for as long as two years without any very considerable loss of antiscorbutic potency. Experiments are in progress to carry out the process on a commercial scale, and Givens and McClugage in America[2] have done the same thing by a modification of the spraying process for drying milk.

Dr. S. S. Zilva.

I shall first discuss the antiscorbutic factor in lemon juice. Lemon juice like juices of similar fruits is antiscorbutically active, but owing to its great acidity it does not find a ready application as an antiscorbutic. It was our object at the Lister Institute to investigate the antiscorbutic factor in lemon juice with the purpose of isolating and identifying the principle chemically. It was first necessary to ascertain whether it was associated with the citric or organic acids, which as you know form the best part of the solids of lemon juice, or whether it was contained in the residue which remains behind after the acids have been removed. We adopted the following procedure in order to remove the acids: An excess of $CaCO_3$ was added to juice which was further treated with two volumes of absolute alcohol. It was then filtered and the filtrate was evaporated *in vacuo* at a low temperature until the alcohol was removed and the solution brought up to its original volume.

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[1] *Journ. Roy. Army Med. Corps*, 1919, xxxii, p. 48.

[2] *Amer. Jour. Dis. of Child.*, 1919, xviii, p. 30.

This solution, which was free from the acids and contained little solid matter, was found to have retained the greater part if not the whole of the antiscorbutic properties of the lemon juice. Moreover it could be concentrated to any volume and even be brought down to dryness under certain conditions without losing its potency. It became evident that this was a very potent antiscorbutic containing little extraneous matter and would prove of therapeutic value. A rapid cure effected on a monkey suffering from acute scurvy justified our anticipation. At this stage we were fortunate in securing the collaboration of Dr. Still, and the preparation was tried on scorbutic infants with success. We have since simplified our method by eliminating the alcohol treatment and the subsequent evaporation *in vacuo*. We find that by adding an excess of precipitated chalk to juice and filtering, an antiscorbutically active neutral solution is obtained which is well tolerated by infants. We think that owing to the simplicity of the preparation, which could even be carried out at home, this solution ought to find a wide application as an antiscorbutic prophylactic, especially in the case of infants who cannot take large quantities of fruit juice on account of its acidity.

The second point to be mentioned is the effect of incipient scurvy on the teeth of guinea-pigs. There is little reference in the literature to the structure of the teeth of scorbutic patients. All who have had the experience of handling scorbutic guinea-pigs must have noticed the abnormal macroscopic appearance of the teeth sometimes present. Major Wells and I therefore undertook the investigation of the teeth of scorbutic guinea-pigs histologically. Decalcified and stained sections of such teeth have shown disorganization of the cellular tissue of the pulp and the differentiated layer of cells of the pulp situated near the dentine. Incipient changes in the structure of the teeth were observed in guinea-pigs which had subsisted on a scorbutic diet for only a few days, although at that time no macroscopic scorbutic lesions could be discerned.

These observations will become important if analogous changes can be shown to take place in human teeth under similar conditions. You will agree with me that under the conditions in which we live at present, both adults and infants may feed on a diet not deficient in the antiscorbutic factor to the extent of producing declared scurvy but deficient enough to bring about changes similar to those referred to. This investigation is being pursued further at the Lister Institute, but the matter cannot be settled entirely in the laboratory. The co-operation of the observant clinician is needed, and that is the reason why I am raising the subject here to-day.

31.

Section of Therapeutics and Pharmacology.

President—Dr. H. H. Dale, C.B.E., F.R.S.

DISCUSSION ON THE VALUE OF ALCOHOL AS A THERAPEUTIC AGENT.[1]

Dr. H. H. Dale, C.B.E., F.R.S.

The aim of this discussion is to arrive at a clearer definition of the scope and value of alcohol as a therapeutic agent. It ought to be possible for any of us to hold a strong view, in either direction, as to the desirability of alcohol as an article of ordinary consumption by the healthy, without in any way prejudicing his opinion of its value in the treatment of the sick. It is the latter which we are asked to discuss to-day, as an independent and purely scientific question. We are asked to endeavour to put ourselves, by imagination, into the position of medical men practising in a country in which there is no "alcohol problem" of ordinary life—in a country, for example, which effectively prohibits the popular consumption of alcohol. The question before us is whether alcohol, under such conditions, would have a proper place in the Pharmacopœia, except as a solvent, and, if so, for what purpose it should be included. I venture to appeal to all who take part in the discussion to assist us in rigidly keeping it confined to the consideration of this scientific, therapeutic question, which is our only proper concern as a Section of Therapeutics and Pharmacology.

My own contribution to the discussion will be merely an attempt to review the physiological and pharmacological evidence as to the actions of alcohol which can be experimentally demonstrated. I shall leave it to those who follow to discuss the extent to which the action of alcohol in practical therapeutics fulfils, exceeds, or falls short of the expectations thus aroused.

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[1] At a meeting of the Section, held February 17, 1920.

In the first place, I think it must now be regarded as settled beyond dispute that alcohol can be used by the body as a source of energy, and that, in that limited sense, it can act as a food. The very careful experiments carried out especially by Atwater and Benedict indicate, indeed, that it is possible, for a limited period, to supply a surprisingly large fraction of the ordinary total energy-need of the body in the form of alcohol—as much as one-fifth. Dr. Mellanby, I believe, will have some new evidence to give us as to the limitation of the body's power to use alcohol. The question as to whether alcohol is a desirable substitute for part of the carbohydrate or fat in the diet of the healthy is not here involved. That is a question which each must determine by his view as to the significance of its other actions in normal life, and does not concern us to-day. Whatever our views on this question, we must recognize that a type of action, which may be deleterious to the normal activities of the healthy, may be harmless or advantageous to the sick. In the treatment of the sick, moreover, alcohol as a source of energy has certain points of positive advantage over the normal non-nitrogenous constituents of a diet. It requires no digestion, though in this respect it may be argued that it has no advantage over dextrose; it is not susceptible to such fermentation by yeasts or bacteria as to cause a deleterious production of gas in the alimentary canal; and it is absorbed with great rapidity. Reasoning purely from experimental knowledge of its properties, one would expect that there are conditions in which the digestive secretions are paralysed, as in acute fevers, in which absorption is imperfect, in which fermentation is liable to be excessive, and in which, therefore, the food value of alcohol should give it a place in therapeutics; its function being to supply to a system, unable to obtain it from the normal sources, the minimum of energy necessary to tide over a critical period. The discussion will doubtless enlighten us as to the extent to which alcohol can fulfil this rôle in practice. Concerning a very special case of its use for this purpose we shall hear, I believe, from Dr. Otto Leyton—namely, its use in the modern treatment of diabetes, during the period in which all other food is withheld, in order to render the urine free from sugar.

Entirely different from the question of this use of alcohol, as what I may call an emergency-source of energy, is its reputation with some practitioners in the diet of the convalescent. I do not think that any one will now contend that alcohol as such has a specific effect on metabolism, a power of "building up the system," such as that with which an earlier and uncritical generation credited especially certain liquors containing it. Such views have probably as much scientific basis as that which credits red wine with a special power of forming red blood. So far as any rational basis can be found for the use of alcohol in convalescence, it seems to be this: that certain patients are made more comfortable and contented, worry less and take their food with keener enjoyment, if they are given alcohol. The treatment, therefore, would seem to involve the giving of the alcohol in a form in which the patient enjoys it. It would hardly be maintained that the same quantity of the drug, administered in a distasteful mixture, would have the same effect. So that the use of alcohol in convalescence will probably be decided by the relative importance attached by individual opinion to its appetizing qualities, on the one hand, and the danger of forming a habit on the other. No uniformity of opinion can be expected on such a subject from a meeting like this, and I hope that this point will not be the subject of lengthy discussion.

We are led, then, to consider the other actions of alcohol which may have therapeutic significance: its action as a drug, as distinguished from its action as a food. Here again, a prolonged controversy, which formerly divided the world of experimental pharmacology, seems to be practically settled. Most of you will be aware that there were two schools of opinion, one, of which the leading exponent was Binz, which maintained that alcohol was a stimulant, and the other, led by Schmiedeberg, which maintained that in all its actions, other than that of direct irritation to mucous membranes, alcohol was a depressant and narcotic. The question seemed to have some practical importance, since a large part of the therapeutic reputation of alcohol was based on the assumption that it was a respiratory and circulatory stimulant. I think anyone who studies the experimental evidence will reach the conclusion that the view of Schmiedeberg and his followers was the correct one, so far as the normal heart and respiratory centre are concerned. The most recent series of experiments in America, conducted on normal men, failed to show that alcohol had any stimulant effect on the action of the heart, or the activity of the respiratory centre, in doses up to those sufficient to produce a mild intoxication. I think it may be taken as the generally accepted pharmacological teaching that alcohol has no stimulant action on any of the vital functions of the normal animal or man. The slight acceleration of the heart which often follows its ingestion is attributable, as Professor Dixon, among others, has shown, to the reflex effect of its direct action on mucous membranes. Its only other action on the normal circulation is the flushing of the general skin surface which it produces.

I think that it would be an important result of this discussion if those who will speak from wide clinical experience can do anything to clear up the discrepancy, between this lack of any sound experimental evidence, that alcohol is a stimulant and its still widely prevalent use in therapeutics as a stimulant of depressed or failing respiration and circulation. One must allow something, of course, for the greater hold of tradition on clinical practice than on experimental science; but I am not satisfied to regard this as the whole explanation of the anomaly. I think a good deal of weight may be allowed to the consideration that the experimental results have been obtained on normal subjects, while clinical experience is dealing with functions depressed, not merely by actual weakness, but by inhibitions due to reflex action or to influences from the higher centres. The weakening of such central inhibitions, which would be entirely compatible with the action of alcohol as demonstrated by experiment, would have the appearance of a stimulation; and from the strictly practical point of view it may seem a matter of minor importance whether the effect is really one of true stimulation or of mild narcotic action. If and when alcohol assists recovery from syncope due to fright or pain, I imagine that we have a condition in which the action of the heart and the vasomotor tone are subjected to severe central inhibition, and that alcohol helps to weaken and remove this inhibition. Dr. Haldane's recent work seems to show that excessive reflex stimulation, or excessive reflex excitability of the respiratory centre may, by causing respiration of the ineffective, rapid and shallow type, actually impair the oxygenation of the blood in the lungs. Administration of oxygen is the obvious remedy, but I do not think that the possibility can be excluded offhand, that the mild sedative action of alcohol may have a valuable adjuvant effect in such a condition. Dr. Willcox is to put before us some results of practical experience on the combined use of alcohol and oxygen in pneumonia, and I hope he will be able to give us his views as to the mode of action of this treatment. Anything will have value which, in such a condition, can assist in restoring the quiet, deep rhythm of effective respiration, in thus securing for the blood the maximum oxygenation which the still functional lung tissue can give it, and in thereby sending well oxygenated blood to a heart threatened with overloading and dilatation. I hope our discussion will enable us to form a clear opinion as to whether alcohol has any such action. If it has, it is a valuable drug. The issue has been too long obscured by the discussion as to whether it is a stimulant or a narcotic, as though a stimulant action were necessarily always beneficial and a narcotic action always to be avoided. Recognizing that alcohol is essentially and always a mild depressant and narcotic in its action, we should recognize also that such an action may have valuable therapeutic application in the weakening of excessive and deleterious inhibitions, and in controlling a morbid reflex excitability. It may be added that, assuming that such mild sedative action on the centres is what is required, the claim might be made for alcohol of a special position, in that it is the only narcotic drug at our disposal which at the same time materially contributes to the energy requirements of the body.

But while care may be necessary, lest we refuse to recognize what may be a beneficial action of alcohol, because of a prevalent misconception of its mechanism, I think it will be generally admitted that alcohol is still prescribed in a very indiscriminate and unscientific manner. A drug is of real value in therapeutics in proportion as we know clearly what it can and what it cannot do. I hope that this discussion may at least make an attempt towards a closer definition of the conditions in which alcohol is demonstrably valuable, and those in which it is useless or harmful. I suspect that a large proportion of the conditions in which it is professionally recommended, to say nothing of those in which it is used by the laity for self-medication, would fall into the latter category. Our business, I take it, is primarily to discover the condition in which it has value, and only incidentally to deal with those in which it has none. Perhaps some contributor will find time to deal with its reputation as a prophylactic against catarrhal infection, and, especially at the present time, against influenza. We shall agree, I imagine, that there is no evidence whatever to warrant the assumption that alcohol has a specific action against infection of any kind. As to its use against the lowering of resistance by chill, I suppose that the common-sense interpretation of its action is as follows: There is no doubt that it causes an acceleration of blood-flow through the skin, so that its use as a preliminary to, or during, exposure to cold would appear to be dangerous. After exposure, as an aid to the effect of external warmth, it may have value, by promoting superficial vaso-dilatation. Doubtless other particular examples of its action will appear in discussion. So far as they have any real basis, I believe that they will prove to be capable of interpretation along the three known lines of action of this substance—as a limited source of quickly available energy, as a depressant of the nerve-centres, and as a weak superficial vasodilator.

Dr. E. MELLANBY.

In the short time at my disposal I propose to deal as briefly as possible with the question as to the amount of energy that can be supplied to the body by alcohol, and the relation which alcohol bears to the other energy-producing foodstuffs.

Atwater and Benedict's calorimetry work has placed on an experimental basis the fact that a considerable amount of energy can be obtained from the combustion of alcohol in the body—25 per cent. has been mentioned by Dr. Dale. There is good reason, I think, to believe that this is a low estimate. One gramme of alcohol on oxidation gives out 7 Calories of heat; this compares very favourably with proteins and carbohydrates, each of which yields 4·1 Calories of available energy. Practically all the alcohol taken is absorbed and oxidized, so that, disregarding the toxic action of alcohol for the time being, we see that alcohol can supply a considerable quantity of energy to the body.

Fig. 1 shows the rate of entrance into, and of disappearance from, the blood of alcohol in the case of a dog. The dog, weighing 13·5 kg., had 50 c.c. of diluted absolute alcohol put straight into the stomach. This was completely oxidized at the end of twenty hours, that is to say, 2½ c.c. of alcohol were oxidized per hour, showing that 1·04 Calories of heat per kilogram body weight per hour was supplied by the alcohol. This represents about 40 per cent. of the total heat lost during the period. By other methods Völtz and Dietrich calculated that in resting dogs 42 per cent. of the total energy was supplied by alcohol during a ten hours' experiment and 35 per cent. in a fifteen hours' experiment.

Fig. 2 (curve A) shows the result of an experiment performed on a man, whose weight was 70 kg. He took whisky containing 171 c.c. of absolute alcohol. It can be calculated from the curve that he oxidized 10 c.c. of alcohol per hour. That is to say alcohol furnished about 54 Calories per hour, or roughly 1,300 Calories *per diem*. This would represent about 40 per cent. of his total energy loss.

Having stated this fact let us see how alcohol, from the point of view of energy, resembles or differs from other foodstuffs.

In fig. 3 can be seen the disappearance of alcohol from the blood of a dog which on different occasions received doses of alcohol, varying from 20 up to 55 c.c.—the latter producing profound intoxication. You will notice from these curves that the rate of disappearance of alcohol from the blood, which almost certainly represents its rate of oxidation, is very similar in each case. That is to say, the rate of oxidation of alcohol does not depend, within fairly wide limits, on the amount present in the blood. This result confirms that obtained by Higgins, using an entirely different method. The respiratory quotient for alcohol is 0·66—i.e., much lower even than that of fat, which is 0·71. Higgins showed that, under constant conditions, the depression in the respiratory quotient produced by alcohol was the same whether 30 or 45 c.c. of alcohol was drunk. This condition does not hold for carbohydrates and

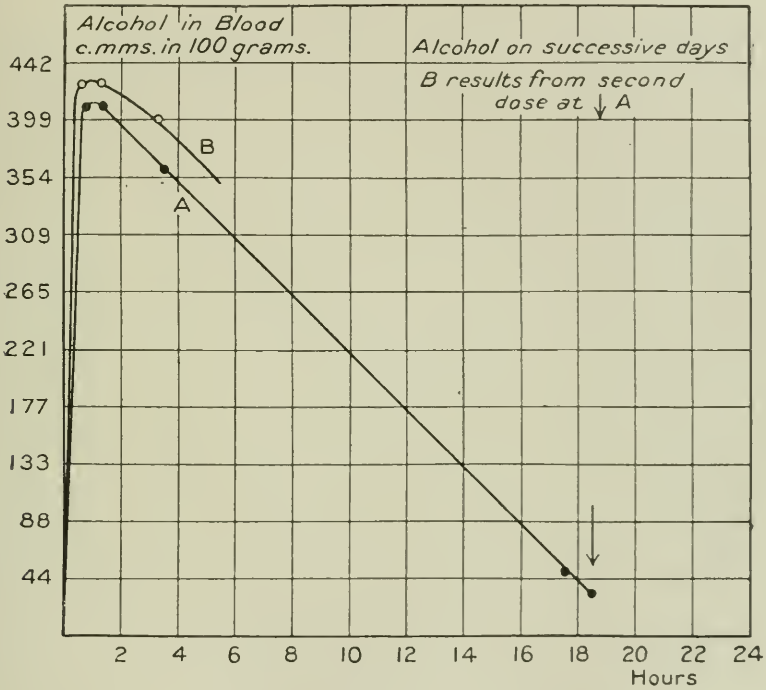

FIG. 1.

Alcohol in the blood of a dog after receiving 250 c.c. of a 20 per cent. solution (50 c.c. absolute alcohol). Note the rapid entrance into and slow exit from the blood. A similar dose was given eighteen and a half hours after the first, and a slight accumulative effect on the alcohol in the blood is evident. Intoxication was also more profound after the second dose.

fats. Gephadt and Du Bois have shown that the rise in the respiratory quotient produced by carbohydrate depends on the amounts eaten. Similarly Forssner and also Magnus-Levy have obtained results demonstrating that the rate of fat metabolism in certain conditions varies with the amount eaten. This, then, is the first great difference between alcohol and other foodstuffs—the rate of combustion is not as dependent on the amount consumed in the one case as in the other.

As mentioned above, the man of 70 kg. weight, when at rest, oxidized 10 c.c. of alcohol per hour, so that if the constant rate of oxidation holds even with very low concentrations, it is only necessary to give doses of the order of 10 c.c. per hour to such a man to get the maximum amount of energy in unit time. The smaller the dose the less the toxic action, so it is possible to get out of alcohol the maximum amount of energy in unit time and eliminate most of the acute effects due to its drug action. Most foodstuffs, besides supplying energy to the body,

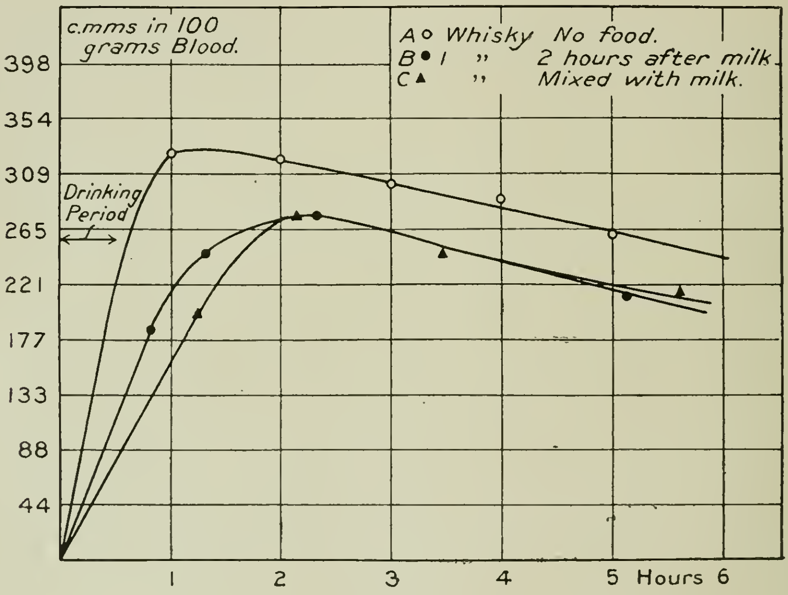

FIG. 2.

Experiment on a man who drank whisky containing 171 c.c. of absolute alcohol on three occasions: A, on an empty stomach; B, two hours after taking a pint of milk; and C, the whisky was diluted with one pint of milk. The rate of disappearance of alcohol from the blood indicates that he oxidized about 10 c.c. per hour. Note also the depressing effect of milk which was further evident by the reduction of symptoms of intoxication.

have the power of stimulating metabolism. This property is spoken of as their specific dynamic action. For instance, according to Rubner, the specific dynamic action of meat protein is 44; of fat, 16; of carbohydrate, 10. Alcohol has no specific dynamic action. If, for instance, a man is starving and alcohol be given him his production of heat will not increase. If he is given carbohydrates his production of heat will increase slightly. If given protein the metabolism will be considerably stimulated. We see, therefore, that, although alcohol supplies heat it does not make any more energy immediately available for the body. It simply conserves the stores of protein, fat and carbohydrate in the body. Here again we have a great difference between alcohol and other foodstuffs.

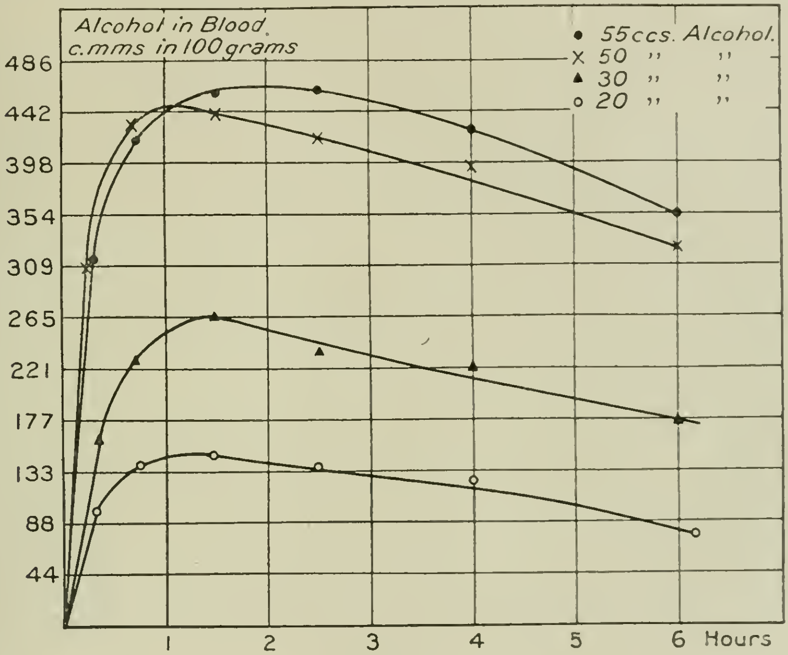

FIG. 3.

A dog was given, on different days, 55, 50, 30, and 20 c.c. of alcohol in a 20 per cent. solution. Note the similar rates of disappearance in spite of the difference of amount in the blood.

Not only does alcohol not increase the amount of energy immediately available to the tissues, but, in consequence of the heat loss following vaso-dilatation of the skin vessels, it can be seen that a fall in body temperature may follow. More especially will this be the case if the dose of alcohol is so great that it depresses the neuro-muscular mechanism and thereby directly diminishes the heat production. I think everybody is agreed that to give alcohol as a restorative in cases of exposure without prospects of other food or warmth is a most harmful procedure.

It has been stated by some that alcohol is oxidized at a greater rate when more carbohydrate is being metabolized than when fat is the predominant substance being used. This deduction is made from the fact that alcohol has a greater depressing effect on the respiratory quotient when the latter is high—i.e., when more carbohydrate is being metabolized. The following figures of Higgins demonstrate this effect.

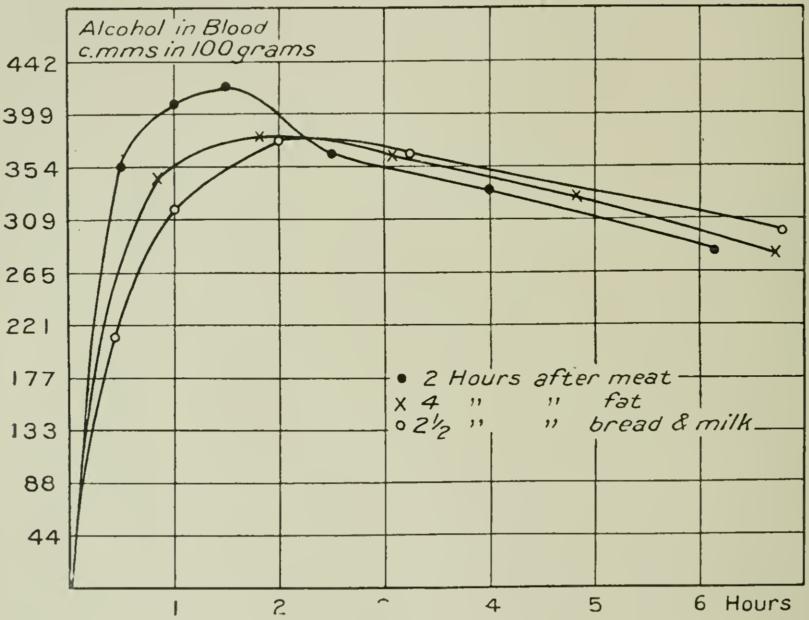

Fig. 4.

These experiments on a dog, who received the same amounts of alcohol (50 c.c. in 20 per cent. solution) on different occasions after eating meat, fat, and bread and milk respectively, show the constant rate of oxidation of the alcohol in spite of variations in the metabolism.

Table. I.—Higgins.

30 *c.c. Alcohol drunk.*

R. Q. before alcohol	R. Q. after alcohol	Drop due to alcohol	Percentage of O_2 consumed by alcohol (calculated)	Amount of alcohol burned per hour (calculated)
0·90 ...	0·805 ...	0·095 ...	40 per cent. ...	4·91 c.c.
0·85 ...	0·795 ...	0·055 ...	28 ,, ...	3·68 ,,
0·80 ...	0·775 ...	0·025 ...	20 ,, ...	2·75 ,,

R. Q. = respiratory quotients, CO_2 output, O_2 intake.

Notice also the calculation based on these figures of the amount of alcohol burnt. You will see it is calculated, that with a high respiratory quotient, 4·91 c.c. of alcohol were oxidized per hour, but when the respiratory quotient was 0·80 only 2·75 c.c. of alcohol were oxidized per hour. The calculation of these figures is probably dependent on an incorrect hypothesis, for, if you look at fig. 4, you will see that the rate at which alcohol leaves the blood is remarkably constant whatever the previous diet. The respiratory quotient of the dog must have varied greatly after its diets of meat, fat, and bread and milk respectively, and yet the rate of combustion of alcohol is apparently the same. The interpretation of the respiratory quotient results may be that, when the respiratory quotient is high alcohol spares more carbohydrate, and, when low, more fat.

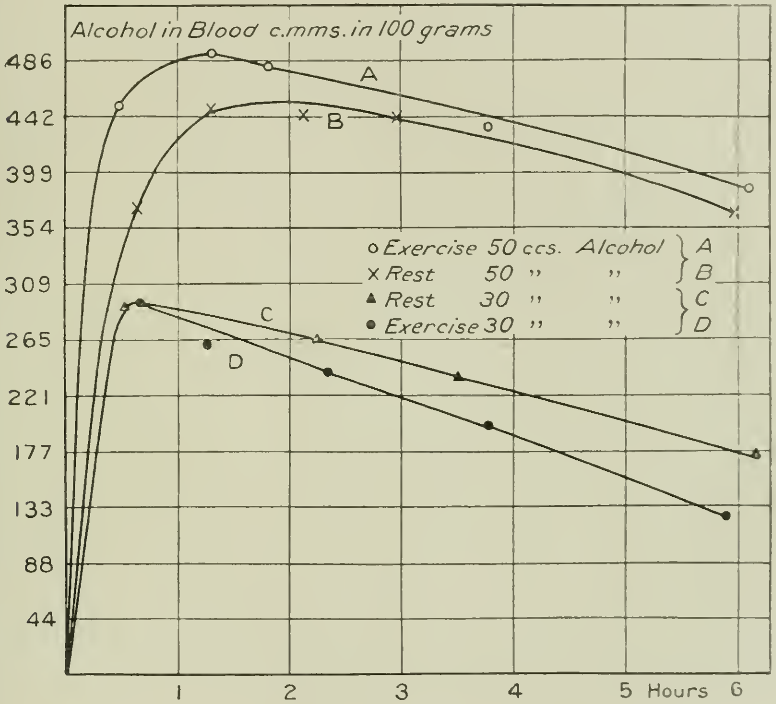

FIG. 5.

With high concentration of alcohol in the blood (A and B) running about had no influence on the rate of disappearance of alcohol from the body. With lower concentrations (C and D) exercise (D) increased the rate of disappearance. Experiment on dog.

Another point of difference between the oxidation of alcohol and of other energy-bearing foodstuffs is seen when the effect of exercise and mechanical work is considered. In the case of Atwater and Benedict's experiments it was clear that the rate of alcohol combustion increased at the same rate as other combustion processes and that alcohol supplied its share of energy both in the resting and active condition. The quantity of alcohol given in these experiments was comparatively small (72 c.c.) and spread over the whole day. In more recent work by Durig on mountain climbing, it seems that in hard muscular exercise alcohol does not play its full share even when only small quantities are used. In my own experiments on dogs it appears that in the case of normal running at a slow rate the effect of exercise depends on the amount of alcohol imbibed. When the amount is small the rate of combustion is considerably increased by exercise, but with higher concentrations in the blood, the effect of exercise becomes smaller, so that, finally, the rate of oxidation may be the same in the active and resting conditions. This fact is seen in figs. 5 and 6. There again we have evidence in favour of using small doses of alcohol given frequently rather than large doses.

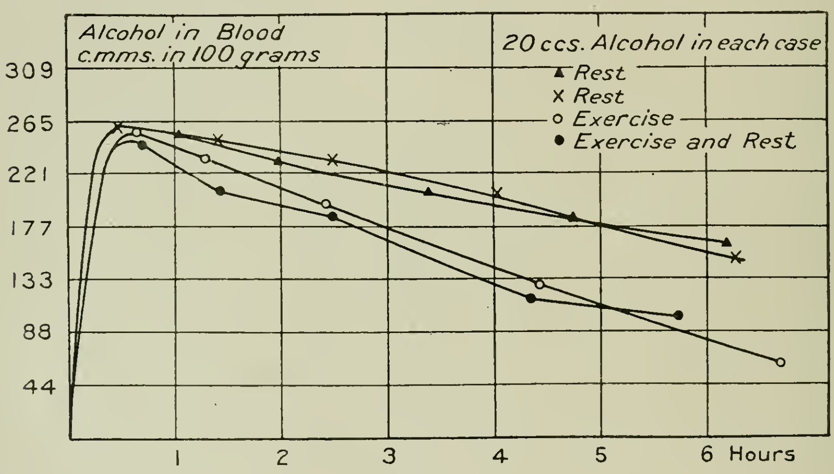

FIG. 6.

Smaller amounts of alcohol were given to a dog and lower concentrations in the blood were attained than in fig. 5. The effect of exercise in hastening oxidation is evident. In the last curve the periods of exercise and rest are seen by changes in rate of disappearance of alcohol.

To sum up, then, we see that considering alcohol as a source of energy, it has the following attributes :—

(1) It can supply as much as 40 per cent. of the heat lost.

(2) That it differs from other energy-bearing foodstuffs in the following ways: (*a*) The rate of oxidation is, within wide limits, independent of the amount taken. Increasing the amount does not increase the energy obtainable from it in unit time. (*b*) It has no specific dynamic action and therefore does not increase the immediate available energy. It only spares the other foodstuffs and has no stimulating effect on metabolism. (*c*) Its rate of oxidation is independent of the other material in the body being metabolized. (*d*) Exercise only increases its rate of combustion to an extent comparable with other foodstuffs when lower concentrations are present in the body. With high concentrations, in the case of dogs, exercise has no apparent effect in increasing the rate of combustion.

Finally, I should like to raise the question as to whether a substance which only supplies energy and with no other favourable metabolic characters is necessarily always of value to the body. For instance, it does not appear to me quite certain that alcohol, given in such a way as to eliminate or reduce to a minimum its drug effect, would extend the life of a man dying of starvation. Taken with other foods it will undoubtedly help to convert a negatively balanced diet into a positive one, thereby conserving the proteins, fats and carbohydrates of the body ; but, when taken as the only source of energy, which may well happen in some diseases, it is not self-evident that existence will be prolonged by it. A starving man or animal does not die because of the lack of energy-bearing material in his body. Whether, by increasing the amount of these substances as the result of giving alcohol, life would be prolonged is to me a matter of doubt.

Dr. W. H. WILLCOX, C.B., C.M.G.

In 1909 and 1910 Professor B. J. Collingwood and myself carried out a number of physiological and clinical investigations in order to determine the therapeutic value of alcohol vapour when mixed with oxygen. Our results were read before the meeting of the British Medical Association, London, July, 1910, and were published in the *British Medical Journal* of November 5, 1910. Since that time I have constantly used a mixture of alcohol vapour and oxygen as a therapeutic remedy and the previously recorded experience of its undoubted value has been confirmed by the results obtained with a large number of cases of different types of disease in which cardiac failure was a predominant symptom. I have no doubt as a result of the above experience that a mixture of alcohol vapour with oxygen is a very valuable cardiac stimulant in many cases of heart failure.

Physiological Experiments.—Professor Collingwood found that, when cats were submitted to poisonous doses of chloroform vapour, chloroform apnœa was caused. The blood-pressure fell very low and cardiac action became very feeble, often only weak auricular contractions being present. In this condition it was found that a mixture of oxygen and carbon dioxide would restore the respiration in many cases but it did not improve the cardiac failure. Oxygen alone did not raise the fallen blood-pressure. A mixture of air and alcohol vapour however gave better results than oxygen alone, and with a mixture of alcohol vapour and oxygen the most marked stimulating effect occurred, the blood-pressure in many cases rapidly rising to normal when the other remedies had failed. Schäfer and Scharlieb,[1] in their paper on the "Action of Chloroform on the Heart and Arteries," stated that alcohol vapour had a powerful antagonizing effect on the dangerous tendencies of chloroform. They advised a mixture of 10 per cent. of alcohol with chloroform for anæsthetic purposes.

The above physiological results led me to try a mixture of alcohol vapour and oxygen as a therapeutic remedy in cases of severe cardiac failure and remarkably good results were achieved. The method of administration consisted in bubbling the oxygen through absolute alcohol placed in a wash bottle, the oxygen being delivered to the patient by the exit tube fitted with a glass funnel which was held over the patient's mouth and nose. Special facepieces were not found practicable owing to the extreme state of illness of most of the patients on which the remedy was tried ; no doubt were these used different percentages of alcohol vapour might be employed. Experiments showed that the optimal percentage of alcohol vapour was about 4 per cent. by volume. Percentages from 3 per cent. to 5 per cent. were desirable. The percentage of about 4 per cent. can be obtained by the use of absolute alcohol, the inlet tube dipping below its surface, the temperature being about 13° C. Rectified spirit (90 per cent. alcohol) gives slightly lower percentage of alcohol vapour but still sufficient to exhibit a well-marked therapeutic effect. With brandy, whisky and other spirits the percentage of alcohol vapour is so small as to be of little value unless the temperature of the wash bottle is raised—e.g., to 25° to 30° C.

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[1] *Proc. Roy. Soc. Edin.*, 1904-5, p. 311.

Therapeutic Effects.—The mixture of alcohol vapour and oxygen should be given for ten minutes or longer until a definite improvement in the cardiac condition is produced; afterwards it may be given for five minutes, every half hour or hour as required. In cases with severe cardiac failure with low blood-pressure and rapid pulse the colour of the patient quickly improves, the cyanotic or lilac grey appearance being replaced by a more normal colour. The pulse is usually improved in volume and when rapid reduced in frequency by ten or twenty beats or more. The blood-pressure in many cases has been observed to show a rise of 30 mm. Hg.

It is important to note that oxygen alone does not give the above results of cardiac improvement. Improvement of colour occurs, but the marked improvement in the character of the pulse and in the blood-pressure has not been obtained except when alcohol vapour has been added to the oxygen. Recently the mixture was tried on a case of very severe ulcerative colitis with marked toxæmia and symptoms of inhibition, the pulse being extremely irregular and feeble and about 160 in rate. Within fifteen minutes the pulse became regular and 84 in rate, with fair wave and tension, when a mixture of alcohol vapour and oxygen had been given, and the cardiac condition was easily controlled by the judicious use of the mixture, the patient making a good recovery.

The mixture has been used in cases of pneumonia, pleurisy, morbus cordis, chloroform (anæsthetic) poisoning, typhoid, cholera, asthma with heart failure, angina pectoris, and collapses of various kinds—e.g., after surgical operations, narcotic poisoning, &c. Very good results have been obtained in the above conditions.

Mode of Action.—It is difficult to explain the precise manner in which the stimulating effect of the mixture of alcohol vapour and oxygen is brought about, and further work is necessary. Important factors are:—

(1) Method of administration: Here the inhalation of alcohol vapour undoubtedly causes a rapid and enhanced action of the drug. Amyl nitrite is a classical example of the great effect produced by a small quantity of a remedy given by inhalation. Very minute quantities of alcohol are administered by this method, only about 1 to 2 gr. per litre being present in the mixture, so that very small quantities of alcohol

are actually absorbed and this would be insufficient to produce any harmful effect on the liver, kidney and other organs.

(2) Physiological action.—This no doubt acts in two ways. (*a*) By direct cardiac stimulation. Professor Dixon[1] pointed out that moderate doses of alcohol increased the activity and beat of the heart and in cases of cardiac failure the rise of blood-pressure following the administration of alcohol was directly due to action on the heart. It seems probable that when alcohol is given by inhalation the small quantity absorbed passes directly to the heart and has a direct stimulating effect. (*b*) By weakening all inhibitory impulses, central and peripheral, no doubt the alcohol vapour when administered with oxygen has an important action.

Alcohol vapour mixed with oxygen does not appear to have any stimulating effect on the normal heart. Its use is recommended in cases showing signs of cardiac failure, and in these cases a marked beneficial result has usually been observed.

Dr. Robert Hutchison.

The therapeutic effects obtained from alcohol correspond more closely with its pharmacological action than is usual with drugs. As regards some of its effects there will be general agreement amongst clinicians: (1) Alcohol is undoubtedly a food and is of special value for this purpose in diabetes and in prolonged fevers. (2) It is a digestive stimulant and is often useful in cases of impaired appetite, gastric atony and in convalescence. The form in which it is administered has, however, a great influence on its digestive value. It is also useful as a carminative in cases of severe flatulence. (3) Its effect in dilating superficial blood-vessels is of use in counteracting the effects of chill as well as in rigors and in helping to reduce blood-pressure during an attack of angina. (4) Its narcotic action is helpful in some cases of insomnia, especially in old people, and in cases of delirium and restlessness in acute illness.

The chief point of controversy will be as to whether alcohol can be regarded clinically as a cardiac stimulant. In cases of acute heart failure, as in syncope and fainting, its action in reflexly stimulating the heart is certainly of great use, but in my opinion there is no clear clinical evidence that it acts as a heart stimulant in cases of acute infections. On the other hand by calming the nervous system, counteracting delirium and promoting sleep it may benefit the heart, in a case of prolonged illness, indirectly, and I therefore usually make use of it in such circumstances, though I am guided in prescribing it rather by the state of the patient's nervous system than by the pulse. As to whether alcohol increases a patient's resistance there is considerable difference of opinion, but my own view is that there is some reason to believe that in septic infections at least it has some power in this direction.

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[1] *Journ. Physiol.*, 1906-7, xxxv, p. 346.

Sir WILLIAM HALE WHITE.

The President has given us an admirable survey of the subject. Therapeutically, it seems to me, alcohol is of great value as a food, for example, in certain cases of anorexia and vomiting, such as are met with in phthisis and cancer. I remember, for instance, a man afflicted with phthisis who was vomiting constantly, and could not retain any food except alcohol for some days, and then, gradually, his power to take and retain ordinary articles of diet returned. I can only say it was the opinion of all of us who saw him that the giving of alcohol during those days saved his life for the time. Of course, alcohol must never be given in functional anorexia and vomiting, because they are just the patients who would form the alcohol habit.

I will not go into the question of its value in diabetes during the starvation period—though I am convinced it has a value there—because we are to hear more about that later.

In acute fever, as Dr. Hutchison has just said, when anorexia and vomiting or indigestion are present, so limiting the intake of ordinary food, alcohol is an admirable food, because it requires no digestion and is easily absorbed. And it has the inestimable advantage, in these conditions, of making the patient feel comfortable, and sending him to sleep. The disadvantages of it in such conditions are, that occasionally there is a complaint of headache, and some do not easily sleep when they have had alcohol. But in the majority of cases of acute fever, when the patient cannot take food well, I think you will find alcohol is beneficial, and has no deleterious effect.

Coming now to the President's second heading—convalescence, there again, alcohol often does good, much good. He who has just recovered from a severe illness, or from an attack of diarrhœa or influenza, often feels better for a glass of wine with his meals. His illness has left him weak, his digestion is weak too, and he is, as he says, "all on edge," which, translated into physiological language, I take to mean that all sorts of peripheral stimuli which would not worry him in health do worry him now. A glass of wine will annul these peripheral stimuli, and without doing appreciable harm. It will promote his digestion, he will feel better and more comfortable for it. I remember that Sir Samuel Wilks, when he was just upon 70 years of age, had a severe attack of influenza. When able to be about, he had many important public duties to perform and speeches to make. He told me he derived great benefit from carrying in his pocket a flask of sherry, so that he could drink a little when feeling extra weak and tired. I think that is valuable testimony in proof of what I am saying, for here we have the evidence of an abstemious and scientific man making an observation on himself.

Personally, I have never known the alcohol habit contracted from taking it during convalescence, such as I have just described; certainly Sir Samuel Wilks did not, because, we know, he lived to 86. Those who are suffering from chronic disease may, in the same way, take alcohol if the illness is such—as in the case of cancer—that it will be fatal before there is any risk of the habit being formed.

We pass now to the very difficult subject of the effect of alcohol on the circulation. That, I confess, is extremely hard to estimate, unless we take the simple case of strong concentrated alcohol given to a person to rouse him, when, I take it, the alcohol acts simply reflexly. When I entered the profession, large amounts of alcohol were given to cases of pneumonia; I have known a pint in twenty-four hours given by a physician, as a regular treatment. Nowadays most such cases receive no alcohol, yet I am unaware that the outlook on pneumonia is any better than it was. Epidemics of pneumonia vary so much in intensity, that I cannot express an opinion on the subject. I am not convinced, from watching these patients, that it did them grievous harm, and yet I am convinced that many such patients can do very well without it. Possibly in those who took it in large quantities it acted beneficially as a food. Sometimes it appeared to slow the pulse, sometimes to quicken it, not an altogether surprising result when, as Dr. Hutchison said, some of the slowing of the pulse may have been due to the damping-down of obnoxious peripheral stimuli, while the quickening may have been the direct effect of the alcohol.

The President, in his address, referred to the effect of alcohol on the respiratory centre, and in that respect there is a difficulty in deciding whether alcohol is beneficial or not; the cases vary so much among themselves. But if it was shown to have an effect on the respiratory centre, I do not think it would make much difference in the treatment of acute fevers, because most patients who are going to die from acute fever do not die from failure of respiration, but from heart failure. Even pneumonia patients are not often livid, and everyone will agree that the majority of fatal cases of pneumonia die of cardiac failure.

To sum up, I suggest that the chief therapeutical effect of alcohol in a beneficial sense is, that it is a pleasant depressant, peculiarly efficacious in inhibiting peripheral impulses, such as pain here, and discomfort there, that it diminishes those trivial worries which bother the sick. In larger doses it has the advantage of inducing sleep. It has the great advantage of being easily and rapidly absorbed. The small quantity usually given nowadays is not of very much value as a food, because a fluid ounce of brandy is only worth 100 Calories. I have never satisfied myself that, given in these moderate doses, it does any harm, nor that it benefits the respiratory and circulatory apparatus any more than it might be expected to do from making the patient more peaceful and quiet. But it appears to me that, in all these discussions, we are faced with two great difficulties—which it is almost impossible to surmount. One is, that it by no means follows that the effect of a drug in health is the same as its effect in disease, and its effect in disease is often very difficult to estimate. The second difficulty in such a discussion is, that no person, whether actually ill or convalescent, is ever given pure alcohol and water. We have to remember that perhaps there are other bodies present in wines and spirits, which have their actions; certainly there is a widespread belief that they have. For example, it is considered that brandy is the best form of alcohol for diarrhœa, that a glass of wine is, as Dr. Hutchison said, the best form of alcohol for one whose digestion is tired. Then, again, there is a firm belief—certainly among the laity, and, I believe, among many doctors as well—that old brandy is very much more efficacious for all therapeutic purposes than is a new raw spirit.

Dr. O. Leyton.

In the very able introduction with which Dr. Dale has favoured us he has indicated the lines upon which the discussion should run, and stated that I would give my experience of the use of alcohol in diabetes mellitus: but before doing this I should like to refer to one or two uses of alcohol in other diseases.

On some number of occasions I have seen alcohol administered in cases of which it has been thought that the heart muscle would cease beating and death ensue from that cause, and I must own that it has been extremely difficult to draw any conclusion as to the effect of treatment. In my opinion at the present day we have no method of determining whether the heart is likely to stop beating, and in several cases in which that termination has been feared the patients have died from respiratory failure. The evidence placed before us by Dr. Willcox makes me wish to repeat my observations.

Dr. Dale referred to alcohol as a diet in cases in which absorption is extremely bad, and I am of the opinion that in prolonged diseases such as typhoid fever alcohol may prove a most valuable adjunct to dextrose and other easily assimilable substances. The tendency to administer food in short fevers is ill-advised, if the patient does not crave for food, for most individuals can live upon their own tissues for many days without detriment.

I should like to refer to one other use of alcohol, namely, that of reducing the inhibitory tracts. I remember many years ago, before Freud's theories had reached these shores, there was a woman in the London Hospital who was able to cough but not to phonate, and therefore could only carry on conversation in a whisper. She was quite content, but remarkable to relate her husband was not, and with his and her consent she was given alcohol in order to see whether she might not become merry and talk, and once having talked the spell would have been broken. This method of treatment I believe has been used in some number of cases with some success. In the case under my observation the patient went to sleep and the excitement stage did not arise: but perhaps other observers may be able to tell us the result of this line of treatment.

I will now discuss the main point of alcohol as a food in diabetes mellitus. Before the Allen treatment was adopted at the London Hospital the weekly allowance of alcohol issued from the stores to my patients was usually *nil*, or upon exceptional occasions when students or residents were under my care, the quantity was negligible and usually less than that prescribed by any of my colleagues. I mention this in order to show that even if I was not a practical teetotaler I was a theoretical teetotaler. During the last four years the quantity of alcohol supplied from the stores at the London Hospital to my patients weekly has headed the list, but it is only diabetic patients that have had alcohol administered to them.

It has been my custom to order alcohol as a foodstuff to those patients who have been accustomed to take alcohol in the past, during the period of very meagre diet. There seem to be two advantages in this: first, a supply of a small amount of energy, and secondly (one to which Dr. Dale has referred), the patients are less irritable, pangs of hunger are more easily borne, and are less acute, and the patients seem able to stand treatment better. I still hold that the less alcohol the patient takes the better, but a large number of cases of diabetes mellitus have come under treatment in which the maximum diet they could take represents about 20 calories per kilogram per diem, or rather less than half that which was thought to be necessary in order for the patient to maintain life and energy. It is to these that in my opinion alcohol is essential.

I will not weary you by giving you a large number of instances but will refer to one patient who has been under observation nearly three years. A young woman who weighed about 5 st. 6 lb., and presented an appearance of great emaciation, was found to have a tolerance of only: carbohydrate 20 gr., protein 45 gr., and fat 60 gr. If any of these was increased, sugar appeared in the urine. Since the diet was so meagre it seemed to me hopeless to try to find one in which sugar in the blood was normal. This diet represents about 800 calories or less than 25 calories per kilogram. It seemed to me very unlikely that the patient would be able to enjoy her life on this diet, and therefore I added to it slowly and gradually 2 oz. of absolute alcohol daily. Absolute alcohol was chosen because the patient intensely disliked brandy, whisky, gin and other alcoholic beverages. The absolute alcohol was ordered to be well diluted and to be taken with meals, for it has long been realized that by giving it with meals it is absorbed more slowly and is less likely to produce any excitement or somnolence.

During the last year or so an improvement on dilution with water has been made by incorporating the alcohol into a jelly made with agar, and flavoured with citric acid, oil of lemon and saccharine. This jelly without the alcohol is—as far as I know—of no nutritive value, but tends to satisfy hunger. From it the alcohol diffuses out comparatively slowly and is slowly absorbed, so that in many ways it is an ideal method of administering alcohol.

The addition of 2 oz. of alcohol to the diet increased the value of the patient's diet by 350 calories, so that the energy of the diet was raised to 33 calories per kilogram, a value which experience has shown suffices to allow an individual to take exercise and gain weight slowly. The patient to whom I am referring walks six miles daily, and would enjoy her life if it were possible to prevent her making food her deity. This case confirms what we have just heard from Dr. Mellanby who told us that 30 per cent. of the energy in a diet can be supplied by alcohol.

I had come to the conclusion that alcohol was a necessity in some of the more advanced cases of diabetes mellitus and I have on record patients who have maintained their weight whilst taking alcohol, but who have been persuaded by their friends to stop taking it and have lost weight, the diet having been maintained constant in every other respect.

I think we have to realize the extreme instability of the metabolism in diabetes mellitus, and therefore how careful we must be in drawing conclusions. When a patient is on a small diet and sugar free, a slight disturbance with a vaccine of influenza, streptococci, and pneumococci may lead to the appearance of sugar in the urine, and any infection, or even some emotional disturbance, may produce the same effect. But bearing all these things in mind I had come to the conclusion expressed above, and am now full of interest to learn what is happening in the United States of America, for I hear that Dr. Joslin gives no alcohol to his diabetics. I have written to him asking what he does in cases similar to those whose limits of tolerance are so low as to make it impossible to maintain life without the addition of energy given in some form other than carbohydrate, protein or fat.

Professor W. E. Dixon.

I regret I am unable to remember the exact context of my remarks in the paper on the action of alcohol on the circulation to which Dr. Willcox has referred. I refer to two points in the discussion:—

(1) The meaning of the word "food." My opinion is that from the physician's point of view in the use of alcohol in the treatment of acute disease, the insistence that alcohol is a combustible substance and not a food is of little practical significance, since it can be substituted for foods in any dietary.

(2) The clinical significance of a cardiac stimulant is important and it is very difficult to draw conclusions as to the action of alcohol on the heart of man. I may refer to Schäfer's experiments on the effect of alcohol in chloroform administration, and I suggest that the action of alcohol on the heart after absorption is most easily explained by the fact that it is oxidized and used up by the heart (Hamill) and acts in much the same way as glucose.

Dr. C. F. Harford.

I desire to speak as a member of a group of medical practitioners who are interested in questions concerning alcohol, particularly with reference to alcoholism, but who have given considerable thought to the various problems connected with the use of alcohol whether as a medicine or otherwise.

We are greatly indebted to the President and to the Medical Research Committee for the new scientific aspect which has been given to the consideration of this subject, which is warmly welcomed by those of us who belong to the Society for the Study of Inebriety. To-day's discussion has shown that we are as yet only on the outskirts of knowledge as to the effects of alcohol on the human organism, the study of which in the past has been left chiefly to the energy of individuals who have pursued their researches often at great sacrifice to themselves, and their views have often been regarded as biased.

I have to-day commenced to read the "Life of Sir Victor Horsley," who but for his lamented death in the war would doubtless have been with us to-day, and, however much we may have been opposed to some of the views which he held, we cannot fail to honour the sincerity of his convictions and realize the loss which the profession has sustained by his death. Horsley was never tired of demonstrating from the hospital statistics of recent years the extraordinary change which has taken place in the attitude of the medical profession to the use of alcohol as a medicine, owing to a recognition of the scientific facts of the case.

I think the importance of to-day's discussion is that it will mark the beginning of a new scientific period in the study of alcohol and its place in treatment. If I may venture to do so I would point out that the views of some of the clinical workers who have spoken to-day seem to be based not so much upon the results of scientific experiments as upon the impressions gained in their own practice, which, though important in their way, are often illusory. In these days we need to go deeper, and the researches of Mellanby and others need to be carefully weighed in their relation to clinical practice. Clearly, I think we have got to this point, that alcohol is essentially a narcotic drug and should not be regarded as a stimulant, and this fact should be generally recognized and widely known.

In the prescription of alcohol there are certain practical points which need to be emphasized. In the use of drugs one of the most serious dangers is self-medication, and we feel this often in regard to antipyretics, hypnotics and so forth. There is a special danger in the case of alcohol which is more readily available than any other potent drug. It is a common superstition that alcohol is a panacea for most ailments that flesh is heir to, and this needs to be earnestly combated.

In the second place there is the great difficulty of accuracy of dosage which in other remedies is regarded as of prime importance. I remember that in my student days the late Sir Benjamin Ward Richardson pointed out the unsatisfactory method of prescribing alcohol in the form of whisky, brandy, wine or beer, containing as they do unknown quantities and varieties of other drugs, instead of being able to prescribe an exact dose of ethylic alcohol. This is a matter which has never received due consideration.

In conclusion, may I refer to a remark which the President has made from the chair as to the possibility that alcohol might be useful in taking in some measure the place of food in cases in which there is much fermentation in the alimentary tract? As one who is specially interested in tropical medicine, therefore, before this meeting I was looking up the literature of sprue, a disease in which there is an excessive amount of fermentation taking place in the intestinal tract. All the authorities say that alcohol is contra-indicated in sprue owing to the irritation which it produces, so that what seems likely to be beneficial, theoretically, may have practical drawbacks.

I lay no claim to speak as an expert on the therapeutical or pharmaceutical side of this subject, but speaking as one who realizes the vital importance of true scientific knowledge in this direction, I feel that we are in need of largely extended research, and I believe that this need has been proved by the discussion to-day.

Dr. Langdon Brown.

We are much indebted to the President for his extraordinarily well-balanced opening of the discussion. But I am afraid the discussion does leave us with the impression that all of us are apt to use alcohol without any very definite grounds for the faith that is in us. I have no doubt that tradition does count for a great deal in this matter, and, as the last speaker said, the change in our practice has been so enormous that it shows tradition must have weighed very heavily with us in the past, as in the present, in arriving at our conclusions. I would like to specify four definite conditions on which, I think, alcohol seems to have some real therapeutic action—they have all been referred to in some form.

First, in the treatment of diabetes by a great reduction in the diet, to be able to secure a certain amount of energy in the form of alcohol is a point of real value. And I was very interested in Dr. Leyton's remarks about that patient to whom the alcohol was given as ethyl alcohol, so that other considerations did not come in. It enabled the Calorie value of the food to be raised from a level which would only keep the patient going as an invalid, to one at which she could enjoy life and display some energy. One of the things we have learned from the Allen method is, that if you can balance metabolism at a reasonable level, then the outlook is better, both for enjoyment and duration of life, than if the intake has got to be so reduced that the energy derived from food is not sufficient to carry on. If we can raise the Calorie value—as, apparently, we can—by alcohol in this way, it is a point of considerable value.

My second point, I am aware, trends on the ground we have been asked not to deal with—namely, its value to convalescents: but as several speakers have dealt with it, I may mention it was firmly established by Bickel that alcohol was amongst the substances which definitely produced a flow of gastric juice: the substances were, I think, meat extracts, dilute alcohol, condiments, and aërated waters. It therefore has a value in conditions of achlorhydria and depressed metabolism.

The third point is one on which the President threw much light —namely, the removal of inhibitions. It has most definitely come out of this discussion, that we have no real evidence of alcohol acting otherwise than as a depressant. Just as it removes our higher inhibitions, so it can remove peripheral inhibition too. I think much of the effect which has been noted in disease has been, as you suggest, due to that. If we can make our patients more comfortable and enable them to go through their temporary disablement in that way, it is a distinct gain.

The fourth point is this: I have a general impression that Dr. Willcox is right as to the benefit which can sometimes be secured to patients by the administration of oxygen over brandy: I have done it in a number of cases, and I have considered it helped. But there again our impressions are apt to be but vague: one never knows what would have happened if a particular drug had not been given. One does not know what are the absolutely fatal signs, and it is seldom that one sees signs which are regarded as fatal, and which can be removed by the administration of any drug. After all, the great difficulty in all clinical work is the absence of controls.

The President.

There has been no mention in this discussion, so far, of the effects of alcohol on children. Perhaps Dr. Esther Harding will say a few words on that aspect of its use.

Dr. M. Esther Harding.

(1) Children are valuable for estimating the effects of alcohol because the æsthetic effects may be entirely discounted. The uses of the drug fall under the same heads as in adults:—

(*a*) In respiratory embarrassment, especially in the rapid, shallow, inefficient breathing of broncho-pneumonia, alcohol quiets the respiration, and so makes it more efficient. It is true that the indication for the use of alcohol is commencing failure of the right heart, but the effect of the drug is not to flog the already overburdened heart, but to relieve it of some of its intolerable burden, by slowing the respiration, and improving the oxygenation of the blood.

(*b*) As a sedative. Alcohol is, I suppose, the most valuable sedative and hypnotic drug we possess for infants and young children. Hot toddy as a sleeping draught is good for infants and for the aged.

(*c*) As a food in cases where no ordinary diet can be taken: (i) In milk intolerance of marasmic infants, alcohol will sometimes tide a patient over a few days till milk or whey tolerance can be re-established. (ii) In the persistent late vomiting which kills so many patients after severe diphtheria I have kept a child alive on more than one occasion under such circumstances on saline, brandy and sugar by the stomach for three weeks.

A word of warning on the danger of the persistent use of alcohol in young children is hardly needed, but it is wise to remember that small doses of spirit given to an infant over a period of weeks, a large proportion perhaps of its whole life, may easily result in damage to its liver.

The PRESIDENT (in reply).

I think nothing remains for me except to thank those who have taken part in what I believe has been an interesting, and will probably prove to have been a fruitful discussion.

Obviously, there are certain points outstanding on which agreement is not yet possible. Perhaps the most remarkable thing about the debate is the extent to which agreement has been in evidence; but on the question as to whether alcohol is, or is not, a direct stimulant of the heart, for example, it is clear that we are not yet at one. The conviction I expressed in my opening remarks is not, I confess, at all shaken by Dr. Willcox's evidence, or by that which Dr. Dixon mentioned. It is true that Dr. Collingwood, also Sir Edward Sharpey Schäfer and Dr. Scharlieb, demonstrated some inhibiting effect of alcohol on the production of heart failure by chloroform; but I think it would be very rash pharmacology to deduce from that the general conclusion that alcohol is likely to have a stimulating effect on a heart weakened by any cause. My own feeling still is that which I ventured to put before you.

With regard to Dr. Harford's emphasis of the fact that alcohol is purely a narcotic, I think there is some danger of our losing sight of the fact that, from the point of view of therapeutics—we are not talking of use in ordinary, healthy life—there is no inherent desirability in a stimulant action, and no inherent drawback in narcotic action. The latter may be what we want. It appears that the clinician has found, by practical experience, certain conditions in which alcohol definitely does good. From the practical point of view, I think it is a matter of comparatively small importance, with regard to those conditions in which the evidence is already available, whether the good is done by narcotic action or by stimulant action. It is only when a wrong conception of the proved actions leads us to wrong deductions of other actions, and to wrong applications in new directions, that the matter becomes one of practical importance.

Section of Therapeutics and Pharmacology.

President—Dr. H. H. Dale, C.B.E., M.D., F.R.S.

DISCUSSION ON THE THERAPEUTIC USES OF OXYGEN.[1]

Mr. J. Barcroft, C.B.E., F.R.S.

The subject of oxygen therapy is of course no new one; for that reason it is likely to make an excellent subject for discussion. If such a subject has survived the course of years at all it is sure to have opened up a number of different points of view. A subject of this kind may pass through three stages: it dies early or there arises around it in different directions considerable diversities of opinion, or, finally, it reaches an ultimate zone where there is unanimity, and little more can be said about it. I need not remind you that the final state is an extremely uncommon one. It is certainly not one which is within sight so far as oxygen therapy is concerned. There can be no doubt that oxygen as the therapeutic agent is somewhere in the middle zone.

I hold a very definite view as to the best method of treating a subject when it is in a condition of high controversy, namely, a method that produces facts; and here I suppose the Royal Society of Medicine to be with me, for I can conceive of no other reason why I should be asked to open this discussion. My work on oxygen therapy has been restricted to an attempt to ameliorate the lot of men gassed either in battle or in the manufacture of munitions of war. This field is so small, so unlikely to be opened up again in our time, and so far removed from the broad lands of general practice, that I cannot suppose that you want to hear much of my opinions on—say, what might have been the fate of my gassed men if they had not had oxygen. In the conduct of that work, however, I did try—Major Hunt tried, Miss Dufton (now Mrs. Wilson) tried—we all tried our best to get at the facts, to formulate them in such a way that they should be simple, intelligible and capable of repetition, and took care that in their interpretation neither our opinions nor those of the patients should play a part. And I imagine that it is because you are in sympathy with this ideal, because you wish to get in the field of oxygen therapy; though some plot, even if it be a small one, which has been cultivated by the method of ascertained fact rather than of formulated opinion or unproven theory, that you have asked me to open your discussion.

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[1] At a meeting of the Society, held January 20, 1920.

Method.

The method of oxygen administration as carried out in the Cambridge Physiological Laboratory by us was of the simplest possible character. A glass room of about 1,000 cubic ft. in capacity formed a tiny ward. In this were three beds. The ward was gas tight and fitted with air locks large enough to serve for the exit and entrance of the patients. Small air locks were provided for the admission of food, &c. The composition of the atmosphere was kept at about 40 to 50 per cent. oxygen and 50 to 60 per cent. nitrogen. The carbonic acid and aqueous vapour were removed by the passage of the gas through an external circuit which contained scrubbers, soda-lime and calcium chloride.

Our motive for using this type of administration was merely that it was the simplest. The whole treatment being experimental we desired that the patient should be subjected to only one altered condition of life—namely, the composition of the atmosphere which he breathed. For this reason we avoided all types of apparatus for the administration of oxygen through a mask or otherwise, considering that a second variable was introduced by hampering the patient with apparatus.

No attempt, therefore, was made by us to study the best method of administration; the matter is still one which is entirely an open experiment. In this connexion I will only say that the chambers subsequently put up at the North Staffordshire Infirmary at Stoke-on-Trent seemed to be better in every way than ours at Cambridge. No doubt Dr. Shufflebotham will give some description of them. For many purposes for which oxygen may be wanted a chamber is clearly out of the question. Several of those who will speak—Colonel Cummins, Dr. Douglas, Dr. Haldane, and Dr. Peters—have used this apparatus with excellent results, and will give you the benefit of their experience.

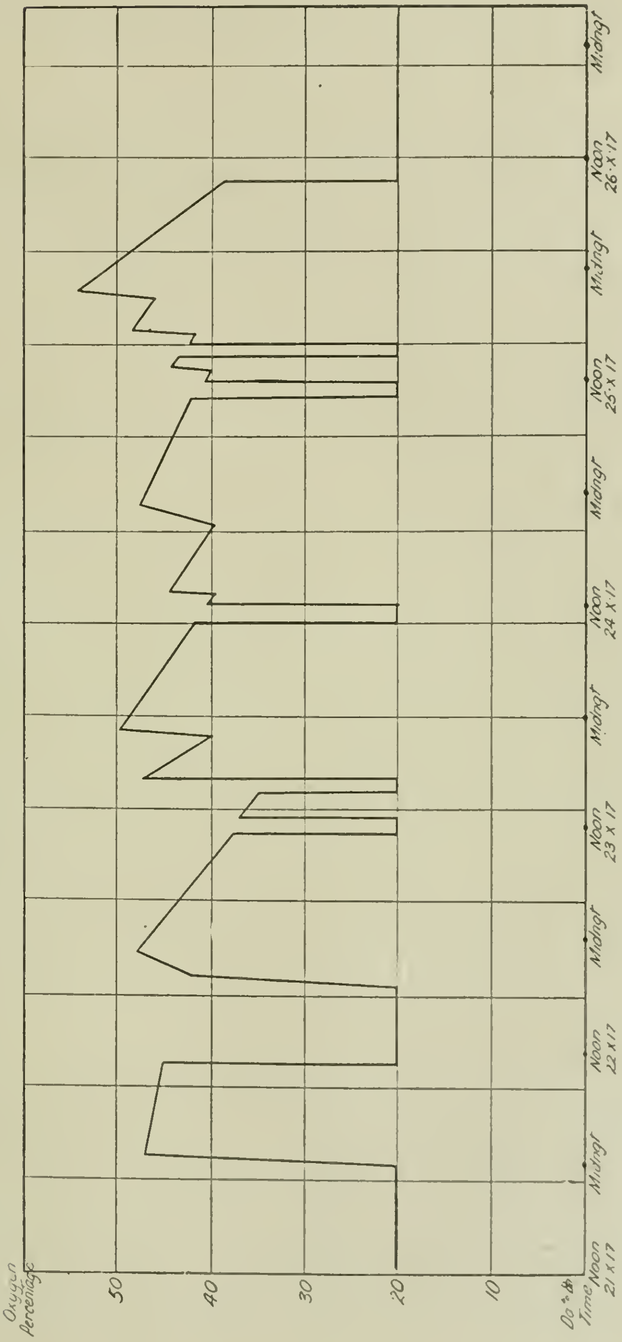

Oxygen Percentage
50
40
30
20
10
Do % 0
Time
Noon 21 X 17
Midngt
Noon 22 X 17
Midngt
Noon 23 X 17
Midngt
Noon 24 X 17
Midngt
Noon 25 X 17
Midngt
Noon 26 X 17
Midngt

FIG. 1.

Our own routine with patients was to put them in the chamber from about 5 p.m. till 10 a.m. for five consecutive days. Fig. 1 shows the composition of the atmosphere to which the patient was exposed, and is a fair sample of most of the rest.

First, let me say that the cases with which I had to do were all chronic cases, treated in this country; the treatment was in part worked out on the basis of experiments on animals and in part from reports which reached me from France. Such benefit as may accrue from our work at Cambridge you owe, therefore, very largely to two persons, both of them here, I am glad to see, this afternoon, and both of whom will address you on portions of the subject—Colonel Cummins and Lieutenant-Colonel Douglas. They not only wove in France the strings which were put into our hands, but made it their business not to rest till they had ascertained that our fingers really gripped the cord.

A chronic case of poisoning with chlorine, phosgene or chlor-picrin usually complains of one or both of the following: Nocturnal attacks of dyspnœa, with which may be associated sleeplessness and physical distress out of proportion to the exertion which causes them. What objective measurement may be made for the purpose of defining the man's condition? How can his improvement, if any, be expressed in units? Clearly of the two things of which the man complains, the exaggerated response of the system to exercise offers a better mark for objective expression than does the nocturnal dyspnœa.

The problem of how an exaggerated response to exertion may be stated in units is one which has concerned many workers within the last few years. The response either of the circulatory or the respiratory system may be taken as the guide. Each has its advantages and its drawbacks. The pulse is easy to count, its variations in rate are susceptible of simple and accurate statement, but its significance in relation to the strain put upon the heart or the volume of blood propelled round the body is obscure, whilst the mechanism of its regulation was at the time of our experiments difficult to interpret. The respiratory system is in some respects more hopeful; not only can the frequency of the respiratory rhythm be measured, but the total ventilation, the composition of the air expired and of that retained in the lung and the depth of respiration can all be recorded. On the other hand the respiration is, to a much greater extent than the pulse, under the influence of the will, and therefore the records when obtained are to that extent of more doubtful value.

In the first fourteen cases which we studied we took the response of the pulse to effort as our criterion, in the last twelve the respiratory response. Of the first fourteen cases I will now speak:—

PULSE AS AN INDEX OF EFFORT SYNDROME.

The procedure as far as the pulse was concerned was as follows: After a general examination of the patient had taken place he was made to rest for fifteen minutes. His pulse was then counted. He was then given an exercise to perform, the severity of which was judged appropriate to his condition. His pulse-rate was counted again when the exercise was completed, and the time observed from the end of the exercise till the pulse had dropped to within six beats of its resting rate. In nearly every case the pulse of the gassed man took an unduly long time to return to its normal rate. A concrete example may illustrate just what took place. The results of an actual test are given in fig. 2. Here the pulse-rate of two men are compared—the

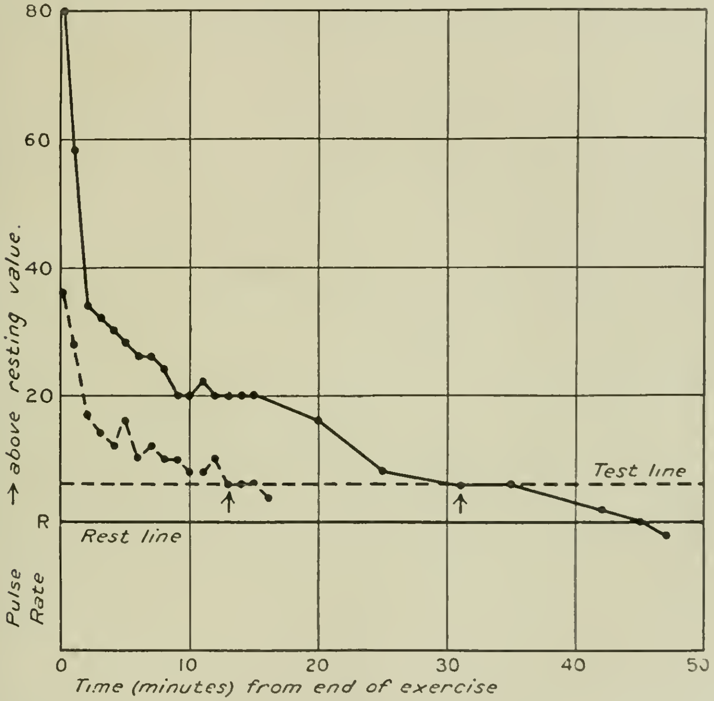


FIG. 2.

rates are plotted vertically, taking as a zero the rate of the pulse at rest; the figures denote number of beats in excess of this value. The abscissa of the graph is time in minutes. The upper line represents the pulse-rate of the gassed man, the lower line that of a normal person who was given the same exercise, in this case a stair-climbing test. The horizontal line labelled " test line " is drawn six beats above the resting value. The question then is, What interval of time elapses between the end of the exercise and the intersection of the test line by the pulse curve? In the case of the gassed man this was thirty-one minutes, in that of a normal individual it was thirteen minutes. Our exercises were for the most part much less severe than this.

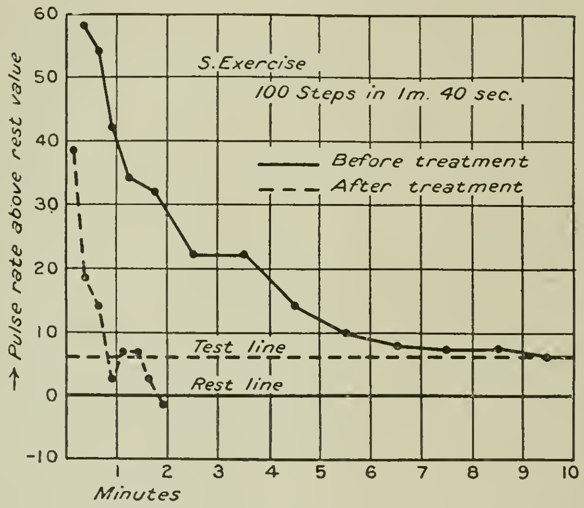


Fig. 3.

Results of Pulse Tests before and after Treatment.

After the treatment this man was given the same test as before it.

(*a*) *The Immediate Effect.*—The case of Sergeant S. was typical of the best results which we obtained. This man was gassed in the original gas attack at Ypres on April 24, 1915, presumably with chlorine. He was treated by us on October 31, 1917, that is two years and a half after being gassed. The effect of the treatment on his pulse is shown in fig. 3. His pulse, which before treatment took nine to ten minutes to fall to the test line after exercise, fell after treatment to the line in the fourth quarter minute.

(*b*) *The Fourteen Cases as a whole.*—Columns 2 and 3 of the following table give the recovery times of the pulse after exercises specified in the last column. There was not one of the cases that did not receive more or less benefit.

TABLE I.—TIME (MINUTES) WHICH ELAPSES BETWEEN END OF EXERCISE AND RETURN OF PULSE TO WITHIN SIX BEATS OF NORMAL.

	Before first treatment	After first treatment	Before second treatment	After second treatment	2 to 2½ months later	Normal man doing same exercise as patient	Control exercise
T. D.	32	6	>21	1½	—	×	—
T. J.	1½	1	½	1	—	½	Four flights in 1 min. 20 sec.
A. A.	15	5	—	—	—	1	Ten flights in 2 min. 45 sec.
Spr. C. ...	5	1	1	½	½	½	Three flights in 1 min. 5 sec.
Pte. D. ...	2	1	1	1	1	1	Five flights in 1 min. 5 sec.
Pte. A. ...	1	½	—	—	½	½	Two flights in 41 sec.
Sgt. S. ...	10	1	—	—	2	1	Six flights in 1 min. 40 sec.
Pte. O'B. ...	5	1	—	—	½	½	Three flights in 1 min. 15 sec.
Gnr. A. ...	>21	1½	—	—	1½	1	Four flights in 1 min. 20 sec.
Cpl. H. H. ...	50	40	—	—	—	1	Ten flights in 3 min. 40 sec.
Lieut. P. ...	1	½	1	1	—	½	Three flights in 1 min.
Pte. F. ...	1	½	1	1	—	½	Four flights in 1 min. 2 sec.
Lieut. R. ...	2	1½	—	—	—	½	5,650 ft. lb. in 1
Pte. H. ...	Did not fall in 6 min.	1	—	—	—	½	Six in 2 min.

Permanency of Effect on Pulse.—You will naturally ask, How far is this improvement of the pulse in effort syndrome lasting? On that subject we can give some data, though in the nature of the case our observations are less complete than we would like. Dr. Sowry, however, has had the opportunity of following his cases much longer than we have and to him I look for a statement on this point. We were able to examine several of them about two and a half months after they were treated, with the results shown for Sergeant S. in fig. 4. The dotted line shows his pulse recovery. It is not quite so satisfactory as immediately after treatment, but as compared with his original condition there is a great improvement. Of the five patients so examined three were normal whilst two others were nearly so.

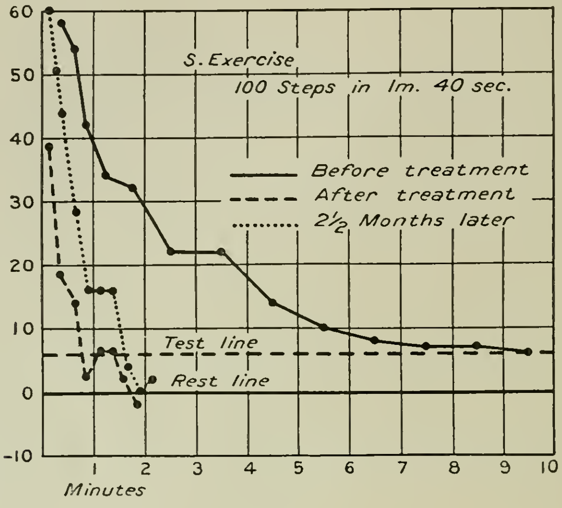


FIG. 4.

TABLE II.

		Before first treatment	After first treatment	Before second treatment	After second treatment	2 to 2½ months later
T. D. ...	Red cells	6·1 millions	5·2 millions	5·9 millions	5·2 millions	—
	Hæmoglobin	102 per cent.	96 per cent.	103 per cent.	100 per cent.	—
T. J. ...	Red cells	7·9 millions	6·2 millions	7·0 millions	5·8 millions	—
	Hæmoglobin	111 per cent.	106 per cent.	112 per cent.	109 per cent.	—
A. A. ...	Red cells	6·5 millions	5·1 millions	—	—	5·4 millions
	Hæmoglobin	94 per cent.	88 per cent.	—	—	90 per cent.
Spr. C. ...	Red cells	6·5 millions	5·0 millions	5·7 millions	5·2 millions	6·2 millions
	Hæmoglobin	103 per cent.	94 per cent.	96 per cent.	93 per cent.	98 per cent.
Pte. D. ...	Red cells	6·3 millions	5·4 millions	6·1 millions	5·2 millions	5·2 millions
	Hæmoglobin	105 per cent.	97 per cent.	104 per cent.	100 per cent.	102 per cent.
Pte. A. ...	Red cells	6·0 millions	5·1 millions	—	—	5·7 millions
	Hæmoglobin	93 per cent.	92 per cent.	—	—	—
Sgt. S. ...	Red cells	5·9 millions	4·9 millions	—	—	5·2 millions
	Hæmoglobin	104 per cent.	97 per cent.	—	—	99 per cent.
Pte. O'B. ...	Red cells	4·8 millions	4·9 millions	—	—	5·3 millions
	Hæmoglobin	88 per cent.	87 per cent.	—	—	88 per cent.
Gnr. A. ...	Red cells	6·3 millions	5·3 millions	—	—	6·2 millions
	Hæmoglobin	101 per cent.	92 per cent.	—	—	98 per cent.
Cpl. H. H.	Red cells	5·6 millions	5·1 millions	—	—	5·4 millions
	Hæmoglobin	111 per cent.	102 per cent.	—	—	103 per cent.
Lieut. P. ...	Red cells	7·5 millions	4·9 millions	5·3 millions	5·2 millions	—
	Hæmoglobin	104 per cent.	97 per cent.	98 per cent.	100 per cent.	—
Pte. F. .	Red cells	6·5 millions	4·6 millions	4·8 millions	5·4 millions	—
	Hæmoglobin	96 per cent.	98 per cent.	94 per cent.	102 per cent.	—
Lieut. R. ...	Red cells	4·9 millions	4·6 millions	—	—	—
	Hæmoglobin	96 per cent.	98 per cent.	—	—	—

THE RED BLOOD CORPUSCLE COUNT.

A sign which was frequent, but by no means invariably present in cases gassed with phosgene or chlorine was a high red blood corpuscle count. It was largely the occurrence of this sign which suggested the oxygen treatment to us. Occurring as it does in persons exposed to atmospheres deficient in oxygen we argued rightly or wrongly that the normal atmosphere to these patients presented an oxygen deficiency and that therefore an increase in the oxygen inspired might reduce the red blood count to a normal value. Whether our argument was sound or unsound the event proved wholly satisfactory, as is shown by Table II (*see* p. 66).

For not only was it true that the high blood counts were reduced during the treatment to a normal value, but in contradistinction to those the blood counts which were normal before the treatment remained normal throughout. There is of course room for great error in corpuscle counting, yet I feel sure that no patient shows a succession of counts like the following :

			Millions	
October	11	p.m.	7·87	In chamber
,,	12	a.m.	7·88	
		p.m.	7·79	
,,	13	a.m.	7·45	
		p.m.	6·95	
,,	14	a.m.	6·95	
		p.m.	6·65	
,,	15	a.m.	7·10	
		p.m.	6·98	
,,	16	a.m.	6·80	
		p.m.	6·71	
,,	17	a.m.	—	
		p.m.	6·42	

without the fall which they indicate being a *bona fide* phenomenon.

Two and a half months after the treatment counts were made in several cases ; in none was the count very high, the highest being 6·2 millions. It is fair, however, to say that none of these cases had very high blood counts to start with.

The third sign to which we paid a good deal of attention was nocturnal dyspnœa. The treatment was satisfactory to this extent that out of six cases in which these attacks were a marked feature only one showed no improvement. In one case they were much improved, though not completely abolished, whilst in the other four cases the attacks were completely and permanently abolished. No man had an attack during the treatment.

Much more stress would be laid on the abolition of the dyspnœic attacks but for the fact that they are signs of a much more subjective type than persistent quickening of the pulse on exercise or high red blood counts. A word may be said in conclusion as to whether the results which I have put before you can be attributed to some subjective cause. Three remarks may be made on this subject.

(1) As regards the red blood corpuscle counts, it is extremely unlikely that they should drop from say 7·5 million to 5 million as the result of suggestion. Moreover in gassed rabbits precisely the same phenomenon occurs.

Date and condition	Atmosphere	Red count
April 23—Before gas	Air	4·9 millions
Gassed	—	—
May 9	Air	6·80 millions
,, 9—Put into	30 per cent. O_2 and 70 per cent. N.	
,, 10	,,	5·6 ,,
,, 11	,,	4·8 ,,
,, 12	—	4·8 ,,
,, 13	Air	5·8 ,,
,, 14	,,	5·8 ,,
,, 15	,,	5·8 ,,

(2) As regards the pulse twelve cases of D.A.H. not attributable to gas poisoning were given oxygen treatment. These taken as a whole showed no clear benefit from the treatment. There seems no reason why their pulses should not be as susceptible to suggestion as the gassed cases.

(3) As regards the nocturnal attacks, in the case of one man a pantomime was enacted of blowing in oxygen in such a way as to make him suppose that he was getting the treatment, whilst in reality he was not getting it, for the oxygen was expelled from his chamber, unknown to him, as rapidly as it entered. He complained that the treatment did him no good and that his nights were not improved. On the fourth night the treatment really commenced and the man at once improved.

Dr. G. H. Hunt.

Mr. Barcroft has already told you that treatment with oxygen diminished the dyspnœa which "gassed" patients experience on exertion. Although we were quite satisfied from ordinary observation that this was really the case, we were anxious to find some more exact method of estimating the decrease in the breathlessness, and, if possible, of measuring the improvement in this respect. We were faced with the difficulty that there was no numerical measure of dyspnœa which satisfied our requirements. It was clear that any standard must fulfil three conditions: First, it must be fairly constant for the same man provided his physical condition did not alter appreciably; secondly, it must give approximately the same result for a number of men in the same condition of physical fitness; thirdly, it must distinguish healthy men from men suffering from any pathological condition causing dyspnœa.

Our method of investigation was as follows: The subject breathed through a meter while at rest, and his total ventilation and a respiratory rate per minute were noted; he then did a given amount of work on Martin's bicycle ergometer, and his respiratory rate and total ventilation were measured during exercise and for each successive minute after exercise. A few experiments showed that the total ventilation and respiratory rate in all these periods varied considerably in the same man, and were widely different for a number of healthy men. None of these measurements then satisfied our conditions. It is well-known that the main difference in the breathing of healthy and breathless men lies in the time taken for each to recover from the exercise, and return to the condition found at rest; after going up a flight of stairs the breathless man remains dyspnœic for a much longer time than the healthy man. We therefore tried to estimate the time taken after exercise for the respiration to return to its pre-exercise condition, but for many reasons this was found impracticable. Instead of this we compared the total ventilation after exercise with that at rest, and estimated the relation between the total ventilation during the second, third, fourth and fifth minutes after exercise with that during four minutes when the subject was at rest. By dividing the former by the latter we obtained a figure which we called the "dyspnœa ratio." Fig. 1 illustrates how this ratio is obtained. The figures opposite the horizontal lines give the number of litres breathed per minute; those opposite the vertical lines show the minute in which the observations were taken; thus A-B gives the total ventilation during four successive minutes with the subject at rest; B-C the ventilation during an exercise lasting one and a half minutes; C-D the ventilation during the first minute after exercise; D-E the ventilation during the second, third, fourth and fifth minutes after exercise; the dyspnœa ratio is given by dividing the shaded area between D and E by that between A and B. In this case the ratio would be $\frac{50}{20} = 2{\cdot}5$. We found that the dyspnœa ratio fulfilled our requirements satisfactorily, for it was reasonably constant for the same individual doing the same amount of work in a given time; it was approximately the same for eight healthy men who were in good "training" and for four who were in bad "training," and clearly distinguished healthy men from those suffering from any disease known to cause dyspnœa.

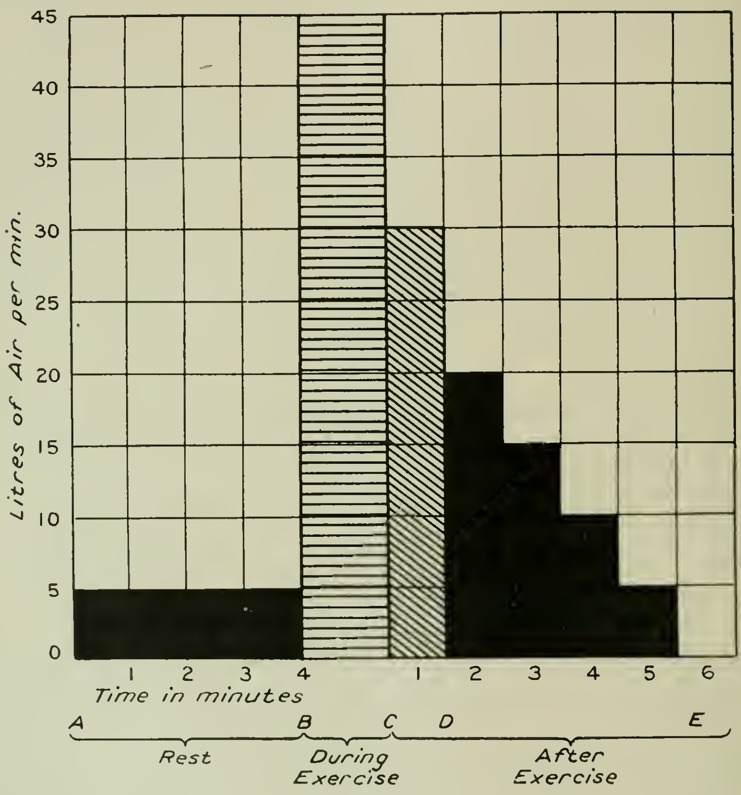
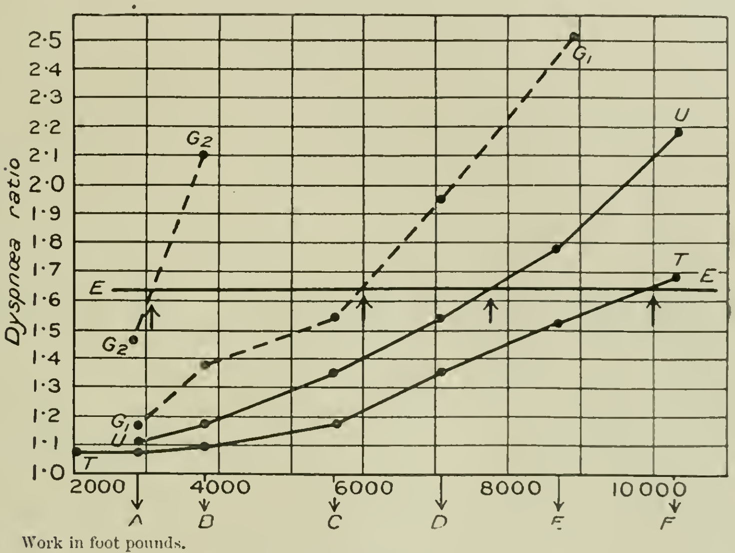


FIG. 1.

Fig. 2 shows the dyspnœa ratios from a number of different subjects over a series of six exercises of increasing severity, each lasting one and a half minutes. The ordinates give the dyspnœa ratios, the abscissæ the amount of work in foot-pounds done in each exercise. The letters A, B, &c., indicate the amount of work done in each exercise. The line T-T shows the average ratio for eight trained men, the line U-U the average ratio for four untrained men, the lines G1-G1 and G2-G2 the ratios for two men suffering from gas poisoning. If the ratios for the trained men are considered, it will be seen that over the first two exercises the value is nearly 1; this is an expression of the fact that within a minute after the cessation of the exercise the ventilation had almost returned to the condition found at rest. As the exercise increases in severity the ratio gradually rises. The ratios for the untrained man approximate to those of the trained man over the first three exercises; over the fourth exercise there is an obvious difference; over the fifth exercise the difference is more marked, and over the sixth exercise it is considerable. We should expect this result if the dyspnœa ratio is an accurate measure of breathlessness, for to ordinary observation there is no difference in the respiration of trained and untrained men when they are doing an easy exercise, but it becomes obvious when they are taking hard exercise; if, for example, a trained and untrained man walk a hundred yards together, it will be impossible to say whose respiration is the more affected; if they run a mile together, it will be very obvious that one is more breathless than the other. In both the "gassed" men it is clear that the ratios are higher than for the trained and untrained men.



Exercise A = 2900 foot pounds.
,, B = 3800 ,,
,, C = 5600 ,,
,, D = 7020 ,,
,, E = 8740 ,,
,, F = 10380 ,,

FIG. 2.

Fig. 2 also affords us a measure of a man's efficiency as judged by his respiration. If we take the curve of the trained men (T-T) we can calculate their dyspnœa ratio for a given amount of work; thus if they do 8,000 foot-pounds the dyspnœa ratio is found by drawing an ordinate from the 8,000 and finding where it meets the curve; in this case it is 1·44. Conversely we can calculate the amount of work required to produce any given dyspnœa ratio by drawing an ordinate from the curve on to the abscissa; thus to produce a dyspnœa ratio of 1·64 a trained man must do 10,000 foot-pounds of work. Now a dyspnœa ratio of 1·64 is given by the untrained man doing 7,750 foot-pounds; that is to say, with the same degree of dyspnœa the untrained man only does 77·5 per cent. of the work done by the trained man; in other words, his efficiency as judged by his respiration is only 77·5 per cent. of the trained man. The efficiency of the two "gassed" men (G1 and G2) is only 30 per cent., and 58 per cent. that of the trained men. We thus arrive at a figure E, which is a measurement in foot-pounds of the efficiency of a man, and is defined as the amount of work he can do with the degree of dyspnœa which is experienced by a trained man doing 10,000 foot-pounds of work in one and a half minutes.

Having by this way obtained a standard by which we could gauge a man's efficiency in terms of work, we were able to measure the degree of improvement produced in "gassed" patients by oxygen treatment. Two examples of this are shown in fig. 3; the ordinates give the dyspnœa ratios; the letters A-F indicate the amount of work done in each exercise; the left-hand curve was obtained before treatment, the right-hand (marked by X) after treatment. In the first case the value of E before treatment was 5,800 foot-pounds and 7,100 foot-pounds after treatment; thus the patient's efficiency had increased 1,300 foot-pounds or 21 per cent.

Seven patients treated with oxygen and tested in this way: in one there was no improvement; in one the value of E rose 11 per cent.; and in five more than 17 per cent.; in three of these cases the value of E rose to over 70 per cent., a figure not far short of 77·5 per cent., the value of E for untrained healthy men; in two others the value of E rose to 67 and 68 per cent. Before we had devised the method of testing patients over a series of exercises, and so measuring the increase in their efficiency, we tried to estimate the improvement of a patient by taking his dyspnœa ratio over the same exercise before and after treatment: this enabled us to decide whether he had improved or not, but not to measure his improvement exactly. In five patients tested in this way there was a considerable fall in the dyspnœa ratio in three cases, and a slight fall in two.

The results of oxygen treatment in these cases may be summarized as follows: of twelve patients eight improved considerably; three improved slightly; one did not improve. I leave it to you to judge from these results how far oxygen treatment is of value in cases of chronic gas-poisoning.

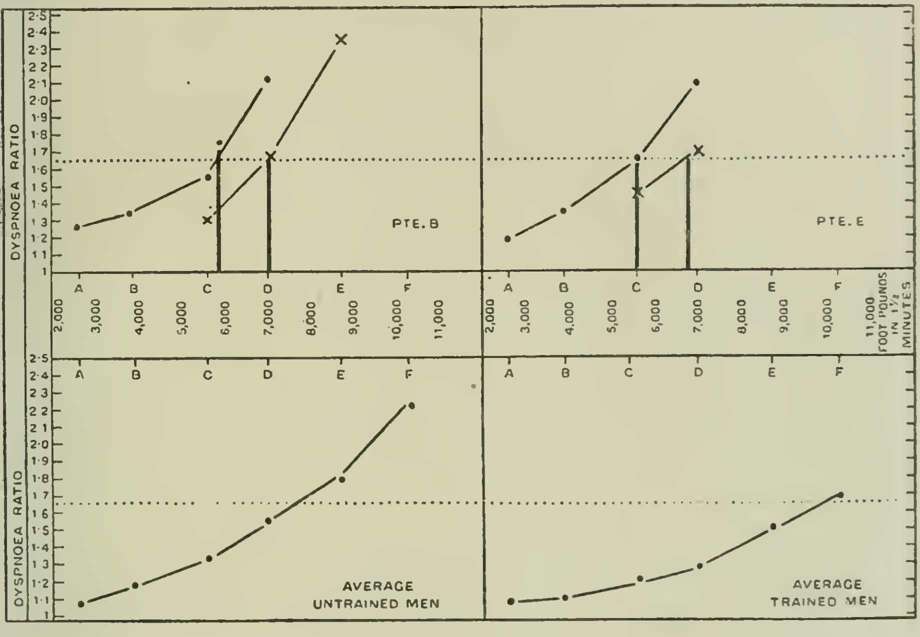


Fig. 3.

•—•—•—• Curve before treatment.
×—×—×—× Curve after treatment.

Dr. Haldane.

I would like, before saying anything in supplement of Mr. Barcroft's remarks and of his and Dr. Hunt's experiments, to draw the attention of the meeting first to some more general points concerning the oxygen treatment of cases of gassing, and especially acute cases.

The first point is, that even a very small degree of anoxæmia (deficiency of free oxygen in the blood) will, if given time, have a serious effect on the individual. The data which show that most strikingly are those which relate to "mountain sickness." One has only to go, unacclimatized, to an atmospheric pressure, say between 8,000 and 9,000 ft., and though at first there is no effect on the breathing, except during exertion, mountain sickness will probably develop in a few hours. There is no doubt that this is due to a deficiency of oxygen, and yet there may be no noticeable cyanosis. Those who have experienced mountain sickness know that it is a horrible complaint; there is vomiting, headache, and often diarrhœa, with extreme depression, and the symptoms may be delayed till after a return to normal level. They are due to the effect of anoxæmia on the nervous system. The same is true of carbon-monoxide poisoning.

The second point I want to make is, that anoxæmia is a common condition among ordinary patients, and is well worth while taking early measures to prevent; for if serious anoxæmia is allowed to go on for a considerable time, the patient gets into a hopeless condition. This is seen particularly in carbon-monoxide poisoning. A man unconscious from that condition may be brought into the fresh air, and may get rid of the deficiency of oxygen in two or three hours, as can be ascertained by examining the blood, but may still remain unconscious, and be very likely to die, simply from the effects of lack of oxygen during the exposure to the carbon-monoxide. A patient who has been in a bad way for some time from oxygen deficiency has little chance of recovery. It must not be expected that giving him oxygen so that his lips turn red will result in an immediate recovery. Slight anoxæmia can be recognized by giving a little oxygen and noting the change in colour and in the pulse. In a person who is not anoxæmic there is no change in the colour of the lips.

With regard to the existence of anoxæmia in acute cases, there is important recent American evidence in papers in the *Journal of Experimental Medicine*, September, 1919, by Stadie and Harrop. They took samples of arterial blood from the radial, and determined, by a rough method, but good enough, the deficiency in saturation of the arterial blood. Stadie's cases were principally pneumonias of different types, partly broncho-pneumonias after influenza, but also croupous pneumonia. In practically all there was a deficiency in saturation of arterial blood; in some it was as great as 50 per cent. With such a deficiency as that the patient always died, and I do not wonder. There is a deficiency in croupous pneumonia, even when the patient is going on well. Harrop's series were heart cases, emphysema, bronchitis, &c. If arterial anoxæmia is present and is doing harm to the patient, it is possible to prevent it.

There are two causes for the arterial blood in the lung not being properly saturated. The first is, that the epithelium has become thickened and clogged by exudation, hence the rate at which the oxygen gets into the blood is very much diminished, and there is not time for the blood to be saturated as it passes through the lungs. It is certain that this was the case in acute lung-irritant gassing; all the evidence pointed to this. Another cause was revealed by the experiments of Professor Meakins, Dr. Priestley and myself, at the Taplow Canadian Hospitals, in chronic gas cases. It is a cause which is apt to occur in all sorts of cases. It is that if the lung is not expanding equally, or if air is not entering at an equable rate to all the alveoli, some alveoli will contain much too little oxygen to saturate the blood, others will have more than enough. The result of that must be—there is physiological evidence from dissociation curves, &c.—that the arterial blood, though containing no more than the normal amount of CO_2, has a deficiency of oxygen, and hence there is anoxæmia.

How is that brought about? Our experiments show that it can be brought about in a normal person by shallow breathing. We found that shallow breathing was one of the defects in the so-called chronic cases of gassing, so we determined to ascertain what was the effect of shallow breathing on the blood of normal people. In my own case, for instance, if, while sitting quietly, my rate was in consequence of shallow breathing, increased to 30 or 40, instead of my normal 15 per minute, in two or three minutes I developed Cheyne-Stokes breathing, with the long stately periods, waxing and waning. That was abolished by adding a little oxygen to the inspired air. Professor Arthur Keith pointed out, in a paper published about ten years ago, that the lungs open out something like the opening out of a lady's fan. If the breathing be shallow, only certain leaves of the fan open out. There is stagnation of the other parts, and consequent anoxæmia.

Priestley and I were responsible for the present methods of getting samples of alveolar air, and this was an eye-opener. The alveolar air we got with the shallow breathing was normal, and yet there was arterial anoxæmia. The effects of shallow breathing are very much more marked lying down. I get Cheyne-Stokes breathing if my breathing is so slightly limited that I have to breathe 22 per minute, instead of the normal 8 or 9 when I am lying down; and that throws a great light on orthopnœa. In bronchitis and such conditions the distribution of air among the alveoli is interfered with by resistance. Air is coming in quickly to some parts, slowly to others, so that there is this cause of anoxæmia operating, although the lung epithelium may be all right all the time.

With regard to chronic gas cases, we approached them from the same point of view that Mr. Barcroft was doing at Cambridge. Judging by the stethoscope, the lungs appeared to be all right, but these patients were short of breath, and we found that this shortness was of a very peculiar type. If the man went on working or walking, his breathing became very rapid and ineffective, and there would be, perhaps, only 1·5 per cent. of carbon dioxide in the expired air. The shallow breathing was abnormal; it was dyspnœa, and not simply hyperpnœa. The respiratory centre was abnormal or neurasthenic, and we found that ordinary fatigue caused by resistance to breathing produces the same condition. Various toxic influences and anoxæmia itself have a similar action. One can see the respiratory centre failing in a steel chamber at very low atmospheric pressure. We watched that in experiments last summer, when we took in an unacclimatized subject and watched his breathing gradually going to pieces; it got shallower and faster, and he got more and more stupid, until we had to take him out. He could not remember his last hour in the chamber at all. The chronic gas cases had respiratory neurasthenia, causing shallow rapid breathing on exertion, instead of slower deep breathing. In these and similar cases there are also fits of very rapid shallow breathing with urgent asphyxial symptoms; often these acute attacks occur at night.

I regard the experiments which have been made at Cambridge and Stoke as of extraordinary interest, as showing how such cases can be helped by putting them into air enriched with oxygen. When they are in the enriched air they never get the bad attacks. Also, they can sleep soundly if they are breathing enriched air. Thus they have a proper chance of real rest and recovery.

I had meant to show the mask apparatus which was used in France and at Taplow, for administering oxygen to individual patients; but as time presses this can only be shown at the end of the meeting. I think this apparatus is likely to be useful in a very large variety of ordinary medical cases; but I hope that oxygen chambers may also soon become available.

Colonel CUMMINS.

I fear I was, and am, only "the man in the street" in so far as concerns the use of oxygen in gas cases, but I was very fortunate in being associated with so many people who really knew the physiological side of the subject. I think one of the great difficulties from which an administrative body like the headquarters of an Army Medical Service necessarily suffers from is, that knowledge of a kind which may prove to be essential tends to be shut up in the heads of ultra-specialists, people who have worked perhaps for a lifetime at a particular line, who have published their results in very special journals which are not read generally, and whose knowledge at the same time is suddenly found to be absolutely essential at some juncture. And that, I think, is what happened when the Germans started gas warfare in France. Thanks to Wright, Leishman, Bruce, and others, the Army had developed a school of pathology to meet the needs of the ever-present military problems, but we had never developed a school of physiology. Perhaps, however, that is not quite accurate, because Colonel Melville had done so in regard to food and exercise.

With reference to oxygen, I do not think there was a regular officer in the Army Medical Service who had so far specialized in physiology as to be able to deal adequately with the therapeutic problems that arose on the use of gas. We were, however, at once able to exploit the services of a splendid team of men, and I only wish it were possible in some way to express the obligation which the Army is under to Dr. Haldane, Mr. Barcroft, Colonel Douglas, and so many other physiologists who worked for us. At the end of the war the situation was much clearer. It was obvious to anyone who had had opportunities of seeing it, that oxygen did good in gas cases, but I do not think that even yet the situation is absolutely clear. Much has been learned, but much still remains to be learned.

Mr. Barcroft has given us the history of the treatment of chronic cases and it seems to be a clear story. In the future people suffering from chronic gas poisoning will be dealt with in that way. I hope Colonel Douglas will talk about the treatment of acute cases, which is a more intricate problem, as oxygen chambers cannot be organized under battle conditions.

Speaking as a "man in the street," my impressions at the end of the war are these: That although it is certain oxygen can do much good in the acute case of poisoning, as, also, I feel certain, in influenzal bronchopneumonia and shock, in both of which there is an element of anoxæmia, still there is a reluctance on the part of general practitioners to use oxygen as freely as it should be used; and in saying this I use the terms general practitioner and R.A.M.C. officer as interchangeable. The men who were reluctant to use oxygen were the practitioners in uniform.

and very good officers they were. How is this reluctance to be explained? In the first place, though it is easy to put a man into a chamber that does not irritate him at all, when a man is suffering from intense anoxæmia, as in an acute gas case, it is very far from easy to administer oxygen through a facepiece or mask. The Haldane apparatus is excellent: it saves much oxygen because none is lost during expiration and it certainly increases the alveolar oxygen pressure, but it was difficult to apply in cases in which the men were suffering from much nervous irritation and great difficulty in getting breath at all. It is difficult for such men to stand the facepiece. I remember trying it in a case, and sitting up all night with the man, but he could only tolerate the facepiece for short intervals. Perhaps it was my own fault, for Colonel Douglas, when he tackled similar cases, was able to get the facepiece worn for a much longer time. I do not know how small the smallest possible chamber would be. Perhaps it might be possible to design a better facepiece or mask, something with rigid walls, that might be applied over a man's head, with a glass front, so that one could see the change in colour, in which the oxygen could be given at a regulated pressure, and CO_2 absorbed or otherwise got rid of—an apparatus which would not at the same time irritate the patient. I do not know whether any experiments have been done on those lines. Barcroft's method is specially important in military practice at bases, and these chambers should be in all hospitals, civil or military. There were alternative methods to the Haldane apparatus which were sometimes tried in France. Many people who found it difficult to use a facepiece gave oxygen simply by connecting up flexible rubber tubes with cylinders and passing the tubes up the nose. I think this method was started by Adrian Stokes immediately after a gas attack in 1916. It was possible, from one cylinder, to give many men oxygen at the same time. But it was very irregularly distributed, and there was much waste. Yet much good was done by it, and it was tolerated by patients who might not have tolerated a facepiece. I frequently afterwards saw oxygen used in that way spontaneously by officers in military hospitals for influenzal pneumonia and gas cases. It occurs to me that its popularity is due to the fact that it is an easy method to apply and easy to look after, although not calculated to give quite such good results as the more complicated Haldane apparatus. This points to the need of research towards getting easier and more universally applicable methods of administration of oxygen in acute cases and perhaps this discussion may yield suggestions as to how this may be accomplished.

Dr. C. G. Douglas.

We, in France, saw the aspects of gas-poisoning from a different point of view from that of Mr. Barcroft and his colleagues, as they were dealing with the cases at a later stage, while we saw them in the acute stage.

It was obvious after the first gas attack that oxygen administration did some good to cases exhibiting cyanosis resulting from acute pulmonary œdema. After an interval of months, during which time the Germans made no drift gas attacks, we suddenly got another batch of cases, many of which were in the gravest condition. It was some time before we could satisfy ourselves as to how much good the oxygen was doing. The cases were either deeply cyanosed, of a purple colour, obviously short of oxygen, or they had that grey colour of lips and tongue which is associated with a profound deficiency of oxygen and collapse. At first efforts were made to give oxygen by means of a simple tube and funnel. I admit that you can, if you use enough oxygen, partially relieve the cyanosis, but it is a wasteful and ineffective method and needs constant attention.

The next stage in the development of oxygen administration was, as Colonel Cummins has said, the nasal tube method, which was first used after a very serious gas attack in August, 1916, when I saw at the same time in two adjacent clearing stations at least one hundred cases of the most severe type of acute pulmonary œdema. This method of administration proved successful on this occasion. It was improvised by Stokes on the spur of the moment, and it was frequently used afterwards. At about the same time Haldane produced his apparatus for giving oxygen, and we owe it largely, if not entirely, to the energy of Colonel Cummins that we were able to try the apparatus, to find out its advantages, and gain some insight into any defects it might have. There is no doubt one can often do an enormous amount of good by administering oxygen by a suitable method, such as that devised by Haldane, but many methods of administration are not really effective.

I will quote a couple of cases I came across, as they illustrate the type of case we often met in France. These two cases had been gassed about twenty-four hours before I saw them. They were both treated by the Haldane apparatus, which proved very convenient, and they tolerated the mask without difficulty. The first of these men was in a very serious condition, pale, lead coloured, his speech incoherent, and he was practically unconscious. Pulse 140, very weak and irregular, in fact hardly countable; respirations 50 or more. I could relieve his cyanosis and get a definite change of colour by giving 4 litres of oxygen a minute, and if I gave 5 litres a minute, mixed with his inspired air, his colour became normal. But what was in some ways even more striking was the change in the pulse, for in a moment or two the pulse-rate fell by twenty beats, and became regular and of very much better quality than before. And so long as we kept up the oxygen administration the improvement continued. On stopping the oxygen, cyanosis returned in a moment or two, the pulse again became fluttering. But again there was improvement as soon as oxygen was given again. As Dr. Haldane says, when a man has been subject to a profound lack of oxygen for some time you cannot expect immediate complete recovery to ensue as soon as you give oxygen, though the colour may change and the circulation show improvement. In this man a number of hours elapsed before there was material diminution in the rate of the respirations. His whole condition, however, was obviously improving and he was becoming more conscious. We kept him on practically continuous oxygen administration for three days and he got better, and when I last saw him he was almost well.

The second case was even worse. The man appeared to be absolutely moribund. I was able to get some improvement in his colour by giving him 5 litres of oxygen a minute by the Haldane apparatus, but even when I gave him 10 litres a minute he was not of quite normal colour. I had to keep him on about 6 litres of oxygen a minute, because with a faster rate of supply the escaping gas made enough noise to disturb the patient and cause him to become restless. I was up all night with him, and next morning his colour was definitely improving, and then I had to leave the oxygen treatment to be carried on by others. That man was eventually evacuated to England and recovered perfectly. He went out to France again, when, unfortunately, he was wounded.

These instances are enough to show how one may tide a case over a critical period by treatment with oxygen.

So far as I can make out, from the observations of Dr. Peters and other medical officers, the earlier that oxygen administration can be started in cases of acute pulmonary œdema from irritant gas poisoning the greater is the chance of rapid recovery.

I think Colonel Cummins is right when he talks of the reluctance medical officers sometimes display in using the Haldane apparatus. I think they are apt to be somewhat alarmed at it because it is a machine, and sometimes fail to realize the simplicity and convenience of the apparatus. But in several instances in which men had taken up this apparatus and had used it with judgment, I was very much struck by the reliance that they placed upon it. There were not very many cases which would not tolerate the mask. Those men who did not tolerate it could be advantageously treated by the nasal tube method, though this involves a good deal of wastage of oxygen. The effect of oxygen treatment in the acute cases was so obvious that I know of one unit abroad in which, as a routine measure, the medical officer of the ward instructed the sister-in-charge that if a case was admitted exhibiting even the slightest signs of gas poisoning he was to be instantly treated with oxygen, pending the arrival of the medical officer, who might be engaged with other cases. The two cases I have cited were treated with nothing but oxygen. There are, of course, other valuable methods, such as venesection, but that is not a matter for discussion at this meeting. Sometimes gas casualties were admitted in a very grave condition of collapse and pallor, and the medical officer found that by treating them with oxygen for a couple of hours or so considerable improvement set in, and venesection could then be carried out with advantage, because after giving oxygen the collapse diminished and the character of the case changed for the better.

One other point about oxygen administration, as we saw it, was that a number of medical officers failed to appreciate the fact that if a man was suffering from grave and long-continued lack of oxygen, it was necessary to keep up the oxygen administration practically continuously until cyanosis no longer reappeared when the oxygen administration ceased. I have known gravely cyanosed gas cases which were treated with oxygen for only a brief period every hour, though an oxygen apparatus was at hand and in good order, the supply of oxygen adequate, and the beneficial effects of increasing the oxygen concentration in the air in the lungs perfectly obvious to the administrator, the cyanosis disappearing and the pulse improving within a few moments of the treatment being started.

Dr. Ryle.

Such observations as I can record to the meeting are purely clinical, and they are based upon that particular gas attack of August, 1916, to which Colonel Cummins has already referred. We were suddenly flooded with a large number of gas cases as a result of a drift-gas attack with combined phosgene and chlorine. We knew that the method of giving oxygen by the funnel was inefficacious. We tried to improvise masks, but they were not tolerated, and apparently did not give sufficient oxygen. It was then that the idea occurred to Captain Adrian Stokes of using the nasal catheter. That method I applied, then and frequently afterwards, and, I think, with very great success. The method was to attach to the tube leading from the oxygen cylinder a No. 8 rubber catheter. This was lubricated with vaseline, and passed directly along the floor of the nose to a distance of 4 in. into the nasopharynx; the catheter was looped up to the side of the face and fixed there by strapping, and the oxygen allowed to run in gently. By that means we were able to keep alive men who had been apparently desperately gassed, and often had the good fortune to see them recover. By that means we gave oxygen continuously for twenty-four, forty-eight, even seventy-two hours. I have frequently seen men admitted in a state of grey pallor and cyanosis, the condition one associates with that extensive œdema of the lungs produced by phosgene. They have come in with an almost imperceptible pulse, with, on several occasions, a respiration rate of over 60, in a semi-conscious condition, cold and collapsed, and I have seen these same men, after seventy-two hours of oxygen inhalation with the respirations down to 24 make a steady recovery and get quite fit to travel down the line by train without accident. A pink colour returned to the face as soon as the oxygen was given, and, as Colonel Douglas has said, striking changes in their pulse and respiration rates were noticed.

One of the chief obstacles to the use of the Haldane mask, one which has not been emphasized sufficiently, is the serous fluid and froth which the men get rid of by the mouth in bad cases of lung œdema. As much as a pint of serous froth may be poured out in a few hours. It is very important to keep the nasal passages and mouth clean and free from froth, and care must be taken that the mask is not clogged by that froth. Further it is necessary to give the patients fluid nourishment, and occasionally even solid food. This is possible when they are having oxygen through the catheter. When using the catheter the men could be placed into any desired posture, and a thing which they appreciated was, that if the catheter slipped out of place, they could themselves replace it with the greatest ease. Until we developed a better technique, it was not unusual to see men re-inserting their tube. We were able to supply four men from one kerosene drum improvised to serve as an oxygen distributor.

We classified our cases into three groups: the mildest were the cases which needed merely the oxygen they could get by being out of doors. The medium cases seemed to be sufficiently benefited by a small dose of pure oxygen from a nitrous oxide bag given at intervals of five minutes in every quarter of an hour, sometimes at longer intervals. Finally there were the severe cases, for which continuous inhalation of oxygen was obviously the only thing.

It was suggested that we should try a double nasal catheter, one passed down each nostril, but a practical difficulty is that so many persons have deflections of the nasal septum, and it was frequently found that only one nostril would take the tube. I subsequently used the catheter method not only for gas cases, but also for the lung œdema of trench nephritis, a condition which gives a closely similar picture to that produced by phosgene. I have seen cases of trench nephritis materially benefited by using oxygen by the nasal catheter. In acute purulent bronchitis it was useful, too, for temporary relief of cyanosis.

I have once also employed continuous oxygen in a case of severe hæmorrhage. It improved the colour, allayed the restlessness and appeared to tide the patient over the danger period. The only other common use of oxygen at casualty clearing stations was in association with warm ether or nitrous oxide anæsthesia.

Dr. R. A. Peters.

My experiences also are active service experiences, and, in view of what Colonel Cummins and Colonel Douglas have said, I think it is unnecessary to say any more about the value of the oxygen treatment in gas cases in the early stages.

There is one contribution which I think I can make to this discussion, though what I shall say is more in the nature of impressions than actual notes. I had an opportunity of treating a commanding officer of mine, who had an extremely bad attack of broncho-pneumonia. He was a very active officer, and it was a busy time, and he did not want to go sick. Therefore for two days he stayed there when he ought to have gone away. Finally I got him to bed, and I had to be up with him the whole of one night. It was during that night I thought of using oxygen in this condition. I could not get him into the casualty clearing station that night. As the night progressed his breathing and his heart's action became very bad, and then he commenced Cheyne-Stokes breathing. I got a Haldane apparatus and started giving him oxygen.

Two litres a minute seemed to quieten his breathing down, and I think it did him a lot of good. I gave it at about half-hourly periods, and as the night wore on I got down to twenty-minute periods, then to a quarter of an hour in the hour, and that kept him all right: he got off to sleep, and next morning he was very much better. Though it is impossible to get any evidence as to how he would have fared in the absence of oxygen, I feel it is probable he was actually saved by the treatment. The Cheyne-Stokes respiration ceased directly he had the oxygen. He was not having a much greater strength of oxygen than there is in normal air, but it was enough for the purpose; it would work out at about 30 to 40 per cent. oxygen in the air breathed through the apparatus.

Dr. Frank Shufflebotham.

At this point of the discussion I do not propose to say what I had originally intended to bring before the meeting as I feel I ought to try to crystallize my remarks in the tersest possible way. I have been invited to take part in this discussion because of the work which has been done on oxygen research under the auspices of Sir Walter Fletcher and the Medical Research Committee at the North Staffordshire Infirmary, Stoke-on-Trent.

The early results of the treatment of gas-poisoning cases by the continuous inhalation of oxygen which was carried out at the Physiological Laboratory, Cambridge, under the direction of Mr. Barcroft in 1917, showed such possibilities that experimental research on a larger scale was commenced at the North Staffordshire Infirmary, where two oxygen wards containing four beds were installed. Great difficulties were experienced in completing this installation owing to shortage of suitable materials as well as to the shortage of labour. The wards were open in October, 1918, and up to the present time over a hundred cases have been admitted. The cases include for the most part those of chronic gas-poisoning, but in addition there have been cases of acute gas-poisoning, pneumonia, carbon-monoxide poisoning and two cases of pernicious anæmia.

The model which is shown on the table in front of me gives some idea of the structure of the chambers, and the photographs show that these oxygen wards are furnished in the same way as ordinary wards and are just as comfortable. The short description of each photograph makes it sufficiently clear how the wards are constructed and equipped, and the plan gives some idea as to the arrangement of these wards.

Very shortly an extended report of this work will be published by the Medical Research Committee[1] and from this report it will be seen that the results of the work carried out at Stoke-on-Trent may be viewed most favourably. The patients suffering from chronic gas-poisoning derived great benefit from a course of this treatment in the general state of their health and in many instances symptoms of long standing such as cough, shortness of breath, expectoration, asthma, and irregular action of the heart became less severe in nature and frequently disappeared. The physical conditions of the lungs and heart also undergo a considerable improvement.

The technical details connected with the construction and the working arrangements of this chamber are of the greatest importance and I venture to give a quotation from the report to the Medical Research Committee as follows:—

> The research wards occupy the whole of the third story of a ward which is detached from the main block of hospital buildings. These wards consist of two small oxygen wards and a wide corridor with suitable lavatory accommodation, all of which are shown in the plan. The cubic capacity of Ward I is 709 cubic ft., and that of Ward II, 858 cubic ft.
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> The iron screens which separate the chambers from the corridor, and from one side of each ward, are made with sheet steel at the bottom and plate glass above, the latter being fixed in steel frames.
>
> The windows are double, the outside ones consisting of ordinary wood sashes and the inner ones of iron frames with an air-tight casement, the top of which can be opened by unscrewing the bolts which are attached to the frames.
>
> The doors giving access to the rooms are double, forming a vestibule, as shown in the plans and photographs, and each door is opened by three sets of iron levers. These doors are constructed of iron and are made air-tight by rubber joints and are hung in iron frames. On entering the chamber one opens the outer door by means of the levers and closes it again before opening the inner door. The cubic capacity of the vestibule in each case is 53 cubic ft.
>
> The food doors, which are 18 in. by 12 in., are double and are constructed in exactly the same way as the doors for entering the chambers. An iron shelf is fitted over them for putting articles on while opening and closing the doors. The cubic capacity of the space between the outer and inner food doors is 1·4 cubic feet for each ward.
>
> On the outside of the screen—that is to say, in the corridor—at the foot of

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[1] This report, when published, will be fully illustrated with a diagram of the ground plan and photographs of the wards, chambers and apparatus.

the glazed part of the screen, is a wooden shelf which is specially constructed so that the Haldane air analysis apparatus can travel along and the same apparatus be used for both wards.

The walls and ceilings of the chambers are plastered and have coves at the ceiling and next to the floor in the usual way, and the floor is made of jointless material which has been made and laid on the old bordered floor of the ward. The walls and ceilings are well painted with ripolin, a well-known enamel paint. A mezzanine floor, which is shown in the plan, has been put down to carry the gas bag.

The oxygen cylinders are kept on the ground floor, as indicated in the photographs.

The wards contain the ordinary ward furniture and each ward contains two beds.

In the corridor there are the scrubbers for purifying the atmosphere of the chambers by the absorption of: (1) Carbon dioxide. (2) Excess of moisture in the atmosphere. (3) Other impurities of respiration, such as small quantities of sulphuretted hydrogen, fatty acids from the skin, &c.

The carbon dioxide is removed by means of a scrubber which contains three trays placed one above the other, each tray containing 38 lb. of fresh burnt lime with 10 per cent. solution of caustic soda, and there is a fourth tray which is placed on the top of the scrubber containing a solution of 5 per cent. potassium permanganate and 5 per cent. solution of caustic soda. The second scrubber is for the purpose of withdrawing an excess of moisture from the atmosphere, and it consists of two trays. In the bottom tray is placed ½ cwt. of calcium chloride and in the top ¼ cwt. of sterilized charcoal. It is understood that in the first scrubber the potassium permanganate is used for the purpose of extracting from the impure atmosphere any traces there may be of sulphuretted hydrogen, the fatty acids of the skin, or other bodies.

The temperature of the chambers is regulated by means of a large coil contained in a tank and surrounded by a wooden encasement. This apparatus is described as a cooler on the plan.

The circulation of the atmosphere throughout the chambers is carried out by means of a rotary pump, which is also shown on the plan. The oxygen is transmitted from the cylinders, which are placed on the ground floor, by means of a pipe to the top floor of the building and passed into the chambers by means of a reducing valve. The circulation of gas through the chambers is shown and also the connexion between the chambers and the gas bag, which has been devised in order to effect an economy in oxygen consumption.

The estimation of the amount of oxygen and carbon dioxide is carried out by the Haldane apparatus; and in the latter case a special apparatus has been devised by Messrs. Siebe, Gorman and Co., Ltd., with a view of more accurately determining the percentage of carbon dioxide in the atmosphere of the chambers.

The following points are of interest in the practical working of the chambers :—

(1) It has now been found that in the working of Ward I, with its cubic capacity of 709 cubic ft., after the chamber has been filled that only from 65 to 75 cubic ft. of oxygen are required each day to maintain the percentage of oxygen at between 45 and 50, and with regard to Ward II, with a cubic capacity of 858 cubic ft., that from 80 to 90 cubic ft. of oxygen are required daily for two patients in order to keep the atmosphere at the same percentage which is mentioned in connexion with Ward I.

(2) With regard to the carbon dioxide scrubber, it is necessary after five days to dampen the lime with 10 per cent. of caustic soda solution, and the soda lime should be renewed after ten days' working.

(3) The potassium permanganate solution in the top tray of the first scrubber may require replenishing after five days in order to keep the atmosphere of the chamber free from stuffiness.

(4) The water should be drawn off from the second scrubber twice a day, and at the end of every five days calcium chloride should be replenished by adding about 20 lb. From time to time it may be necessary to break up the calcium chloride, which cakes, and to replenish it.

(5) With regard to the charcoal, it has been found necessary to add 30 lb. after each five days' working.

The arrangements which are here described for the circulation through the chambers afford most efficient method of ventilation.

The chambers are illuminated by electric light. Special care has been taken with regard to the wiring and to the fittings for the prevention of fire.

The corridor is heated by electric radiators, and a fire-escape iron ladder has been erected so that in case of fire patients or nurses will be able to escape through the window marked A in Plan I.

From time to time it has been necessary to make small improvements in the various parts of the installation, and at the present time further improvements can be made, especially with regard to the potassium permanganate scrubber, upon which Mr. T. R. Parsons, of Sidney Sussex College, Cambridge, is experimenting. In the two cases of pernicious anæmia, and in cases of gas-poisoning in which the patients have had a septic condition of the mouth, the air of the chamber has presented the unmistakable odour due to fatty acids of the skin and to other bodies, and in these instances the potassium permanganate scrubber has entirely failed to remove this unpleasant condition. The atmosphere, however, has been rendered odourless by the use of ozone generated by the Quain ozonizer.

It will be understood from the plans that there is no independent circulation for each chamber; but this alteration I hope will be made very shortly, so that different classes of cases may be treated in each chamber.

I do not propose to discuss the physiological questions which have been raised by Mr. Barcroft and Dr. Haldane and I shall leave Dr. Sowry to deal with the clinical aspect of the work which has been carried out at Stoke-on-Trent. All that I will say is this, that men who have been unable to work on account of chronic gas-poisoning and other conditions for a considerable period of time have not only improved so much in health that they could return to light labour but in many cases they have been able to go back to their former employment.

Cost of Oxygen Treatment.—There is one aspect of oxygen treatment which has not been touched upon this afternoon and that is the cost. At Stoke-on-Trent, where, I am convinced, the strictest economy has been exercised in regard to the supply of oxygen to the wards consistent with the percentage of oxygen in the atmosphere being in excess of 40 per cent., and also with regard to the materials necessary to keep the scrubbers in a state of purity, and in spite of all efforts to keep down the cost in every direction, it has not been possible to bring down the daily cost per patient to less than £1 3s. 6d., and this estimate does not include any sum which could be put down to capital charges. When one is dealing with pneumonia cases which, it is hoped, will benefit largely by treatment in oxygen wards, the cost is such that it is very likely to handicap research in this direction unless money is forthcoming from further grants or from philanthropic sources. The high cost of oxygen is due to the fact that the production of oxygen in this country is in the hands of a monopoly. Dr. Harker, who has been interested in nitrogen production, has suggested that we might use for this work instead of pure oxygen from the cylinders the bye-product of the air after the nitrogen has been extracted from it, and another suggestion is that liquid air which contains about 60 per cent. of oxygen could be used for these cases. As far as my own observations have gone, I am convinced that there is a great field of usefulness for oxygen treatment as well as a great field for research, and considering the amount of human suffering which may be alleviated by such treatment, it seems almost a scandal that the work should be handicapped because of an oxygen monopoly.

Dr. G. H. Sowry.

When I was asked by the National Medical Research Committee to take charge of the oxygen chambers at Stoke-on-Trent, I did so with the utmost diffidence. It is twenty-five years since I left St. Bartholomew's Hospital, and during that time I have not been in touch with experimental physiology and pharmacology, except in so far as those subjects directly affect the daily work of a busy practising physician. I could only approach the subject from a purely clinical standpoint, but as this satisfied the Research Committee, and as all new remedies must, before acceptance, satisfy a purely clinical test, I felt that even with my limited time and capabilities I might do some useful work, so I undertook the task.

Dr. Shufflebotham has described the structure and working of the chambers. I should merely like to say that they worked very satisfactorily. The percentages of carbonic acid and moisture were always well under control, and the patients never complained of any discomforts due to the air they breathed.

It was originally intended that the chambers should be used for the treatment of workpeople who were accidentally poisoned at poison gas factories and shell-filling stations in the North of England, but the demands for treatment from soldiers who had been gassed at the Front led to the bulk of the patients being of the latter class. I will deal first with the seventy cases of "gassing." In all, the gassing had occurred at a more or less distant date, varying from four months to over two years. In most cases the nature of the gas could not be ascertained, though in the earlier cases it was probably chlorine, later phosgene, and still later "mustard gas."

Outline of Treatment.—The patients were admitted for periods of five or six days, and remained in the chambers for about twenty hours each day, being allowed out for exercise about four hours each afternoon. The chambers were surcharged with oxygen, the percentage being between 35 and 40 in the earlier cases, and from 50 to 60 in the later. After their discharge, they were asked to report for examination, two, four and six weeks later.

Before treatment the patients were examined and notes made as to: (1) Subjective treatment—such as cough, expectoration, sleeplessness, dyspnœa, palpitation on exertion, asthmatic attacks, &c., clinical examination of heart, lungs, &c. (3) The chest was X-rayed. (4) An examination of the blood was made, the red cell count and hæmoglobin percentage noted. (5) The response to effort was tested by three tests: (i) Short violent effort—by swinging of arms; (ii) medium effort—by going up and down stairs; (iii) a prolonged effort —by walking round the hospital grounds.

Symptoms on Admission.—Most patients complained of: (1) Cough and expectoration. (2) Dyspnœa, palpitation and precordial pain on exertion. (3) Attacks of nocturnal dyspnœa — in all essentials like true spasmodic asthma. (4) Sleeplessness. (5) Poor appetite and dyspepsia. (6) Mental depression.

Physical Signs on Admission.—Cyanosis was present in a small percentage of the cases. *Lungs*: Emphysema was present in about 40 per cent of the cases. Nearly all of them exhibited râles, rhonchi, or crepitations over the whole or part of the lungs. In many cases over the lowest 3 in. or 4 in. of the bases behind there was slightly impaired resonance and fine crepitations. *X-ray* examination in nearly all the cases showed a condition of fibrosis especially near the roots of the lungs. The appearance of the plates strongly suggested a condition of peribronchial phthisis, but in only two cases have I proved the existence of active tuberculosis. If the condition is one of true fibrosis, there can be little hope of a permanent cure. *Heart*: Only a few cases of V.D.H. were found, and in about ten cases there was hypertrophy. The most obvious condition found was a very poor response to effort. At rest the heart sounds were in most cases clear and the action regular—though in a little less than half the cases there was some irregularity with extra-systole. But even slight exertion in nearly all the cases caused a great increase of pulse-rate, with marked irregularity. *Nervous System*: Very few showed any physical signs of organic disease, beyond increased tendon reflexes and some vasomotor instability.

Results of Treatment.—First, to take a general view of the patients. The *immediate results*: In the large majority of cases the patients felt decidedly better for the treatment. They felt brighter, more cheerful, and more energetic. Their appetites improved and their digestions were better. Sleeplessness in most cases improved, and where patients suffered from asthmatic attacks this symptom disappeared. Coughs improved and expectoration was reduced. The response to effort as measured by the tests generally improved in most cases considerably. It is difficult to estimate the results numerically, but a careful classification justifies me in saying that of the 70 cases reported on, 23 were very much improved, 30 were much improved, 10 were improved, and 6 not improved. The *latest results*, as estimated on the last examination (usually six weeks after the end of treatment) were on the whole very satisfactory, though there was certainly a tendency for some of the original improvement to be lost. Numerically expressed, I should say that of the 61 cases who reported, 15 were very much improved, 22 were much improved, 10 were improved, and 13 were not improved. It is important here to notice the fact that throughout the investigations I was constantly confronted by the difficulty that is recognized by all who have to do with cases in which pensions or compensation allowances are in question. In several cases the patients' description of their subjective symptoms signified that there was no improvement. Their coughs, expectoration, dyspnœa, &c., were "no better." But on examination of their chests the pre-existing râles, rhonchi, &c., had disappeared, and their tests were much better performed. In two cases this discrepancy was *admittedly* due to the patients' reluctance to acknowledge any improvement until their pensions were settled.

I will now consider the effect of treatment on each of the common symptoms:—

Coughs were very much improved. Patients who complained of months of sleeplessness due to coughing slept quite well after twenty-four or forty-eight hours' treatment and coughed very little; 25 per cent. lost *all* cough before the end of treatment, and a further 40 per cent. were very much better. In 60 per cent. the improvement was very largely maintained up to the end of the six weeks.

Expectoration improved in a corresponding degree. One very noticeable fact was that many patients who came in with muco-purulent sputum, at the end of treatment only coughed up mucus. In one case —admitted with copious muco-purulent sputum—he has not had muco-purulent sputum since treatment, fifteen months ago.

Dyspnœa and palpitation on exertion also improved considerably. Not only did they feel less distress on exertion, but they performed all the tests with much less disturbance of respiration and pulse.

Asthmatic attacks were complained of by fourteen patients. In seven cases the attacks ceased during treatment, and in four of them there was no recurrence within the six weeks. One patient for months before treatment was regularly awakened by attacks three or four times every night. He slept in the general ward the night before admission to the oxygen chambers, and had three distinct attacks. During his six nights in the chamber he did not have even a slight attack, and only had one mild one during the following six weeks.

Appetites improved almost marvellously, and most of the patients who complained of dyspepsia lost it during treatment.

Condition of the Heart.—Twenty-five patients had physical signs of organic or functional disease of the heart. Of these, fifteen were very much improved immediately after treatment, and twelve of them fully maintained their improvement when last seen. The most common evidence of heart trouble was a bad response to effort. For example, in one case—before treatment: Pulse 76 at rest, 120 after exercise test, very irregular and not returning to normal for eighty seconds. After treatment: Pulse 82 at rest, 96 after test, quite regular and returning to normal in thirty seconds.

Condition of Lungs.—Forty-three patients had physical signs of disease of the lungs, such as râles, rhonchi, &c.; of these, thirty-two were very much improved at the end of treatment, and in thirty-one the improvement was fully maintained at the end of six weeks. It is worthy of note that those patients who had the most marked physical signs of disease were those who showed the most improvement. Several of the cases had slight impairment of resonance and fine crepitation over the lower 3 in. or 4 in. of the bases of both lungs. In nearly every case the resonance was normal, and no crepitations could be heard at the end of treatment.

The *exercise test* of response to effort was one of the most decided evidences of the value of oxygen treatment. Professor Barcroft showed by curves the decided improvement which resulted from this treatment. My experience confirms his in dozens of cases. On admission sixty-two patients performed the test with a response to effort which was below the normal. About forty of them showed a very decided improvement, and more than thirty of them maintained their improvement at the last examination.

Blood Counts.—Owing to the use of a faulty hæmocytometer in my earlier cases, the red counts were not reliable, but in the last twenty-nine cases they were quite reliable. Contrary to reports by previous observers, I do not find anything approaching a uniform polycythæmia. Of the twenty-nine cases, the majority (eighteen) showed a red count *below* the normal, and with one exception showed an increase of red cells after treatment. Eleven cases had polycythæmia in varying degree, and in every case there was a decrease of red cells after treatment. Thus, whether the red cell count was above or below the normal before treatment, the effect of treatment was to cause a return towards the normal.

The results of treatment in "gassed" patients were, in my opinion, very good, whether those results were judged by a general survey of the cases or a careful analysis of the effects on detailed subjective symptoms or objective signs.

In order that these experiments should be purely a test of the value of prolonged inhalation of oxygen, I have not allowed my patients to take any medicine whatever—such as expectorant mixtures, heart tonics,

&c. If, however, this line of treatment is of value—and I strongly think it is—it would undoubtedly be of additional help to the patients if they received, in addition to the oxygen treatment, other forms of treatment, such as medicines, vaccines, &c.

Critics have suggested that the results are due to the rest and good food which the patients receive whilst under treatment, but when it is recognized that nearly every patient has been in hospital for prolonged periods, and there had the same rest and good food, but without the same good results, I think the objection is invalid. Another criticism is that the whole result is a consequence of suggestion, but this is negatived by the fact that the objective signs were as much improved as the subjective symptoms.

I will now briefly discuss the treatment of other diseases :—

Two cases of pernicious anæmia were treated, one for twenty-three days and the other for twelve days. Both were severe cases, one being a relapsing case. Both of them professed to feel much better for the treatment and certainly seemed decidedly improved. But detailed examination of the blood showed that the patients were worse after treatment. After discharge, they both steadily got worse and one has since died. Unfavourable results in two cases—both severe—is not sufficient ground, however, for not experimenting further, especially in early cases.

I now come to five cases in which the predominant symptom was respiratory difficulty, and in all five cases the results were so good as to be almost startling. And it is in such cases that I think the value of treatment in oxygen chambers is most in evidence. Two men were acutely and severely poisoned by poison gas, whilst employed in emptying cylinders returned from France after the Armistice. Both were treated at home by oxygen inhalations, medicines, &c., but they both rapidly got worse. They were admitted to the oxygen chambers, both arriving in a very bad condition. They were cyanosed, breathing heavily and rapidly and coughing incessantly. In both cases improvement began very soon after admission and they rapidly improved. Cyanosis disappeared within an hour, and the breathing—although it remained rapid for twenty-four hours or more—was not at all laboured or distressing. Both were quite well, with their lungs normal, on the third day. A case of acute *coal gas* poisoning was brought to the Infirmary and treated in the casualty department by stimulants, oxygen inhalations, &c., but in spite of the treatment his condition got rapidly worse. He was admitted to the chambers, shivering, very cyanosed, breathing very heavily and in a state of collapse. Five minutes after admission the shivering ceased, and half an hour later his breathing was easy, colour good and pulse regular, slow and strong. A case *of broncho-pneumonia with empyema* in a child, aged 3, who fifteen days before had had a rib resected and the right pleura drained. The child's condition got steadily worse, and on November 29, when death seemed imminent, I admitted her to the chambers. On admission, she was deeply cyanosed, breathing heavily (60 per minute), restless, picking at the bed clothes, her pulse was 120, very thready and weak; she was in a cold perspiration, and almost unconscious. *Two hours later* the nurse reported, "Colour very much improved, breathing less laboured, has asked for a drink." Another two hours later, "Pulse 120, good volume, respiration 48; taking food with apparent relish; breathing much easier, colour quite good; has had several short quiet sleeps." The child continued to improve rapidly for twenty-four hours, and during the time she was quite free from respiratory distress. Unfortunately vomiting and diarrhœa supervened, and she gradually sank and died three days later. I do not claim in this case more than that the treatment relieved her want of oxygen, and stopped all respiratory distress. She died eventually from the toxic effects of her disease, but the oxygen undoubtedly greatly increased her chances of recovery. Lastly, acute lobar pneumonia developed in a soldier, who was undergoing the treatment for gassing. When his illness began he was transferred to the general ward for observation. He had complete consolidation of the whole of both lower lobes, and his was a severe case. He returned to the oxygen chambers twenty hours after the onset of his pneumonic symptoms. On admission, his breathing was very laboured and painful (48 per minute), and there was marked cyanosis and restlessness. In three hours nurse reported: "Colour quite good, pulse much improved, breathing much less laboured, though still painful." After twenty-four hours the pleuritic pain ceased, and from that time his breathing was easy and colour quite good—in fact his condition was remarkably favourable for a man with both lower lobes completely solid. He made a very rapid and complete recovery. I have never in my twenty-seven years' clinical experience seen a severe case of double basal pneumonia make such remarkable progress.

All these five cases suffered from want of oxygen in a severe form. They all improved almost immediately they entered the chambers.

I am convinced that treatment by the continuous inhalation of a *warm* atmosphere surcharged with a known percentage of oxygen, holds out great promise in the treatment of many diseases. The experimental work done on gassed soldiers—successful as I think the results have been—should be merely the beginning of much more experiment, and I venture to predict that in a few years no large hospital will be thought complete unless an oxygen chamber forms part of its equipment.

Dr. P. HAMILL.

My studies of the effects of oxygen administration have been made on patients suffering from cardio-vascular disease and a few cases of opium poisoning. When for any cause an adequate amount of oxygen does not get absorbed into the system the results on the circulation are very severe. Some years ago Dr. Clark and I studied the circulatory changes in Cheyne-Stokes respiration[1] and showed that during the apnœic period there was a fall of blood-pressure and in some cases partial heart-block. Administration of oxygen in high concentration abolished the heart-block and maintained the blood-pressure at a uniform high level. Side by side with the improvement in the circulation there was in some cases a return of consciousness—a striking demonstration of the efficacy of oxygen administration. (*Diagrams shown.*)

In order that really satisfactory results may be obtained it is necessary to administer oxygen in a concentration of 40 to 50 per cent. for hours at a time, which is not an easy matter even in hospital practice. The nasal tube is satisfactory on the whole, but wasteful. Most methods of administration by a mask necessitate the constant attendance of the nurse and are almost equally wasteful. Especially in warm climates, patients complain of a sensation of stuffiness and the slight resistance to breathing caused by the mask is badly tolerated.

There is a great need for a simple portable apparatus on the lines of that used for insufflation anæsthesia, in which oxygen can be circulated and the CO_2 absorbed. On the grounds of portability and cost, sodium peroxide has much to recommend it as a source of oxygen in preference to cylinders.

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[1] *Journ. of Pharmacol. and Experim. Therap.*, 1914, v, p. 357.

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Section of Therapeutics and Pharmacology.

President—Dr. H. H. DALE, C.B.E., F.R.S.

Case of Diabetes Insipidus.[1]

By E. P. POULTON, M.D.

(ABSTRACT.)

DR. POULTON described a case of diabetes insipidus, in which the volume of urine was reduced to within normal limits by subcutaneous injection of pituitary extract twice a day. The dose was 15 minims to start with, and this was later reduced to 10 minims. He referred to Motzfeld's experiments—in which an artificial polyuria, caused by placing water in a rabbit's stomach, was arrested by subcutaneous and intravenous injections of pituitrin. In the anæsthetized animal, pituitrin causes an increased flow of urine, as Schäfer originally pointed out. In the unanæsthetized animal the action is quite different. The anæsthetic probably was the deciding factor in these two types of experiment. According to Motzfeld's work, diabetes insipidus was due to a lack of vasomotor tone of the renal nerves, which could be restored by injections of pituitary extract. The patient had had a tuberculous breast removed, and there were large tuberculous glands present in the groins. A radiogram of the sella turcica was normal.

DISCUSSION.

Dr. OTTO MAY: The following is the record of a case of diabetes insipidus treated by subcutaneous injections of pituitary extract. The patient first noticed slight thirst and polyuria in 1913, when aged 19. He joined the

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[1] At a meeting of the Section, held March 16, 1920.

Territorials in October of that year. He was called up at the outbreak of war, and although slightly troubled by thirst, &c., he completed his training, going to France at the beginning of November, 1914. In the spring of 1915, under the strain of active warfare, the thirst and polyuria became considerably aggravated, and he was sent to hospital in England in April, being finally invalided out in February, 1916. At that time, thirst was intense, and he was passing 15 to 20 pints of urine daily. Soon after this he came under my care. After trying various other remedies with no success, I began treatment with pituitary in July, 1916, using 1 c.c. of "infundin" (B. W. and Co.), injected into the buttock. The effect was immediately most striking. The first injection resulted in nearly twenty-four hours' absolute comfort—no thirst and the secretion of urine falling practically to normal. Since then, the patient has received between 800 and 900 such injections, averaging five a week. There have been absolutely no untoward effects: under the influence of the injection, the specific gravity rises from its usual 1000-02 to 1010-15, and the patient carries out his ordinary life in comfort. He has been doing his work (clerical) since 1916, and plays vigorous tennis in the summer and rows in "pairs" and plays football in the winter. Unfortunately there is no cumulative effect in the injections: after about twenty hours the symptoms reappear, and a further injection is necessary. A trial was made of various pituitary preparations given by the mouth, but none was found to have the slightest effect. It looks at present as though he will have to continue the injections indefinitely; fortunately up to now there has never been even the slightest septic complication, though most of the injections have been given him by a friend whose only training has been a British Red Cross first-aid course. It is interesting to note that the urine has throughout been free from any apparent abnormal constituent—no albumin nor diacetic acid. The injections never have any effect on his systolic arterial pressure, which varies between 115 and 135. During treatment his weight has gradually increased from 11 st. to $11\frac{1}{2}$ st. As regards ætiology, there is no history of any syphilis or other definite illness, nor is there any history of other cases in the family. His father died, he thinks, of phthisis; his mother, brother and sister, the only other members of the family, are all healthy. A skiagram of the skull showed apparently a normal sella turcica, and there are no other symptoms suggestive of gross pituitary disease.

Dr. F. PARKES WEBER: In regard to the part played by the pituitary gland in the causation of diabetes insipidus, I wish to emphasize the fact that grave lesions of the gland (tuberculous, syphilitic, &c.), may be unaccompanied by any change in the size or shape of the pituitary fossa and the sella turcica, as evidenced by Röntgen skiagrams during life. In a remarkable case of diabetes insipidus in a tuberculous subject, which I described with Dr. H. Schmidt in in 1916,[1] there was a peculiar necropsy finding in the posterior lobe of the pituitary gland, though Röntgen-ray examination during life had yielded a negative result. The posterior lobe was obviously *relatively* enlarged, and it almost completely enclosed the anterior lobe. This enlargement was apparently due to the presence of large clumps of big granular cells with small deeply-staining nuclei. The granules in the cytoplasm of the cells were of a lipoid substance, and could be well-coloured by Sudan III. The question of the mechanism by which pituitrin exerts its effect in cases of diabetes insipidus is intimately connected with the nature of the lesion in the pituitary gland or elsewhere.

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[1] F. P. Weber and H. Schmidt, "Diabetes Insipidus with a Peculiar Necropsy Finding in the Posterior Lobe of the Pituitary Gland," *Amer. Journ. Med. Sci.*, Philad., 1916, clii, p. 892.

Dr. LEYTON: Since many problems have arisen this afternoon, I should like to add to them by relating briefly a case which came my way a short time ago, but which I had no opportunity of investigating very thoroughly. The patient—a young man—was suffering from diabetes insipidus, the Wassermann reaction had been negative six times out of seven, and the luetin cutaneous test was negative too. A radiograph of the skull failed to show any alteration in the sella turcica. The administration of large doses of thyroid by the mouth relieved the polyuria greatly, and this relief lasted after the withdrawal of the drug. Ultimately necrosis of some of the bones of the nose seemed to point towards syphilis as the origin of the disease, from which recovery had occurred without specific treatment.

The following communications were also made:—

April 20, 1920.

F. RANSOM, M.D.: "The Action of Strontium on Frog's Heart."

GEORGE GRAHAM, M.D.: "The Source of Uric Acid excreted in Urine after Administration of Atropin."

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War Section.

President—Surgeon Rear-Admiral Sir ROBERT HILL, K.C.M.G., C.B., C.V.O., R.N.

A Retrospective View of Naval Medical Conditions.[1]

By Surgeon Rear-Admiral Sir ROBERT HILL, K.C.M.G., C.B., C.V.O.

Medical Director-General, Royal Navy.

I DEEM it a high honour to have been elected President of the War Section of the Royal Society of Medicine for the first year of its existence. My pleasure in being permitted to deliver the opening address is only tempered by my diffidence and fears for your powers of endurance. To Sir Humphry Rolleston, Sir Rickman Godlee, Mr. C. H. Fagge, Sir John Bland-Sutton, Colonel W. Pasteur, and last, but by no means least, Sir John MacAlister, must be given credit for the conception of creating a War Section of the Society. They have our thanks now, and I feel assured will have the gratitude of future generations of medical men.

On the outbreak of hostilities, medical men in all paths of activity, from all parts of the United Kingdom and Overseas, joined up so whole-heartedly with the Naval, Military and Air Forces, and such a large number of them have now become identified with one or other of the Services, that I feel sure they will be loth to lose that connexion and will be glad of opportunities to meet their old comrades. The meetings of this Section will provide opportunities for many happy reunions.

This will be its social field of usefulness. I can see another—the formation of a skeleton medical organization—by which, should the need ever arise for the Services to call again on the medical profession to augment their ranks, the various elements of our profession will be utilized, each in its proper groove, and the strain be exerted equally throughout the land. Owing to the want of some such scheme during the ordeal through which we have passed, a great deal of specialized talent was lost for a time. Many incongruities occurred, for example, ophthalmic surgeons found themselves in charge of medical wards, and eminent surgeons acted as little more than dressers. One case of hidden talent was discovered in the Grand Fleet after the war had been in progress for over two years. A well known dentist had joined the R.N.V.R., for purposes of recreation before the war, as an A.B. On the outbreak of hostilities he was called up, and served as a most efficient A.B., and was discovered eventually in a mine-sweeper, when engaged one day at Scapa Flow slung over the ship's side in a "bo'sun's chair," painting ship. An Admiral, who had been a patient of his in pre-war days, passed by in his barge, and was considerably surprised to see a bluejacket waving his paint brush at him. He stopped the barge and discovered his friend the dentist. In explaining it to me the Admiral said in a shocked voice: "Here is this man who, when at home, would have a string of carriages waiting for his patients outside his door, slung over a ship's side in a bo'sun's chair."

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[1] At a meeting of the Section, held November 10, 1919.

I think it will be only fair that we, who have our experiences fresh in our minds, should sketch some outline of an organization for the benefit of future generations to whom the war will be of historic interest only.

Those of you who are not associated with the Royal Navy, will pardon me if, as a Naval medical officer, I turn for the main part of my theme to events connected with the Service to which I belong. A retrospective view of the conditions under which Naval surgeons worked in the years of sea fighting which saw the 1st of June, Camperdown, Copenhagen, St. Vincent, the Nile, and Trafalgar, may have an interest for many of us, and a comparison of these conditions with our modern life at sea may lead to useful deductions. Everyone knows the Naval history of those glorious times, but very few have ventured to probe into matters concerning the rank and file of the medical service of the period. Possibly they have been wise, for much has sunk into oblivion, and little has been recorded, except of the leaders in thought or action. What manner of man was the Naval surgeon then, how did he carry out his duties and under what conditions?

During the period to which I refer, 1795-1840, the medical service of the Royal Navy does not appear to have been a popular one, and difficulty must have been experienced in obtaining an adequate number of medical men, except for the ships cruising in home waters. The adventurous life, the large experience to be gained in surgical work, the continuous state of war and the acquisition of prize money were attractions for many, but on the whole the conditions of service were not good. We can gather this from available reports[1] in which are found observations on the point, often humorous though perhaps not so intended. When the newly-appointed surgeon of the *Serapis* arrived on board his ship on the East Indies Station in 1798, and produced the warrant of his appointment, he was greeted by the commanding officer of the ship with the remark, "I am surgeon myself, and have been so near four years. No surgeon is allowed, and I want none." The familiar example of the unhappy ship is occasionally met with, as when the surgeon of the *Violet* in 1810 writes, "Grant me my discharge or death will soon put an end to my troubles." The surgeon of the *Alban* in 1829 deplores the fact "that he has no assistance in the sick berth, and that he has to make his own poultices in the galley," while the medical officer of Ascension objects to rendering professional services to the horses and cattle on the island. The surgeon of the *Monarque*[2] relates how, during an epidemic of typhus, the Admiral, being prejudiced in favour of James' powder, insisted on its being administered in cases of fever. This proved fatal to many from the violence of its operation, and sometimes four or five were buried in a day. The Admiral, on being remonstrated with, gave up his favourite nostrum and, according to the surgeon, "the mortality ceased of course."

A few foreigners were given warrants from time to time, and it appears that occasionally the medical officer of a captured enemy ship was entered. The difficulties which the Dutch, French, and Russian medical men experienced with our language is apparent in some of their official returns, the efforts of the Dutchmen to make themselves intelligible being particularly quaint. In 1820, Surgeon J. Hall, of the *Agamemnon*, but "formerly of the Imperial Russian Navy," writes his journal entirely in Latin, which naturally leads one to suspect that his knowledge of English was limited.

But withal, there were many medical men of originality and high professional attainments serving at sea. Many of them were quick to exploit new methods or put forward new ideas in prevention and treatment of disease. In 1797 cinchona bark in wine was in general use in the West Indies Squadron for prevention of malaria. In the *Aboukir*, in 1808, pneumonia was regarded as an infectious disease, and in 1813 the surgeon of the *Racoon* was so convinced of this, that he isolated the pneumonia patients from the remaining sick. In 1811 the surgeon of the *Crocodile* thus remarked on the cases of diarrhœa which occurred on board his ship: "These all arose in the month of April, at which time the ship was laying guard and flagship in Grouville Bay, Jersey, where oysters are very plenty, and at this time of the year are very apt to produce a looseness almost approaching to cholera amongst the inhabitants." In 1827 the surgeon of the *Atholl* recommended the use of lime as a general purifier of water and precipitant of organic matter. In 1807, in a ship serving on the East Indies Station, the surgeon gave his scorbutic patients "green peas soaked in water and allowed to vegetate," with excellent results, a method which is interesting in view of recent pronouncements on the subject of scurvy, and shows how often throughout the ages an old remedy comes to the fore again in its turn.

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[1] Public Records Office. Admiralty Medical Journals.

[2] W. H. Long, "The Journal of a Naval Surgeon," "Naval Yarns," p. 61.

In the days of the old sea battles, the Medical Service laboured under manifold disadvantages as compared with the profession on shore, for sick berth attendants do not appear to have been employed until thirty-five years after Trafalgar, separate messes for the sick on board sea-going ships did not come into existence until 1804, and probably were not universal until after Trafalgar, the sick being messed in their sleeping places before then, and the earliest mention of first-aid instruction was in 1855. Until the year 1840 we find surgeons deploring the want of trained nursing for the sick and suggesting the formation of a sick berth staff. For skilled assistance the surgeon depended on his mates, of whom he might have three, two or one, the number varying with the class of ship. In a small ship he had no assistance of any kind. But, alas, too often the surgeon's mate was only a partly trained man, and sometimes quite blissfully ignorant of medicine or surgery. The official supply of drugs and appliances can not be described as on a generous scale and the surgeon was compelled to supplement his stores by purchasing various articles from the Apothecaries' Society of London.

It is remarkable how few have made any record of the preparations for reception and treatment of the wounded at sea. Perhaps it was thought that an account of an ever-recurring routine would be only of mundane interest. The fact remains that though some allude to their preparations in a sketchy way, technical details are difficult to find. The duties of the surgeon during and after an action must have been very arduous. He had to undertake all operative work on board his ship, and would consider himself fortunate if an early opportunity presented of sending his patients on shore for further treatment. Contrast this with our modern routine in which only a magnified first aid is attempted, and where the evacuation of the wounded is our first thought, both for their good and also for the greater efficiency of the ship as a fighting unit. Unless the circumstances are exceptional, a Naval surgeon now does not perform any major operation on board, for steam and antiseptics have entirely altered the situation. Even with our modern advantages, trained bearers and nurses, skilled assistants, specially fitted dressing stations and all the surgical equipment possible to provide, we know that the best end-results are obtained if operation is deferred until our patient is placed at rest in the base hospital ship or the base hospital.

All have read descriptions of the cockpit—that small, evil-smelling, ill-ventilated space in the depths of the ship to which the surgeon betook himself during action, there to await the stream of wounded men, without anæsthetic, antiseptic or skilled assistance. Robert Young, surgeon of the *Ardent*, sixty-four gun ship, thus describes his experiences at Camperdown in 1797: "All of these were wounded in the action of eleventh of October, in which I had no mate having been without one for three months before. I was employed in operating and dressing till near four in the morning, the action beginning about one, afternoon. So great was my fatigue, that I began several amputations under a dread of sinking before I should have secured the bloodvessels. Ninety wounded were brought down during the action, the whole cockpit, deck, cabins, wing berths and part of the cabin tier, together with my platform and my preparations for dressing were covered with them, so that for a time they were laid on each other at the foot of the ladder where they were brought down, and I was obliged to go on deck to the commanding officer to state the situation and apply for men to go down the main hatchway and move the foremost of the wounded further forward into the tiers and wings, and thus make room in the cockpit. Numbers, about sixteen mortally wounded, died after they were brought down, amongst whom was the brave and worthy Captain Burgess.[1] . . . Melancholy cries for assistance were addressed to me from every side by wounded and dying, and piteous moans and bewailings from pain and despair. In the midst of these agonizing scenes, I was enabled to preserve myself firm and collected, and embracing in my mind the whole of the situation, to direct my attentions where the greatest and most essential services could be performed. Some with wounds, bad indeed and painful, but slight in comparison with the dreadful condition of others, were most vociferous for my assistance and these I was obliged to reprimand with severity as their noise disturbed the last moments of the dying. I cheered and commended the patient fortitude of others, and sometimes extorted a smile of satisfaction from the mangled sufferers and succeeded to throw momentary gleams of cheerfulness amidst so many horrors. The man whose leg I first amputated had not uttered a groan or complaint from the time he was brought down, and several exulting in the news of the victory declared they regretted not the loss of their limbs. An explosion of a salt box with several cartridges, abreast of the cockpit hatchway, filled the hatchway with flame, and in a moment fourteen or fifteen wretches tumbled down upon each other, their faces black as cinder, their clothes torn to shatters and the rags on fire. . . . After the action ceased fifteen or sixteen dead bodies were removed before it was possible to get a platform cleared and come at the materials for operating and dressing, those I had prepared being covered over with bodies and blood and the storeroom blocked up. I have the satisfaction to say that of those who survived to undergo amputation or be dressed, all were found next morning in the gunroom where they were placed in as comfortable a state as possible, and on the third day were conveyed on shore in good spirits cheering the ship at going away, smoking their pipes and jesting as they sailed along, and answering the cheers of thousands of the populace who received them in Yarmouth Bay." The *Ardent*, a ship with a complement of 485, had 41 killed and 107 wounded at Camperdown, the total casualties being 30·51 per cent. of complement.

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[1] Captain of the *Ardent*.

Truly a harrowing description. It is of pathetic interest to know that her equally gallant little namesake, the destroyer *Ardent*, was sunk at Jutland with the loss of seventy-eight out of her complement of about ninety men. To turn to a more pleasing incident, illustrating how the old-time Naval surgeon had to be ready for all emergencies, it is recorded[1] that at the Battle of the Nile during the heat of the action a woman was delivered of a fine boy on board the *Goliath*. Nature, in kindly effort to restore the nation's losses, did not choose a propitious moment, for the surgeon at the time had to deal with the casualties of the ship's company, twenty-one killed and forty-one wounded.

[1] W. H. Long, "Battles of St. Vincent and the Nile," "Naval Yarns," p. 198.

It is interesting to note that there are points of similarity between experiences at the Battle of Jutland and those of the surgeon of the *Ardent* at Camperdown. MacLean and Stephens,[1] medical officers of the *Lion* at Jutland, write: "Nearly all the casualties occurred within the first half-hour. A few cases found their way to the foremost station, but the great majority remained on the mess deck. During the first lull, the medical officers emerged from their stations to make a tour of inspection. The scenes that greeted us beggar description, even were the censor to permit a detailed account. Most of the wounded had already been dressed temporarily. Tourniquets had been applied in one or two instances, but we were able to remove these later. Hæmorrhage on the whole was less than we anticipated. . . . The battle was thrice renewed during the evening, but in the lulls all the wounded were carried to the mess deck. . . . During the evening ten of the desperately wounded and burned succumbed. . . . At 7.30 a.m., on June 1, we were informed that it would be safe to bring the wounded up from below. The vice-admiral's and captain's cabins were cleaned, dried and thoroughly ventilated. This process took some time since both were full of water and smoke. The captain's bathroom was rigged as an operating theatre, and by 8.45 a.m. we commenced. . . . As might be expected, lack of water was a source of anxiety. At first it was brought in mess kettles all rusty and brown. Later the cold supply was laid on, and finally about noon a free supply of both hot and cold was at our disposal. In all fifty-one cases were dealt with, and a general anæsthetic, chloroform and ether in equal parts, was administered to twenty-eight. . . . Only urgent operations were attempted; as all the patients had already waited a long time, speed was essential. Our work was severely handicapped by having 44 per cent. of casualties amongst the medical officers and sick berth staff."

Penfold,[2] medical officer of the *Barham* at Jutland, writes: "When the action commenced, all the arrangements in the dressing stations had been completed, the necessary instruments sterilized and laid out in trays, lotions made up, morphia solution in Jena bottles and the staff clean and ready for their work. One slight casualty had come to the fore station during the first hour of the action and this had been dressed; the man would have been sent back to his post, but just after dressing his wound a heavy shell burst close to us in the fore part of the ship and completely wrecked the dressing station, causing many casualties in this part of the ship. On recovering from the effects of this explosion, it was found that all the lights were out in our compartment, which seemed to be filled with débris, and the air thick with smoke from the adjoining store-room which was on fire, but no flash had been felt and no nitrous fumes were noticed; water was rushing in (from burst pipes) and the compartment was quickly flooded."

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[1] A. MacLean and H. E. R. Stephens, "Surgical Experiences in the Battle of Jutland," *Journ. Roy. Naval Med. Ser.*, ii, No. 4, p. 421.

[2] E. A. Penfold, "A Battleship in Action," *Journ. Roy. Naval Med. Ser.*, iii, No. 1, p. 44.

At Jutland, the *Lion* had ninety-five killed and fifty-one wounded, representing 11·87 per cent. of complement, and the *Barham* had twenty-two killed and forty-six wounded, or 6·04 per cent. of complement.

Tables of the casualties at the Battles of Camperdown, the Nile, and Trafalgar, are of interest when compared with a table showing the casualties at the Battle of Jutland.

I will now attempt to analyse Table IV, *Casualties at the Battle of Jutland*. It should be remembered, however, that such analysis, though prepared in the calm seclusion of an office, must necessarily be somewhat arbitrary in its various distinctions, but nevertheless it may give some idea of the causes and character of injuries.

BATTLE OF JUTLAND, MAY 31-JUNE 1, 1916: REMARKS ON CASUALTIES (*see* TABLE IV).

Killed outright or Drowned.—Many of the killed had combined injuries—shell wounds and burns—but it is estimated that the chief *fatal* cause in 343 cases were shell wounds and in 114, burns, while in four there was no trace of injury. The remainder of the deaths, 5,553 (shown in first column) are presumed to have been from drowning, though it is probable that in the ships which were sunk there were numerous cases of injury from the enemy's fire. The number of deaths from shell wounds and from burns respectively varied greatly in individual ships. Thus, in the *Barham* all (22) were deaths from shell wounds and in the *Malaya* all (33) from burns; in the *Lion* 48 of the killed had combined wound and burn, 49 had shell wounds only and 2 had burns only; in the *Princess Royal* and the *Tiger* all the deaths (19 and 20 respectively) were from shell wounds. There were no deaths from scalds.

Died of Wounds.—There were many cases of combined injury, but the chief causes of death in 27 were shell wounds and in 46 were burns. In the *Malaya* all the cases under this heading died from burns.

TABLE I.—BATTLE OF CAMPERDOWN—CASUALTIES.

Ships	Complements	Killed	Wounded	Total casualties per cent. of complement
Russel	584	—	7	1·19
Director	485	—	7	1·44
Montagu	584	3	5	1·36
Veteran	485	4	21	5·15
Monarch	593	36	100	22·93
Powerful	584	10	78	15·06
Monmouth	485	5	22	5·56
Triumph	634	29	55	13·24
Venerable	587	15	62	13·11
Ardent	485	41	107	30·51
Bedford	584	30	41	12·15
Lancaster	485	3	18	4·32
Belligueux	485	25	78	21·23
Isis	338	2	21	6·80
Totals	7,398	203	622	—

Total force at Camperdown, 8,221.

Total casualties per cent. of force, 10·03.

TABLE II.—BATTLE OF THE NILE—CASUALTIES.

Ships	Complements	Killed	Wounded	Total casualties per cent. of complement
Goliath	584	21	41	10·61
Zealous	584	1	7	1·36
Orion	584	13	29	7·19
Audacious	584	1	35	6·16
Theseus	584	5	30	5·99
Vanguard	589	30	76	17·99
Minotaur	634	23	64	13·72
Defence	584	4	11	2·56
Bellerophon	584	49	148	33·73
Majestic	584	50	143	33·04
Swiftsure	584	7	22	4·96
Alexander	584	14	58	12·32
Leander	338	0	14	4·14
Totals	7,401	218	678	—

Total force at the Nile, 7,985.

Total casualties per cent. of force, 11·22.

TABLE III.—BATTLE OF TRAFALGAR—CASUALTIES.

Ships	Complements on October 21, 1805*	Killed	Wounded	Total casualties per cent. of complement
Victory	815	57	102	19·50
Royal Sovereign	811	47	94	17·38
Britannia	786	10	42	6·61
Temeraire	711	47	76	17·29
Prince	679	—	—	—
Neptune	595	10	34	7·39
Dreadnought	703	7	26	4·69
Tonnant	664	26	50	11·44
Belleisle	556	33	93	22·66
Revenge	610	28	51	12·95
Mars	621	29	69	15·78
Spartiate	599	3	20	3·83
Defiance	582	17	53	12·02
Conqueror	622	3	9	1·92
Defence	568	7	29	6·33
Colossus	617	40	160	32·41
Leviathan	592	4	22	4·39
Achille	605	13	59	11·90
Bellerophon	569	27	123	26·36
Minotaur	586	3	22	4·26
Orion	543	1	23	4·41
Swiftsure	557	9	8	3·05
Ajax	668	2	9	1·64
Thunderer	556	4	12	2·87
Polyphemus	461	2	4	1·30
Africa	475	18	44	13·05
Agamemnon	475	2	8	2·10
Euryalus	273	—	—	—
Naiad	275	—	—	—
Phœbe	294	—	—	—
Sirius	258	—	—	—
Pickle	31	—	—	—
Entreprenante	35	—	—	—
Totals	17,772	449	1,242	--

Total force at Trafalgar, 17,772.

Total casualties per cent. of force, 9·51.

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* *Numbers on Board on October* 21, 1805.—Neither the total force at Trafalgar nor the complements of the ships can be found in any historical account of the battle. These figures have been obtained from the Admiralty Muster Books, preserved at the Public Records Office. The majority of musters took place on October 20, 1805, but some a few days earlier. For various reasons only the "numbers mustered" have been taken.

TABLE IV.—BATTLE OF JUTLAND: CASUALTIES.

Class	Ships	Killed outright or drowned	Died of wounds	Wounded		Burnt		Combined Injuries: Wounded and Burnt		Gas Poisoning		Total wounded	Total casualties	Complements on May 31, 1916	Total casualties per cent. of complements
				Severe	Slight	Severe	Slight	Severe	Slight	Died	Recovered				
Battle-ships	*Barham* ...	22	4	4	9	19	8	—	2	—	—	46	68	1,124	6·04
	Colossus ...	—	—	2	6	—	—	—	1	—	—	9	9	884	1·01
	Marlborough ...	2	—	—	2	—	—	—	—	—	—	2	4	1,119	0·35
	Malaya ...	33	32	1	4	7	23	—	—	—	1	68	101	1,032	9·78
	Valiant ...	—	—	1	—	—	—	—	—	—	—	1	1	1,063	0·09
	Warspite ...	9	5	4	15	5	3	—	—	—	—	32	41	1,048	3·91
Battle Cruisers	*Lion*	95	5	9	11	3	13	7	3	—	—	51	146	1,229	11·87
	Princess Royal	19	3	8	6	6	52	6	—	—	—	81	100	1,202	8·31
	Tiger	20	2	5	8	8	9	5	1	3	5	46	66	1,281	5·15
	Queen Mary ...	1,258	1	1	4	—	—	—	—	—	—	6	1,264	Ship sunk	—
	Invincible ...	1,026	—	—	1	—	—	—	—	—	—	1	1,027	„ „	—
	Indefatigable ...	1,017	—	—	—	—	—	—	—	—	—	—	1,017	„ „	—
Cruisers	*Defence* ...	902	—	—	—	—	—	—	—	—	—	—	902	„ „	—
	Warrior ...	65	3	9	19	1	2	—	—	—	2	36	101	832 (ship sunk)	12·13
	Black Prince ...	856	—	—	—	—	—	—	—	—	—	—	856	Ship sunk	—
Light Cruisers	*Calliope* ...	7	3	9	17	—	—	—	—	—	—	29	36	363	9·91
	Caroline ...	2	—	—	—	—	—	—	—	—	—	—	2	338	0·59
	Castor	12	1	9	16	—	—	—	—	—	—	26	38	356	10·67
	Chester... ...	29	6	8	22	3	7	2	1	—	—	49	78	424	18·39
	Dublin	3	—	6	21	—	—	2	3	—	—	27	30	460	6·52
	Southampton ...	29	5	15	20	4	1	—	—	—	—	60	89	498	17·87
Flotilla Leaders	*Broke*	47	—	9	23	—	4	—	—	—	—	36	83	200	41·50
	Tipperary ...	184	—	2	2	—	—	—	—	—	—	4	188	Ship sunk	—
Destroyers	*Acasta*	6	—	1	—	—	—	—	—	—	—	1	7	—	—
	Ardent	78	—	1	—	—	—	—	—	—	—	1	79	Ship sunk	—
	Defender ...	1	—	—	2	—	—	—	—	—	—	2	3	—	—
	Fortune ...	67	—	1	1	—	—	—	—	—	—	2	69	Ship sunk	—
	Moorsom ...	—	—	—	1	—	—	—	—	—	—	1	1	—	—
	Nessus	7	—	5	2	—	—	—	—	—	—	7	14	—	—
	Nestor	6	1	7	—	—	—	—	—	—	—	8	14	—	—
	Nomad	8	—	3	1	—	—	—	—	—	—	4	12	—	—
	Obdurate ...	1	—	1	—	—	—	—	—	—	—	1	2	—	—
	Onslaught ...	5	—	3	—	—	—	—	—	—	—	3	8	—	—
	Onslow	2	1	—	2	—	—	—	—	—	—	3	5	—	—
	Petard	9	—	3	3	—	—	—	—	—	—	6	15	—	—
	Sparrowhawk..	6	—	—	—	—	—	—	—	—	—	—	6	—	—
	Spitfire ...	5	1	5	13	—	—	1	—	—	—	20	25	—	—
	Shark	85	1	1	1	—	—	—	—	—	—	3	88	Ship sunk	—
	Porpoise ..	2	—	1	1	—	—	—	—	—	—	2	4	—	—
	Turbulent ...	89	—	—	—	—	—	—	—	—	—	—	89	Ship sunk	—
	Totals ...	6,011	74	131	243	56	122	23	11	3	8	674	6,688		

Total Force, Grand Fleet, about 60,000. Total Casualties per cent. of Force, 11·14.

Nature of the Wounds.—Amongst those that died from wounds and amongst the wounded were the following compound fractures: skull 14, upper jaw 1, lower jaw 2, clavicle 3, scapula 2, humerus 13, ulna 2, spine 2, ribs (with chest penetration) 5, pelvis (crest of ilium) 1, femur 12, tibia and fibula 15, tibia 2, fibula 3, pulping of feet 1. There were several cases of eye injury requiring enucleation, and many of injury to the hands or feet, necessitating amputation of fingers or toes. In the majority of the slightly wounded cases, the lesions were lacerated wounds of the soft parts, caused either by minute shell splinters, by splinters formed by parts of the ship, or by shrapnel in a few instances.

Burns.—The burns were caused either by the momentary flash of enemy shell explosion (chiefly noticed in light cruisers and small craft) or by ignition of cordite. The former produced the more numerous cases, but in the burns from cordite ignition, owing to the more prolonged exposure to the flame, the mortality was much greater than in those caused by shell explosion. The large majority of the deaths from burns on board the ships resulted from cordite ignition; in the *Malaya* 65 deaths were from this cause. Amongst the surviving wounded, it was noted also that burns from cordite ignition were the more extensive and became the more septic. The exposed parts of the body, the face, neck, scalp and extremities, were burnt in nearly all cases, and in 15 the trunk was affected also.

There were only 6 cases of scald (caused by burst steam-pipes).

Gas Poisoning.—The few cases of gas poisoning which occurred, were caused by the burning of cordite or by shell explosion. I believe there is no evidence that the enemy used poisonous gases in their shells. Six cases, two of which proved fatal, were due to nitrous gases, the symptoms appearing at a considerable interval after inhalation and, in the fatal cases, including cough, expectoration, dyspnœa, cyanosis and râles, indicative of œdema of the lungs. In the remainder of the cases (5) the effect was produced probably by a mixture of gases, including carbon monoxide; in one of these on board the *Tiger*, death occurred very rapidly.

So long ago as 1598, George Whetstone, an Elizabethan gallant, poet, soldier and traveller, in a work which is probably the earliest devoted to Naval medicine in our language,[1] wrote: "In the expeditions of our time to the Southern parts, from whence in this and former ages the English have returned with renowned victory, yet exceedingly oppressed with extreme and penurious sickness, that hath much more prevented the proceeding and performing of their pretensions than the power of enemies." We have only to glance through the centuries to find how often history has been repeated and how true is Whetstone's saying even as applied to our own times. Though a war to involve this country may seem far distant and we hope will never occur again, it will be our duty to leave nothing to chance and to safeguard future generations by all the means in our power. I venture to think that in this direction the War Section of the Royal Society of Medicine will exert a great and lasting influence.

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[1] C. Singer, "The Cures of the Diseased in Forraine Attempts of the English Nation, London, 1598," p. 26.

DISCUSSION.

General Sir JOHN GOODWIN: Ladies and Gentlemen,—I would ask you to join with me in according a very sincere and hearty vote of thanks to Sir Robert Hill for the extremely interesting address to which we have just listened with so much pleasure. Most of us, in our younger days, read the accounts of naval actions and sea warfare by Captain Marryat. The horrors of a naval engagement in those days pale into insignificance when compared with the concomitants of modern warfare—high explosives, cordite flashes, torpedoes, &c. What must impress one greatly is that whereas in those days casualties may have been, and doubtless were, less appalling in their individual severity yet owing to the lack of anæsthetics and modern appliances the relief to suffering which could be afforded must have been infinitely less than is nowadays, owing to the advance in surgical science, possible. An interesting point, brought forward by Sir Robert Hill, is the fact that gas shells were not used by the enemy in sea warfare during any of the recent naval engagements. These gas shells formed a powerful and formidable weapon in our land engagements, and it is a matter of relief to know that the Navy was, at all events, spared one of the horrors of war. Sir Robert Hill's address, and the figures as to casualties with which he has furnished us, have been of the very greatest interest to me, and, I feel, also to all of you. I have very great pleasure in proposing a very hearty vote of thanks to him.

Sir JOHN BLAND-SUTTON: It is a great pleasure to have the opportunity of seconding the vote of thanks, for Sir Robert Hill is an old friend and fellow-student. Much criticism has been aroused in recent years by the splitting process which had prevailed in the surgical half of the pre-Amalgamation Society; since the famous amalgamation, division has prevailed, Orthopædics and Proctology have become sub-sections, and now it is proposed that Urology should become an independent section, by subtraction: now fortunately the War Section has become an addition, for in after-dinner speeches before the war we glibly talked of and drank to the Navy, Army and Auxiliary Forces:

in this new Section all these are combined. The war has so cemented them, and with them the civilian medical men who co-operated with the Imperial Forces and performed such useful work during the war. This noble Home of the Royal Society of Medicine is a fitting place for such an important Section. Surely it is an auspicious beginning that the Section should have been established during the distinguished Presidency of Sir Humphry Rolleston, a Rear-Admiral, with the assistance of one with a genius for organization like Sir John MacAlister, and an Inaugural Address from its first President, Rear-Admiral Sir Robert Hill. Hercules from his foot, and so with Sir Robert Hill; when we dissected at the same tables my friend also found opportunity to distinguish himself at sports, and the same vigour which he applied to the football has enabled him to rise to the summit of his profession with honour, distinction, and the affection of his brother officers. It has been well said that as a rule naval men write little and the little writing done by them is forgotten in books of travel, in State papers, or buried in archives in the vaults of the Admiralty. Seamen, we know, are powerful in expletives, but some possess the power of literary expression. We had an opportunity of proving this when Commander Horace Stephen lectured, to a select few, on the Limitations of Naval Surgery in Action. His powerful description of the effects of the flash produced by the ignition of a large quantity of cordite on a battleship made a great impression. The flash passed along the covered ways killing the men, and some, so suddenly, that they remained upright though dead. The description immediately recalled to my mind the seventh plague of Egypt in which reference is made to the "thunder and hail and the fire ran along upon the ground." The post of surgeon in the Navy teaches the young man to be resourceful, self-reliant and confident. The youngest man may be called upon to deal with a serious accident, or to deal with an uncomfortable disorder in an officer of high rank which may try his nerve. Lord Kitchener, during the war, crossed to France in a destroyer. He was a bad sailor, and asked the surgeon, a very young officer, recently appointed, for some bromide. The officer was somewhat bewildered as to the dose and after some anxious consideration gave Lord Kitchener 15 gr., who, in a little while, went to sleep, more probably from fatigue than from the salt. Then the surgeon's anxiety was great for fear he had given him 5 gr. too much! His description of the way he listened to the great soldier's breathing and snoring to assure himself that he was alive was amusing, and his joy was great when entering harbour to find him awake and refreshed by the journey. In the Navy men learn to think and act for themselves, and in this new Section we may hope to hear accounts of their actions and resourcefulness on common ground where they can be discussed in a friendly spirit. The Section had a great future. In seconding the vote of thanks the seconder wished Sir Robert Hill all success during his presidency.

War Section.

President—Surgeon Rear-Admiral Sir ROBERT HILL, K.C.M.G., C.B., C.V.O., R.N.

Prevention of Disease on Active Service.[1]

By General Sir JOHN GOODWIN, K.C.B., C.M.G., D.S.O.

THE Royal Society of Medicine has done me the honour of asking me to read a paper. After reviewing in my mind the many and diverse subjects on which I might speak, I thought that it might possibly be of interest if I addressed you on the subject of prevention of disease on active service, that being a problem upon which the Army Medical Service has been very closely engaged during the last five years. At the same time I would point out that this subject is so vast and so important that it will be impossible for me to do more, in the brief time at my disposal, than deal very superficially with the question.

Some fourteen years ago I had the honour of reading a paper on this subject at the Royal United Service Institute, and a few weeks ago, when sorting a mass of old documents, I came across a copy of this paper, the existence of which I had almost forgotten. On re-reading it I noticed that at the commencement of that paper I said: "The future success of an army in the field must, and will, to an enormous extent, depend on the efficiency with which measures for the prevention of disease are carried out." In the light of our present day knowledge and experience we might think that that statement was a truism which it was quite needless to state, but I would remind you that fourteen years ago it was by no means so generally accepted as it is now. Matters have changed greatly for the better since then, and I believe that it is largely owing to the unceasing and unwearying efforts in the direction of education and training in army hygiene for the ten years preceding the war that our success has resulted.

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[1] At a meeting of the Section, held December 8, 1919.

I propose to review, very briefly, some of the various communicable diseases which have threatened our Armies in the recent war, and the means which have been adopted towards meeting them.

THE TYPHOID GROUP OF DISEASES.

This group has, of course, always engaged our very close attention. When we consider the history of past wars, we may easily realize that the typhoid group of diseases must always loom in the foreground as an ever-present danger, a possible factor for great evil, if not, indeed, for failure and disaster.

The following are a few statistics regarding enteric fever in former wars:—

Franco-German War.—German Army: 72,393 cases of enteric, with 6,965 deaths. Number of German troops which crossed the frontier was 1,146,000.

Russo-Turkish War.—Army of the Caucasus: 24,475 cases and 8,900 deaths in a strength of 246,000.

Spanish-American War in Cuba.—Americans: 20,738 cases, with 1,810 deaths, in a strength of 107,973. Admissions 88·5 per 1,000 in 1898.

Boer War.—British Forces: 57,684 cases, with 8,022 deaths. Total strength, 530,000; average strength, 208,226.

Russo-Japanese War.—17,033 cases on the Russian side.

French Operations in Tunis.—4,200 cases, with 1,039 deaths, in a strength of 20,000.

These are grievous and alarming statistics. Now let us consider a few figures regarding the recent war. Firstly, as regards the French army:—

	Cases	Deaths
First three months of the war	8,191 ...	3,076
November 1, 1914, to October 31, 1915 ...	87,618 ...	8,614
November 1, 1915, to October 31, 1916 ...	17,656 ...	700

Inoculation was not made compulsory in the French Army until March 28, 1914, and only about 125,000 men had been inoculated at the time of mobilization. If incidence and mortality had remained what they were at the end of 1914 the number of cases would have exceeded 1,000,000 and deaths 145,000.

Now, as regards the British Armies in France. From the commencement of the war until December 31, 1918, we had 7,423 cases, with 266 deaths, in an average strength of 1,200,000.

I think you will agree with me that these are satisfactory figures, and show a very great improvement over those of any previous campaign. If you ask me to what I ascribe this improvement I would reply that, in my opinion, it is due to the advance of our scientific knowledge regarding this and kindred diseases, to the immense strides which have been made regarding water purification, disposal of waste products and field sanitation generally, to the improved education in hygiene of the Army as a whole, and last, but not least, to the increase in preventive inoculation.

As regards typhoid and para-typhoid vaccine, during the five years of the war 25,068,271 c.c. of vaccine were issued. We owe a deep debt of gratitude to the Inoculation Department of St. Mary's Hospital, which, during the first six months of the war supplied us with 1½ million c.c. of typhoid vaccine.

I said just now that our knowledge and our methods had advanced during recent years. I think that one index to this advance is furnished by the fact that the vaccine department of our Royal Army Medical College—a college which only came into existence after the South African War—has made and issued during the last five years enormous quantities of vaccines, of which I might mention the following:—

	Cubic centimetres
Typhoid and paratyphoid	23,202,789
Cholera	8,507,749
Pneumococcus	84,188
Dysentery	99,225
Influenza	1,806,325

There are some things, however, the value of which cannot be expressed by figures or statistics, and yet which have a power and produce an effect which are beyond calculation, and amongst these I would say that I believe that the instruction of our officers in hygiene and pathology, which has been carried out at the Royal Army Medical College since its institution has had an effect throughout the whole Army which has been of incalculable, though possibly intangible value.

You may notice that I have not made reference to the comparative ratio between deaths from disease and deaths from wounds. As you know, in previous campaigns the deaths from disease have enormously preponderated, but I have purposely omitted to mention the comparative ratios in the recent war—satisfactory though these are from a hygienic standpoint—because I think that perhaps we might be prone to fail to realize the enormous influence which has been exercised by modern weapons of warfare in raising the ratio of deaths from wounds as compared with deaths from disease.

Dysentery.

We know from our histories of previous campaigns that this disease has always been a scourge of armies in the field, and has also been a danger to civilian populations owing to the distribution of infection by "carriers" of the disease. Epidemics have usually occurred in association with rapid and widespread movements of troops. In the recent war it has occurred on different army fronts during periods of great military activity, and when troops first came in contact with defective sanitation amongst Eastern populations.

The disease first made its appearance in France in 1916, and the greatest incidence occurred in August and September of the years 1916, 1917 and 1918. Elaborate arrangements were made for early and accurate diagnosis. Specialist pathologists and well equipped laboratories were established, and arrangements were made for treatment at certain dysentery centres by specially qualified officers. All suspected cases were at once taken out of the line and kept under observation.

As regards *preventive* measures, I may sum them up as follows:—

(1) Precise diagnosis.
(2) Control of cases and "carriers."
(3) Isolation of all suspects.
(4) Careful sanitary supervision and control.
(5) Care of food supplies and food handling.
(6) Education as to personal cleanliness.

In the future I hope that we may add inoculation.

In the United Kingdom we have endeavoured to protect the civil population by the provision of special hospitals, laboratories, pathologists and clinicians for the diagnosis, control and treatment of all cases and carriers arriving in this country. I think we may regard the above measures as efficacious when we consider the fact that we had only 132 deaths (the great majority of which occurred in those suffering from a Shiga infection) from the disease in France; while as regards this country although we have historic evidence of epidemics associated with the return of infected soldiers from seats of war, and although in the present instance the return of cases and carriers is beyond all previous record, yet there have only been one or two isolated domestic occurrences of the disease which might be attributed to imported infection.

Malaria.

As in previous wars, so in our recent campaigns, malaria has proved to be a terrible source of loss of strength to our Armies. Consider the extensive and long-occupied army fronts of Macedonia, Palestine, Mesopotamia, East Africa, Egypt and Italy during the years 1915 to 1918, and you will realize, as the medical authorities did, the inevitable results of campaigning in those areas for at least six months of the year. During those months changes of area of occupation meant rapid and continuous, and, in some areas, massive infection owing to continued exposure to the bites of mosquitoes.

Measures of Control in the Field.—The problems of quinine production, supply and distribution were controlled by international committees, and, in spite of the enormous demand, distribution was so well controlled that almost at no time on any front did serious shortage occur. Specialists in malaria were provided, and laboratories for diagnosis and research, special hospitals, camps and bases—e.g., at Malta, to which cases might be removed and treated efficiently without the chance of continued infection. One can hardly do more than enumerate some of the many measures which were undertaken in order to combat the incidence of this disease. Screened buildings in mosquito-infested areas, mosquito nets for tents, hospitals and billets, veils and netting to protect the soldiers when exposed on duty, culicifuges, ointments, lotions, &c. All these measures demanded initiation, administration and execution to an extent never before called for. Sanitary operations were conducted continuously, swamps were drained, streams and ditches drained, channelled and oiled; isolation and screening of native populations in villages in proximity to camps were undertaken, and the people as well as the soldiers were educated in the facts as to the causes and prevention of malaria.

We have certainly vastly increased our knowledge as to the varieties and habits of mosquitoes in different parts of the world as well as of conditions of temperature and other influences affecting the occurrence and malignancy of malaria. From the end of 1916 to the present time cases of malaria, potentially infective, have been returning to this country, and measures were taken to protect the civilian population and to prevent indigenous infection amongst our troops. Consultants in malaria, with appropriate staffs, were appointed to ensure adequate measures and efficient control. Special hospitals, with experienced medical officers, were established for malaria cases. In conjunction with the Local Government Board, measures of defence in camps were undertaken by the military authorities with a view to preventing the spread of infection to the civil population, especially in such areas as had been notoriously subject to ague or malaria in the past. Concentration camps were established in order to increase control and treatment, to limit the distribution of infective cases and to ensure that adequate treatment was given over a certain period.

I think that our information to date warrants our saying that there has been a creditably small infection in this country, smaller than had been anticipated, and I believe that the continuance for the next year or two of this preventive work will prevent any serious outbreak of malaria in England.

Time does not permit me to enter into a discussion of cholera, typhus, plague, tetanus or other diseases with which we have had to deal, suffice it to say that each one has appeared on the horizon, has threatened ominously, has been met and, I believe, has been combated satisfactorily.

Food and Clothing.

One of the main objects for which the Army Medical Service exists is in order that an army may be maintained in such condition that it may successfully gain ascendancy over the enemy. Of the actual fighting qualities of our Army I shall say nothing, they are well known to all of you, but the greatest optimist could not expect an army to retain its fighting capabilities if it were decimated by disease. It consequently behoves us to see that our Army is adequately protected.

An army, if it is to remain an efficient fighting force, must be properly clothed, well fed, supplied with pure water, and protected from disease. As to feeding and clothing, I can only say that I do not believe that ever, in the history of the world, has an army been so well fed and so well clothed as our Army has been during the recent war, and I would ask you to bear in mind that it has been fighting in every variety of climate, from the tropics to the sub-arctic regions, and under every possible condition. For this feeding and clothing we have to thank the Quartermaster-General's Department, with which the medical directorate is in the closest touch on all these questions.

PREVENTIVE MEASURES AND DISCIPLINE.

Now, as to the measures which are necessary for the prevention of disease, here I find myself confronted with such a colossal subject that I feel I can hardly enter upon it. I could talk for hours on the measures which have been adopted during the recent war for the ensurement of adequate conservancy, for the provision of a pure water supply, for the inoculation of troops against enteric, cholera, plague, &c., for the action taken to meet tetanus and gas gangrene, both of which seriously threatened us at various times, but I feel that, in the limited time, I cannot attempt to enter into details, but must confine myself to the broader aspects of the question.

There are two great principles which, to my mind, underlie and govern disease prevention in the Army, and these are discipline and education. A short time ago I was reading Ludendorff's "Memoirs." He says: "Discipline is the only basis on which an army can be trained for war; discipline is not intended to kill character but to develop it. The purpose of discipline is to bring about uniformity in co-operating for a common goal." Quite so, but discipline will be immensely facilitated and strengthened if officers and men are so educated and instructed as to understand the reason of measures which are ordered to be carried out, the "why and wherefore" of those hygienic recommendations which are insisted upon by the medical authorities.

I apologize for again quoting from my lecture of many years ago as follows: "While fully recognizing that it is the duty of medical officers to make recommendations, and to do all in their power to ensure that every possible measure for the prevention of disease is carried out, I would point out that, unless regimental and staff officers are in sympathy with such recommendations; and unless both they and also the non-commissioned officers and men under them, understand the reason of the preventive measures, recognize their importance, and endeavour efficiently and intelligently to carry them out, it is extremely improbable that a successful result will be obtained."

THE REGIMENTAL MEDICAL OFFICER.

I still hold that view, and consequently I am convinced that we must persistently, unceasingly and unwearyingly carry on the work of educating all ranks of the Army in this most important subject.

There must be mutual understanding and co-operation, we medical men must understand the difficulties and exigencies of active service and the military necessities of every campaign, and we must be able to show that our recommendations and measures are sound and practical, and that they will conduce to the efficiency of the Army and to the final successful outcome of the war. This must be done, not only by the heads at the War Office, but also by every medical officer in the force; each individual must be constantly thinking, striving and educating. I have, I am sorry to say, heard it said that a regimental medical officer had an idle life; I have even heard it said that his work did not occupy him for more than an hour or so daily. Regarding this, I cannot do better than quote Sir Wilmot Herringham who, in his book, "A Physician in France," says: "I have heard that some regimental medical officers complained that their work took them only half an hour a day, and others that it was not important enough to require a qualified doctor. I have no hesitation whatever in saying that such officers did not know or do their duty." I entirely and absolutely agree with Sir Wilmot, and most cordially endorse his dictum. I think I may speak as one having knowledge for I have myself been "through the mill" and have in my younger days been a regimental medical officer on active service.

In later years, when inspecting medical arrangements, I have been very much impressed with the varying qualities of regimental medical officers. To take one example, which is I am glad to say typical of the majority—the regiment is in good form, the medical officer is popular, he is consulted by everyone from the Colonel downwards, the inoculation percentage is very high, the sick rate is very low, all sanitary recommendations are carried out, conservancy is as nearly as possible perfect. Ask this medical officer if he finds his life idle, and he will reply "No, the day is not long enough to get through the work." Now take the exceptional case, that of the medical officer who finishes his work in half an hour. Ask a dozen questions—"What is your enteric inoculation percentage?" "What have you done in the way of training your regimental sanitary detachment?" "How is your water purified?" "What teaching are you carrying out to officers and men regarding disease prevention?" Then go around the regimental lines, visit the kitchens, dining rooms or tents, the conservancy arrangements, &c., and I should be very much astonished if, at the end of a couple of hours, that regimental medical officer was not extremely crest-fallen, extremely conscious of his shortcomings in the past, and extremely anxious to make amends in the future.

The Soldier and his Health.

My experience of the British soldier is that he is very anxious to learn, and always ready to take an intelligent interest in the why and wherefore of all rules concerning his health. He may not be able fully to grasp scientific details, but if he even understands main principles something has been accomplished. I remember many years ago a medical officer gave a lecture on the prevention of enteric fever to a roomful of non-commissioned officers and men. A general officer happened to come round; he was greatly interested and, on the conclusion of the lecture, he said: "Now I want to see if the men understand what you have told them, so I shall call a man up on the platform to tell us what he has learned." The General looked around the audience and every man endeavoured to avoid catching his eye. One unlucky wight was, however, selected, and unwillingly mounted the platform. After considerable throat-clearing and hesitation he said: "Well, there's things called germs, they live in water and other places, and these 'ere flies carries them about and spits them on your food. If you get one of those germs inside you you're a 'case,' and if you get more than one it's likely you'll be for the box, so you've got to look out." This caused a good deal of amusement but, personally, I think that the man, who had probably never heard a lecture of the sort before, had grasped a good deal of valuable knowledge and had expressed it very pithily.

A Tribute to Civil Health Officials.

I cannot over-emphasize the importance of education in hygiene, not only in the Army, but also in the civil community, and I should like here to pay a tribute to the many members of the civil Public Health Service who held commissions in the R.A.M.C. during the War. As far as possible they were employed in the Sanitary Service of the Army at home and overseas. In almost every case the post of D.A.D.M.S (Sanitation) at Army Headquarters was filled by one of their number, others of them were posted as sanitary staff officers for the important health duties at bases and ports. Many of them were detailed as bacteriologists to hospitals, others were in charge of base or those invaluable units the *mobile laboratories*, others again were posted to fever hospitals or to control and supervise water supplies. Perhaps they were most in evidence as officers commanding sanitary sections.

Both in the early days of the war when attached to divisions, and later when allocated to fixed areas, these sections were appreciated by all arms of the Service who realized how much their work contributed to the health, comfort and well-being of the whole Army. Not only most of the officers, but a large percentage of the non-commissioned officers and men of these sections were drawn from the staffs of municipal health departments, and undoubtedly by precept and practice they did much to create a real sanitary conscience among the troops. Indeed it is reported that demobilized soldiers are taking back into civil life the lessons taught by sanitary sections and that some of the ingenious devices of army sanitation have been introduced into villages and small communities with excellent results. The example of the sanitary sections was not the only educational influence of civil health officials on military hygiene. Each army had its school of sanitation with a civil health official as O.C., and with sanitary engineers, sanitary inspectors and craftsmen as instructors. To these schools were sent for courses in army sanitation the medical officers of units, combatant officers, non-commissioned officers and other ranks, so that the effect of the teaching reached every formation in the War Zone. In connexion with the schools were exhibitions where could be seen models of every sort of device for securing efficient sanitation in the area where that particular army was operating. Also attached to the schools were workshops where men detailed from units were quickly taught simple methods for the construction of fly-proof latrines, meat safes, urinals, ablution benches, incinerators and other appliances for camp, trench and billet.

It should be remembered in connexion with these schools that each had to adapt its teaching to the special requirements of the country where it was established. Thus, methods suitable for France and Flanders might be ineffective in Salonica, Gallipoli, Palestine, Mesopotamia, Egypt or in East Africa, where the conditions were so very different. Some of the civil health officials held diplomas in tropical hygiene, but others without that qualification were quick to adapt their methods to meet the special problems to be dealt with, whether in South Eastern Europe, Asia or Africa. The resource that overcame the difficulties of tropical conditions was equally in evidence in dealing with sanitation in the Sub-Arctic areas of Siberia and Northern Russia. Again though most of their work was adapted to field operations, civil health officers serving in the Army have left behind them in such famous cities as Jerusalem and Baghdad improved standards of sanitation and permanent works, which will remain as a monument to their skill and energy for many years to come.

It was not possible for every medical officer of health to abandon his post at home, but those remaining in civil life did much to promote and safeguard the health of the troops in training. In all parts of the United Kingdom, county, borough, and district, medical officers of health have readily assisted the Army in regard to billets, camp sites, water supplies and other matters where their local knowledge was invaluable. In the industrial areas they have been equally active in regard to the health of munition workers. The importance of preventive measures in connexion with infectious and contagious diseases at a time when troops and civilians were in constant movement from one part of the country to another, need not be emphasized.

Command Conditions in the Army.

We labour under a difficulty in the Army, especially a hastily organized Army such as our recent one, and that is that enormous numbers of men, many of whom have previously lived in sparsely populated districts are suddenly brought under communal conditions and live in close proximity to each other. Such men are manifestly especially prone to contract communicable disease. We have, however, the immense advantages of facilities as regards education and discipline; having proved that our measures and recommendations are sound we can teach these principles, and we can then employ disciplinary means to ensure their enaction. As you know, enteric inoculation is voluntary and not compulsory in our Army. I well remember how, in 1914, our endeavours to make the men understand the advisability of inoculation, and to persuade them to undergo the process were invariably met by the production of the page of a certain daily journal which strongly advised against inoculation. I think it says something for the persuasive powers of our eloquence and for the intelligence of the British soldier that we were able to overcome this most pernicious advice, and that 96 per cent. of our Army were inoculated against the disease.

Water Supply and Water Purification.

There are many questions such as the details of army sanitary organization, sanitary sections, *mobile laboratories*, conservancy, &c., into which I should like to enter did time permit. I think, however, that it might be of interest if I very briefly discussed the question of water supply and water purification, these being points of such vital importance to armies in the field.

The provision of a pure water supply to troops is of paramount importance with regard to the prevention of those intestinal diseases which have proved so fatal to armies in the past. In most theatres of war, the sources of water have mainly been shallow wells, generally grossly polluted. Our practice has been to regard *all* water as probably contaminated and to deal with it accordingly, and this attitude has been justified in all theatres of war by laboratory analysis. In exceptional instances municipal supplies or deep wells have been available, but the risk of contamination during collection and distribution justifies the army method of treatment of water even in these exceptional cases.

As the size of the armies increased, the question of the provision of an adequate water supply, especially during the summer months, became an urgent one, and resulted in our developing an extensive organization. This organization included the whole question of supply, the sinking of bore holes, the improvement of existing or damaged wells, and, the distribution by pipe lines leading to central "water points." These "water points" usually consisted of a series of large tanks placed close to main roads, and from them pure water was provided to the units in those areas. In many cases water was laid close up to, or even actually into, the trenches; in other cases the final distribution to the trenches was by hand carriage in petrol tins.

In Eastern theatres the semifinal stage of transport was carried out by mule or camel transport. In France difficulties arose—as may be readily imagined—during and immediately after an advance. In such instances many thousands of troops moved rapidly forward into areas where all supplies of water had been destroyed. The work carried out by the Royal Engineers in rapidly carrying forward the piped system was really amazing, but it was impossible even for the Royal Engineers to carry out the work with sufficient rapidity in all instances, and consequently certain new units were developed, namely, the water tank companies and water barges to meet the necessity.

The water tank companies comprised a number of motor lorries specially adapted and staffed, and these units were able rapidly to transport water from any convenient source to the troops by any available roads, and to deliver sterile water to the troops at the rate of, e.g., 1,200 gallons per hour. The water barges could carry water at the rate of 5,000 gallons per hour over the many available canals and rivers in Northern France and Belgium. In both instances the water was so treated as to render it free from protozoal, bacterial and chemically poisonous matters. The system of treatment was based on a primary alum and sand filtration followed by contact with chlorine gas for half an hour, and then dechlorination in order to remove objectionable taste before final delivery to the troops in a sterile condition.

For scientific control, both bacteriological and chemical, the various mobile laboratories were responsible, and routine examination of all waters in the war zones were carried out. For general use and for the information of medical officers of individual units the Horrocks test outfit was generally supplied, and proved both simple and efficacious.

To meet the case of the individual soldier who might be separated from his unit, tablets of acid sodium sulphate were supplied, these tablets being made up in such a strength as effectively to sterilize the contents of the quart water bottle carried by each soldier, these water bottles being lined with a special enamel.

You will understand from the above that a vast amount of thought and careful work was expended in the provision of a pure water supply to the troops, and to this work we must credit in no small measure the satisfactory absence of such diseases as would ordinarily be conveyed by a contaminated water supply.

As regards our sanitary organization, I think we may say that it has stood the crucial test of the recent war remarkably well, and it has recently undergone, and is still undergoing, certain modifications which will, I believe, render it even more efficient in the future.

Hygiene and Pathology Hand in Hand.

If the three desiderata of education, organization and discipline be carried out steadily and intelligently, we may, I hope, regard ourselves as leading in the methods of safeguarding our armies from disease. Education must be carried out steadily and unceasingly by every medical officer in the Army; army sanitary organization, for which we owe so much in the recent war to Sir William Horrocks, will be safe in the hands of his successors, and it should never be forgotten that hygiene and pathology must go hand in hand. Each is so closely connected with and dependent upon the other, that without the close co-operation of both our methods and organization would break down.

I should like most sincerely to acknowledge my indebtedness and my gratitude to those officers, more especially General Beveridge and

Colonels Harvey, Smales, and Cummins, who have furnished me with many of the data from which I have compiled this brief paper. I must apologize for its shortcomings and omissions but, as I stated at the commencement, the subject is so extensive that I found it somewhat difficult to deal with even the principal points in one brief paper.

DISCUSSION.

The PRESIDENT, after thanking Sir John Goodwin, said that the extraordinarily small amount of preventive disease in the Army in France was often a source of wonder and admiration to those who were serving in the Grand Fleet.

Sir HUMPHRY ROLLESTON, alluding to the elaborate precautions which were taken to prevent the spread of infectious disease, such as dysentery, from returned troops to the civil population, asked Sir John Goodwin if he could explain how it was that disease was not spread by the returning troops after the South African War?

Lieutenant-Colonel W. R. BATTYE, D.S.O., I.M.S.: Sir John Goodwin stated that he would not quote comparative figures of sick to wounded in this and former wars, because he thought we were liable to overlook the relatively larger number of casualties produced by the newer weapons of warfare in this last war. There is a popular book written by an American medical man, which has been widely read and favourably reviewed in some of our leading papers. In it the author states that whereas in former wars the proportion of sick to wounded was usually five or more to one, yet in this war this figure has been trebly reversed, and has been as one to fifteen! And this too in spite of the fact that the proportion of battle casualties to total troops engaged has been smaller in this than in previous wars. This is a startling statement, and I should like to know if Sir John Goodwin can throw any light on it. Sir John Goodwin also referred to the excellent work of civil medical officers of health and other civil sanitarians in the Army, and the effect that the sanitary work of these officers has had on the civil population in various places, and incidentally he mentioned Baghdad. As I have recently returned from Mesopotamia and have been closely connected as an administrative officer with the work done in the city of Baghdad and other parts of Mesopotamia, I can bear personal testimony to the excellent educational value of the Army's work on the civil population. The work in Baghdad has been largely organized by an officer who in peace time was a medical officer of health of a large county area in England, and now he is back in his old appointment. The work was organized on the lines of that in a large English town, and in some respects was, I think, actually in advance of that in English towns. Soon after Baghdad was first occupied by British troops and a sanitary section installed there, some of the leading priests of some of the large mosques made application for the sanitation of their mosques to be reported on and supervised by our sanitary officers. The request was acceded to, and from that day the British Tommy has access as a sanitary inspector to several of the mosques of Baghdad. To all who know the Mohammedan East, this will undoubtedly be recognized as a triumph, the credit for which is due to the Army, whose work Sir John Goodwin has been representing here to-night.

Colonel WILLIAM HUNTER spoke of the incidence of infectious disease, particularly typhoid and paratyphoid fevers among the troops in the Dardanelles, and gave illustrative statistics.

Major-General Sir WILLIAM MACPHERSON, in reply to a request from Sir John Goodwin, gave some statistics regarding the relative incidence of wounds and disease among the troops in France, and pointed out that the relation between the numbers of casualties from wounds and those due to disease depended upon the number of battles which were fought.

Sir JOHN GOODWIN, in replying to the various points raised in the discussion, and particularly to the point raised by Sir Humphry Rolleston, said that probably the length of the voyage from South Africa, which occupied some time, and the fact that men were retained for a longer period in hospital accounted for the non-spread of infectious disease after the South African War.

APPENDIX.

Notes on Casualties in Campaigns, supplied by Major-General Sir William Macpherson, K.C.M.G., C.B.

I do not know what American publication has been referred to but it appears to be hopelessly wrong, if it says that the deaths from wounds were three times less in this war than in any previous war in proportion to strength. I have just been reading a German publication (one of Bodart's, the Austrian, who has written pre-war books on losses in war) in which these very points are gone into. Bodart exaggerates the strength of the British, putting it down as 7,000,000 for combatants, whereas our highest strength at any time in all theatres of operations was only 2,834,690, and in France alone 2,044,627. Bodart's figures of casualties are much nearer the mark, and even with his exaggerated statement of strength in the field, he shows that *killed* is 15 per cent. of strength (it would be very much higher with the corrected lower strength), as compared with 10 per cent in the Russo-Japanese war, 6·7 per cent. in the Russo-Turkish, 3·6 per cent. in the Franco-German, 2·55 per cent. in the 1866 war, 5·45 per cent. in the Crimean war, 7·8 per cent. in the Napoleonic wars. Also he says that on an average of every day of the

1,532 days of the war, 6,046 deaths occurred, as compared with 1,950 in the Balkan wars of 1912-13, 319 in the Russo-Japanese, 930 in Franco-German, and 400 in the Crimea. (These figures are for all armies engaged, as are also the figures of percentage of killed to strength; not for British only.) The point the speaker, or his American authority, seems to have got hold of is that the percentage of *case-mortality* amongst the wounded in the last war was very low as compared with previous wars—i.e., 10 per cent. as compared with 39 per cent. in Crimea, 32 per cent. in the Russo-Turkish, 25 per cent. in Franco-German, and 19 per cent. in Russo-Japanese wars. The *maximum* strength in any one day in France was 2,044,627, including Colonials and Indians, but not labour units. The total killed or died of wounds to end of 1917 was 412,955. As the *maximum* strength greatly exceeds *average* strength, the proportion of killed and died of wounds, amounting to over *20 per cent.*, would be still higher if calculated on average strength, and is practically three times greater instead of three times less than in any previous war, except the Russo-Japanese War, where, according to Bodart, the percentage was ten.

War Section.

President—Surgeon Rear-Admiral Sir Robert Hill, K.C.M.G.,
C.B., C.V.O., R.N.

DISCUSSION ON GAS-POISONING.[1]

Sir Wilmot Herringham.

So much has been said and written on the subject of gas-poisoning, which has been of interest to us all in France—though, I suppose, not of the slightest interest to you, Sir, as your service did not suffer from it—that it would be ridiculous in me to try to give anything like a full exposition of the subject; you all know just as much about it as I do. What I propose, therefore, is to indicate briefly the people I see in this room who can give detailed information. I will content myself with running over the historical aspect, and what happened on the clinical side.

It was on April 23, 1915, that Bowlby and I, who were up at Bailleul, together, heard that a great disaster had overtaken the Army in the north of Poperinghe. There we found that the whole French Algerian or Colonial Corps had come through in a panic the night before, helter-skelter, and nobody knew where they had gone to, nor why they had fled. We went further and came to Vlamertinghe, where there was a Canadian Field Ambulance, and they could not tell us very much more, except to confirm the story. But they also told us the Canadians had turned out and had held the Germans off Ypres. In that Canadian Field Ambulance I came across three French Algerian soldiers, who talked very bad French. I made out, however, that while they were in the trenches looking at the Germans, they saw the

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[1] At a meeting of the Section, held February 9, 1920.

Germans come out and bring out some tubes, or something of that kind, pour some stuff on the ground, and set light to it, and that then great clouds of smoke came down upon them and choked them, and that the whole army had fled. The last item was strictly true; the whole Colonial Division had fled, and I believe that if the Germans had had men enough there to go through the gap, there would have been nothing to stop them between Langemarck and Dixmude. They evidently were not prepared for their own success, however, and so they did not do it. This, besides being our first introduction to cloud gas (drift gas) was my first introduction to lachrymatory gas, for while I was getting the history of this gas attack from the French soldiers there was brought to the dressing room next door a man whose clothes had a curious smell. He was wounded, and, accordingly, they took his clothes off and dressed his leg. In three or four minutes no one could stay in the dressing room, as everyone's eyes were watering badly, although the man had already been lying out four hours on a windy day. That was the first introduction I had, not only to chlorine, but also to xylyl bromide. This attack occurred at the French end and concerned the Frenchmen alone. Three days later, on April 26, the attack was renewed in our lines, and in that case the men were brought into Bailleul. I went up, and found perhaps six or seven hundred in the two hospitals, principally at No. 8, which was in the school building at the bottom of the slope. There they were lying, gasping for breath; it was a most dreadful sight, the most horrible sight I have ever seen, I think. Half of them were blue, and they were coughing and hawking, and bringing up a yellow, thin, frothy fluid. If they lay on their side it ran out of their mouths. Occasionally they vomited. I do not think there is any distress so bad as that of asphyxiation. The men were lying about in all conceivable attitudes, in agony, a sight which those who saw it will never forget. We tried various forms of treatment, but nothing did much good. We did not understand it at first, that is, I did not. I had never seen cyanosis which did not yield to the ordinary application of oxygen. We used the same method of giving the oxygen as we had done for pneumonia and suffocative bronchitis in the hospitals, namely by funnels fitted to the oxygen cylinder. This was not of the slightest use for these cases. From that I was led to think it must be not simply the cyanosis of reduced hæmoglobin, but that of some morbid change in the hæmoglobin, such as methæmoglobin. But I was wrong. McNee first, then Dr. Haldane, whom I drove up two days later, were able to assert it was nothing more than reduced hæmoglobin in the blood.

Soon afterwards we had the opportunity of seeing the results of post-mortem examination. In that, we found three main things: laryngitis, extreme œdema of the lungs, and emphysema—an emphysema which occupied not only the edges of the lungs and their surface, but also burst through the lung and appeared as bullæ under the pleura. I will not enter into the minute details. I think McNee is here, and he knows more about the post-mortem particulars and the minute morbid anatomy of the cases than anybody else who was out in France. In England, of course, experiments were set going at once to try to explain the pathology. And Dr. Haldane was able to show that at any rate the chief part, if not the whole, of the symptoms might be explained by the anoxæmia, due to the impossibility of getting oxygen into the blood through the great wall of œdema which this condition produced. I cannot help thinking that, in the early and very rapid collapse which we saw in some cases there must have been some further action on the heart. But Dr. Haldane is here, and he will be able to tell us what light was shed by the physiological experiments on the pathology.

Gradually we got to be able to divide these cases into three classes. The first consisted of what we called the grey cases; they were collapsed almost from the very first, and the pulse was very small. For those cases one could do nothing at all. Another class consisted of those who were very lightly affected, and who recovered of themselves; there was no particular need to do very much for them. But there was also an intermediate class, in which there was cyanosis but a good pulse. These, if neglected, might turn out to be very dangerous. For them our treatment consisted, at first, in using ammonium carbonate and other kinds of stimulants and emetics. But though vomiting did good when it was natural, we had to give up emetics owing to the exhaustion which the artificial vomiting caused. The great mainstay of our treatment was the administration of oxygen. Colonel Douglas is here, and he was our teacher in this treatment. He used to go all round the Army, instructing men how to treat these cases with oxygen. I remember his bringing down a sergeant to Merville, whom he caught almost at the moment he was gassed, and very badly gassed. He came down all the way with that man and dosed him with oxygen continuously. Undoubtedly he saved the man's life.

I would like to sketch for you one or two of the makeshift apparatus we used; later we had the device of Dr. Haldane, which superseded everything else. These acted very well as emergencies before we got the Haldane apparatus.

The gas attacks continued right through the summer, and we got to be more and more protected against them. The growth of this protection forms a very interesting chapter, which we shall perhaps be able to hear from Colonel Cummins, who was in charge of the administration of it from headquarters. I do not think it has ever been published, and I do not suppose many here know the whole of it. He will tell the processes by which we eventually reached a very efficient stage of protection.

During that summer and the following winter, the attacks were continued with phosgene, a very similar poison. Sometime during the winter of 1916 it was used in shells, and in the course of that year we first began to use it in projectors, a clever way of concentrating a very large amount of it on a given spot.

There was considerable controversy about one form of treatment in chlorine and phosgene cases, and that was bleeding. It was somewhat instructive. There was a certain amount of clinical evidence that it did good, and I was always pushing this; but you know how difficult it is to rely on clinical evidence of benefit from any sort of treatment. Experiments in England seemed to show that bleeding did not do good. It was only when a second set of experiments were carried out, two years after the first, that it was shown that bleeding had a good effect, and then we had rather more support for the bleeding. It was an interesting thing, because, although both sets of experiments were perfectly right, a little alteration in the method was sufficient to vary the verdict.

There was a second point about our treatment which was very important—and we learnt it from Dr. Haldane's teaching, as well as clinically—namely, the need to keep these cases at rest. A case did not seem to do badly if it could be kept at rest, but if moved about, if a man was allowed to walk from the trench, sometimes even if he sat up in the reception room, he was very liable to fall down dead when he had not been expected to die. The slight muscular exertion raised the oxygen demand above the supply.

Another interesting point was the development of the gas-treatment centre. I hope somebody is here who can tell us about that, because the record is well worth compiling and preserving. The idea was introduced by Colonel Skinner in the battle of the Somme, and it were much more developed later. What I saw at Ferfaye was a hut, a field-ambulance affair, with a little room, and a table on which the patient could be laid so as to give him oxygen if he was a phosgene case. There was another room containing a bath, and here his clothes were taken off, and he was scrubbed in the bath to get rid of the mustard if it was a mustard case. He was then put on a stretcher ready for transport to the nearest clearing station as soon as he could be moved. He was treated with warm drinks, and oxygen, and dressings for the eyes. Outside was an elaborate apparatus for treating the clothes by steam. As this apparatus broke down at the end of a week, there was a second form of treatment: sets of clothes lines on which the clothes were hung until the rain and wind had got the material out of them. This was run exceedingly well by an officer (Major Badenoch), who sent us excellent notes, and it was of the greatest possible use. Those things were dotted about all over the Front. I remember the panic which the gas attacks brought to General Headquarters: it was an entire surprise, and there had been no preparations made to meet it. Hence our state of mind was, to say the least, "jumpy." We were continually expecting that some fresh form of gas would be used. Then we got information that quebracho was to be used, and we searched vainly through the books to find what could be the poison of this drug. It turned out to be a red herring, though the information came from an apparently good source. I have since thought "quebracho" was a name which, like our word "tanks," was used to conceal the mustard gas which came later. Prisoners told us the Germans had been expecting to deliver this attack for three weeks, and that it had only been postponed because our offensive altered their intentions.

After I came back from the war I heard a curious story, which I think is true. My brother-in-law, with whom I live and who drove me about in France during the first six months of the war, told me recently that he knew a man who was a chemist to an industrial firm in the North of England, and every year or two went out to Germany to pick up what he could which might be of service in his chemistry. He was out in 1909 near Leipzig or Dresden. He and his friend were walking in the country when they smelt a funny smell. They decided to follow it up, and they did so until they came to a small hill, on which they could see a party of soldiers and sheep, and it seemed to them that some of these sheep were dead. However, they could not get very near and they were instantly warned off. They came home and did not think any more about it, neither did they say anything. I imagine this was a trial of gas for military purposes as far back as 1909.

We shall be glad if McNee can correlate the subsequent histories of these cases with what was found in the lungs, in the brain and other parts. It will be interesting to know if he has been able to follow up any of these cases. And we shall be glad to hear about other sequelæ, such as tachycardia and erythrocythæmia and nocturnal dyspnœa. We have heard a great deal of it from Cambridge, where cases have been treated by the continuous inhalation of oxygen.

On July 13, 1917, the gas was changed to another form—namely, mustard gas—dichlorethyl sulphide. I saw, when I went up there early, the first post-mortems, and when the attack was repeated about a fortnight later at Nieuport I was at the field ambulance when the cases were coming in. Chlorine and phosgene produced enormous œdema of the lungs. The first effect of the mustard was to inflame the eyes, though there was but little permanent injury to them. I saw very few ulcers of the cornea. The second result was a scorching of the air passages; and, thirdly, there was a scorching of the skin. The main dangerous result was the septic broncho-pneumonia produced by the sloughing of the mucous membrane of the trachea, which formed a hot-bed for every kind of infection. The patients died of broncho-pneumonia within ten to fourteen days. I think Dr. Douglas will be able to show you the entire difference between the mortality curves of phosgene as compared with mustard gas.

The military effect was considerable: that of mustard gas was far greater than that produced by phosgene; it was possible by mustard gas to render a position absolutely untenable for as long as a fortnight. One could not keep the mustard gas out: it went through everything, gaiters and clothing, and it was only by the continuous wearing of masks that it could be kept out of the lungs. The other parts of the body were exposed to it always unless one wore indiarubber clothes. In this case also the sequelæ will be of interest. I suppose there must be a good number of bronchitis cases due to it. I do not think there is much occasion to say very much about mustard gas, except so far as sequelæ and mortality are concerned. But there is one thing I would like to know, and perhaps someone here can tell me: Why did we not get mustard gas out there quicker than we did? We did not have enough to use effectively until the very last few months of the war.

[*Addendum.*—The German method of manufacture was known to us, but was so clumsy and prolonged that had we used it we should hardly have been able to produce the gas. We sought and eventually discovered a shorter method, but it took a long time to find it out.]

Dr. C. G. DOUGLAS.

I am anxious to direct your attention for a few minutes to some features of gas poisoning which cannot fail to be of interest when we are considering medical problems arising out of the War. Gas warfare, as we experienced it at the hands of the Germans, can conveniently be divided into three phases:—

(1) The period of cloud gas attacks, April, 1915, to August, 1916, i.e., operations conducted on a limited front by means of the liberation of large quantities of lethal gas (chlorine or phosgene) from cylinders. In addition to the liberation of cloud gas at intervals, fairly extensive use was made of shell containing simple lachrymator substances, such as xylyl bromide, with the idea of obtaining a harassing or neutralizing effect.

(2) The period of lethal gas shell, July, 1916, to July, 1917, i.e., shell containing such substances as phosgene, diphosgene, or chloropicrin, the purpose of which was to cause death or serious disablement, as well as a harassing effect, a smaller amount of simple lachrymator gas shell being used in addition.

(3) The mustard gas period, July, 1917, to the Armistice—the culmination of the German methods of gas warfare. In this period three groups of gas shell can be distinguished, usually named according to the marking on the shell: (*a*) Yellow Cross shell, containing mustard gas or dichlorethyl sulphide. (*b*) Blue Cross shell, containing irritant organic arsenic compounds. (*c*) Green Cross shell, containing lethal gases, e.g., phosgene, diphosgene, and the like.

Chlorine, phosgene, and other gases included under the term "acute lung irritants" are dangerous to life owing to the fact that they cause the rapid onset of acute pulmonary œdema. These gases are however easily stopped by an efficient respirator. If we except the early cloud gas attacks of April and May, 1915, when the troops were either unprotected or only partially protected by respirators, and limit ourselves to the six subsequent cloud attacks between December, 1915, and August, 1916, by which time effective defensive measures against gas were in operation, one finds that the number of casualties caused is by no means large compared with the total number of troops exposed to the cloud. As one would expect, by far the largest number of the casualties occurred in the front system of trenches, for at this point there was least chance of spreading warning of the liberation of gas in time for the men to get on their protective helmets before the cloud was upon them. The mortality amongst such casualties as did occur was on an average 24 per cent., a figure which is not surprising seeing that any man who was slow in assuming his helmet was likely to get a serious dose of the gas owing to the amount of the gas liberated and the high concentration of the gas in the cloud.

Though lachrymator shell had a considerable harassing and neutralizing effect, they did not give rise to many casualties owing to the transitory nature of the smarting and watering of the eyes which they caused. The death-rate from the poisonous action of these shell was nil.

"Lethal" gas shell were first used extensively at the beginning of the battle of the Somme. During the period July, 1916, to July, 1917, they caused but a small fraction of the total battle casualties, whilst the average mortality amongst the gas casualties was only 6 per cent. The low mortality, as compared to that found in cloud gas attacks, is no doubt to be ascribed to improvement in gas discipline and protective measures, as well as to the fact that gas shelling is not a very effective method of liberating a sufficient concentration of lethal gas with such rapidity as seriously to affect a man before he can get on his respirator.

So far as casualties are concerned the picture changes, however, with the introduction of mustard gas. As you are aware, the number of casualties caused by Yellow Cross shell was very considerable. This high casualty producing power is determined by the properties of mustard gas. The fact that it has no immediate irritant action on the eyes or respiratory passages, such as chlorine or phosgene has, hinders its recognition in low concentration, while its high boiling point causes it to persist on the ground for a number of days after a bombardment, and is apt to mislead men who subsequently enter such a contaminated area. Great though its casualty producing power may be, and during the period in which it was employed it caused not less than 80 per cent. of our total gas casualties, it does not give rise to a high mortality in troops well disciplined in gas defence. During the period July, 1917, to November, 1918, the average mortality amongst our gas shell casualties was only 2·5 per cent., and this mortality was mainly due to mustard gas, deaths caused by lethal gas shell forming but a small proportion of the whole.

Blue Cross shell were used by the Germans in the hope that the irritant properties of the chlor-arsine compounds that they contained would be sufficient to put the troops opposed to them temporarily out of action: they were not used primarily with the idea of killing or of creating serious casualties. Though they had some value as harassing agents, the number of men who became actual casualties was small, and such casualties were as a rule fit for duty again in a very short time; there were no deaths.

It is evident that the problem that confronts the medical officer when he is called upon to treat gas casualties in modern warfare differs greatly according to the character and the mode of action of the particular gas to which the casualty has been exposed. In the case of the acute lung irritant gases his immediate and most difficult task may be to tide the patient over the acute stage of pulmonary œdema during the first forty-eight hours: once the critical stage is passed recovery may be uneventful, though slow, without any grave complications. In the case of mustard gas, when death is mainly caused a day or two after exposure to the gas owing to bacterial infection of the damaged bronchial tubes with consequent bronchopneumonia, the mortality is low, and the medical officer has to cope with very large numbers of casualties, affected with lesions of varying severity of the eyes, skin, or respiratory passages, whose ultimate recovery is in the majority of cases certain. The main problem in a case like this is, at least from the military point of view, to cure such casualties and to render them fit for duty again in as short a time as possible, for the number of casualties caused may well be sufficient seriously to affect the man power of the fighting force. A somewhat similar problem has to be faced in the case of the simple irritant gases, e.g., lachrymators and chlor-arsines, where correct and rapid diagnosis and a due appreciation of the mildness of the case may be a matter of some difficulty at an early stage. The correct lines of treatment with a view to avoiding unnecessary wastage of the fighting strength can only be learnt by experience, but as information gradually accumulated during the war successive improvements of treatment led to a marked curtailment of the period of convalescence.

Dr. J. W. McNee.

During the war, my attention was mainly focused on the pathology and morbid anatomy of the various types of gas-poisoning, but since coming home I have been able to examine a certain number of men said to be suffering from the late effects of gassing. My experience of these, however, is not nearly so great as that of Colonel Soltau or Colonel Elliott.

I will first briefly refer to the main points in the pathology of the acute cases, and try to explain what late effects might be expected to arise as a result of the lesions. I will confine my remarks to chlorine, phosgene, and "mustard gas."

In the case of *chlorine*, the lesions were simple, being those brought about by a pure pulmonary irritant. No lesions could be found in any part of the body apart from the respiratory tract. The main lesions were those of intense disruptive emphysema, associated with pulmonary œdema. The emphysema was extreme, as Sir Wilmot Herringham has already indicated. The only subsequent complication of importance was that during the period of convalescence a certain amount of bronchitis was commonly found. Therefore, in considering the possible late effects of chlorine, the only points requiring attention are the emphysema and the bronchitis.

With regard to *phosgene*, the conditions were somewhat different. And there I am in a difficulty, for I have heard no adequate explanation of some of the remote signs of phosgene poisoning with which I am familiar. I have, for instance, never been able to get a statement whether phosgene is absorbed into the body, and if so, in what way. But phosgene did certainly give rise to lesions apart from those in the lungs. In the lungs, the results produced by phosgene were, in a way, closely similar to those of chlorine, namely, intense œdema and disruptive emphysema. These, however, were seldom so severe as with chlorine. It is the remote effects of phosgene poisoning on which I wish to lay particular stress, since they are apt to be forgotten. Clinically, it was obvious to those of us who saw many cases, that phosgene had, in some way I cannot explain, a very definite effect on the heart, such as was not seen with chlorine. Sir Wilmot Herringham has stated that a certain number of cases of chlorine poisoning were definitely grey, and that these cases did badly. This is perfectly true, but among the phosgene cases there were many more who were pallid and suffered from acute circulatory failure. Moreover, it was observed that cases of phosgene poisoning might walk back a considerable distance from the line while apparently well, and then die suddenly, an event which never occurred in cases of poisoning by chlorine. Other distal lesions in phosgene poisoning also require notice. There was one in the lung, namely, that in addition to the emphysema and œdema considerable thrombosis of pulmonary capillaries was frequently encountered. A similar capillary thrombosis was also seen, although rarely, in the capillaries of the glomeruli of the kidneys. In the brain, also, hyaline thrombi were found in the capillaries in a number of cases. This cerebral thrombosis brought about a remarkable condition, a specimen of which I have brought for exhibition. You will see a great number of small ring-shaped hæmorrhages scattered through the white matter of the brain. Whether such hæmorrhages might occur in the brain, and the patient recover, I cannot say.

In considering what sort of symptoms might be found as late effects of phosgene poisoning, the emphysema must first be thought of. We need not think so much of bronchitis, since in the early stages the mucous membrane of the bronchi remains intact. Other effects, chiefly cardiac, must be remembered, such as tachycardia, breathlessness, and so on. These would be partly due to the emphysema, but partly to some action on the cardiac muscle of which we have no accurate knowledge.

Turning now to *mustard gas*, the pathology was entirely different from that of the other pulmonary irritants. After a latent period—generally about a day—and this accounts for the absence of deaths on the first day in Colonel Douglas's curve—an intense inflammatory reaction was found in the mucous membrane of the trachea and larger bronchi, resulting in that membrane becoming completely necrosed. Following that, there poured out into the lumen of the trachea and bronchi a membranous layer of fibrin, very like the membrane in diphtheria, in which micro-organisms found a very suitable pabulum for their growth. The late conditions which might be expected to arise after mustard gas poisoning are chronic bronchial troubles dependent on the remarkable injury to the bronchial walls.

I might now mention some of the actual late effects I have seen in war pensioners who have been gassed. In the North during last October, when we had a severe fog, numbers of these pensioners developed severe acute bronchitis. Most of these men had suffered from mustard gas, but some cases of phosgene and chlorine poisoning were also included. All, evidently, had permanent damage to the bronchial mucous membrane. Then there is a group of cases, which has received much attention from French writers, where a so-called "pseudo-tuberculosis" has developed. In such cases the main symptoms are cough and copious expectoration, but tubercle bacilli can never be demonstrated in the sputum. By the aid of skiagrams, the French writers have concluded that the condition is due to fibrosis around the lumen of the bronchi. The physical signs—modified percussion, &c.—tend to confirm this view. Recently, in the out-patient department I have seen cases in pensioners in which the symptoms suggested that this condition existed, but nothing indicative of fibrosis could be found on X-ray examination. It is remarkable how such definite respiratory distress may be present in such cases with so few obvious physical signs.

I understand, from conversation with Colonel Elliott, that the total number of gas casualties who have become pensioners as a result of these late effects is comparatively small. But my opinion is that most of these men will be *permanent* pensioners, and that their lungs will never fully recover from the effects of the damage produced by the gas.

Colonel S. L. Cummins.

Sir Wilmot Herringham has suggested that I should speak of the work of the Anti-Gas Defensive Organization with which I was associated in France. To deal adequately with this subject would require much more time than is available, and so I shall confine myself to a few points referring to the general lines of organization of an anti-gas defensive service, and which deal with principles rather than details.

As soon as the first German offensive took place, Sir Alfred Keogh sent out Dr. Haldane and Professor Baker to discuss matters and put their special knowledge at our disposal. Both these gentlemen were of immense assistance both during the first days and throughout all the subsequent developments of gas warfare. Sir Alfred also sent out Professor Watson, a distinguished physicist, to advise on the use of sprays, which were thought, at that time, to be likely to be of value in neutralizing gas clouds. Professor Watson, now unhappily lost to science owing to his sad death from influenza, possessed just the qualities of deep scientific knowledge, energy, drive and determination that were wanted in the crisis with which we were confronted, and we were more than fortunate in being able to retain him for work in France.

It had been decided by the Commander-in-Chief to develop a service of offensive gas warfare under the command of Lieutenant-Colonel (now General) Foulkes, R.E. We pressed for the immediate formation of a separate defensive branch as well, and this was at once sanctioned. It fell to my lot as an officer of the staff of Sir Arthur

Sloggett, to organize the defensive branch, and, with the Director-General's consent, I undertook the administrative side of this work.

The defensive branch took shape under the Adjutant-General, Sir Nevil Macready, and the first step was to create at General Headquarters a central laboratory for scientific work in connexion with all questions, both defensive and also, where necessary, offensive, relating to gas warfare. Professor Watson, with the rank of Lieutenant-Colonel, was made Director of the Central Laboratory, and under him were placed Captain Mouat Jones and Captain Auld, both of them highly qualified chemists. Captain Douglas was added to the staff of the laboratory for physiological work and, later, Captain Haines from Woolwich Arsenal, for work in connexion with projectiles.

This team at the Central Laboratory, together with the Chemical Advisers of Armies, rendered magnificent services, and were instrumental in bringing anti-gas defence to the high state of efficiency which was ultimately attained. At the same time, under Sir William Horrocks, a great service of production of anti-gas appliances was developed in London, a fine team of chemists and physiologists being organized at the Royal Army Medical College, and factories for gas masks, respirators and other appliances being created with the help of the Ordnance Department and business men with the necessary knowledge and plant. Few people know what tremendous responsibility, financial, administrative and technical, was cheerfully accepted by Sir William Horrocks. Throughout the years that followed, he never failed us, and he accomplished miracles in producing, in the shortest possible time, the vast number of masks, helmets and respirators that were required by the armies in France. Now as to questions of principle. It was soon evident that anti-gas defence was *not* a medical service, *not* a service of the kind that should work under the Adjutant-General, but a branch of warfare affecting the integrity of the battle line; obviously a function of the General Staff. I remember with what pleasure I found that Lieutenant-Colonel Crossley, a well-known chemist who had been early associated with offensive gas investigation at home, held just the same views that I myself had come to hold on this question. Together, he and I placed the matter before Sir Nevil Macready, and he at once appreciated the weight of our arguments and made a strong recommendation on these lines to the Commander-in-Chief. The result was that the Gas Directorate was formed under the command of General

Thuillier, R.E., in which Colonel Foulkes, while retaining command of the Special Brigade, served as Assistant Director of Gas Services for Offence, while I was appointed Assistant Director for Defence. In this way a great service for gas warfare in all its branches came into existence, and its subsequent history is bound up with the general history of the war.

But while I hold strongly that gas warfare in its defensive as well as its offensive aspects must be a function of a special force of scientific combatant officers and men working under the General Staff, I hold equally strongly that the medical and physiological organization for dealing with gas cases and investigating the effects of gases must be under the Director-General of the Army Medical Services. The knowledge gained by such an organization is essential to the Director-General and also to the General Staff; to the one in order that everything possible may be done for gas casualties; to the other because it is necessary to know the effect of gases on man and animals when the question arises of which gas to use against the enemy. But, while the knowledge gained in such investigations is necessary to the General Staff, the fact that physiology is a branch of medical science makes our service the most suitable *milieu* for the development of physiological and biochemical investigations on the effects of war gases, and brings into prominence one more link between the Army Medical Service and the fighting services of the Army.

In the late war, the experimental work on the effects of gases was at first organized by a War Office Committee, of which Sir William Horrocks was Chairman, the Medical Research Committee, and a Committee of the Royal Society;[1] and a highly efficient team of physiologists and pathologists was organized, under the direction of Mr. J. Barcroft, at the War Department experimental ground at Porton.

Some such organization as this must continue to investigate the physiological side of chemical warfare in the future. There must be the closest touch between the Army Medical Service and the General Staff on this matter; no secrecy, no reservation, no jealousy. We must be completely "in the know" with reference to every new development in chemical weapons of offence, and we must train during times of peace the specialists that will be required in war.

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[1] These separate Committees finally unified their efforts by forming, from amongst their members, a single body, the Chemical Warfare Medical Committee.

Dr. Haldane.

Sir Wilmot Herringham gave us such a clear and graphic account of the early stages of the gas warfare, and the general medical features of it, that I will only venture on a few remarks on certain aspects of the question.

Sir Wilmot referred to the effects of bleeding, and to anoxæmia, and I would like to say a word or two on these subjects. With regard to the effects of bleeding, Sir Wilmot said it was very difficult to see, from mere clinical experience, whether remedies are doing good or not. I, as a physiologist, venture to differ: I trust the clinical evidence almost more than the physiological, and, in this case, I think the clinical evidence was very clear that, in certain cases, the bleeding of men did relieve the symptoms. The effects of bleeding were known to me before the war, through experience in cases of poisoning by nitrous oxide fumes. The indication for bleeding was, that the patient seemed to be bursting with blood. In the blue cases, in which the veins were distended, it was evident from the pulse and the distended veins in the neck that both arterial and venous pressures were high. When there is high venous pressure, we know there is very apt to be œdema of the lungs. That is not the primary cause in gas cases, but an important subsidiary cause. If, in an animal, you raise the venous pressure more than a little, œdema of the lungs ensues in a few minutes, as Knowlton and Starling showed experimentally. By bleeding you reduce the venous blood-pressure and reduce also the tendency to œdema, and relieve the strain on the heart. I believe that the only cases which are benefited by bleeding are the blue cases, in which the veins are distended. The bleeding I got to know about before the war, first of all through the experiences at the Rand mines of Dr. Macaulay and Dr. Irvine, on the excellent effects often produced in cases of poisoning by nitrous fumes, and also through a case Sir William Hale White told me of in this country, at Guy's Hospital, in which he had done the same thing, and the man was evidently, as a result, a great deal better. I think the clinical evidence was quite clear: I do not care how many animals were not benefited by the bleeding. And the physiology of it, I think, is also clear. At first, it is difficult to understand how a man or animal should appear to be so full of blood. I think the explanation is, that the want of oxygen, combined with excess of CO_2, produces a great vasopressor excitation, so that both arteries and small veins are contracted, although the capillaries between are full of blood. As Krogh has shown lately, the capillaries themselves actively dilate and contract, and apparently they dilate in response to the stimulus of want of oxygen and excess of CO_2. But the arterio- and veno-pressor excitation practically diminishes the total capacity of the vascular system, and causes the high venous pressure. It is these cases which are benefited by bleeding. I think the general experience was that bleeding in the grey cases was of no use, and that, anyhow, blood would not flow from the veins, so that no harm was done by attempts at bleeding.

I now come to speak of the effects of anoxæmia. I am very much impressed with this, because I have had so much experience of anoxæmia myself, in one form or another, in studying CO_2 poisoning and the effects of low atmospheric pressures in steel chambers and at high altitudes. I can speak from personal experience about the effects of even slight anoxæmia. If it is continued, the effects are extremely formidable, and no one wants to suffer from anything more unpleasant than mountain-sickness, which is produced by a comparatively slight anoxæmia. Serious anoxæmia is a dangerous condition.

This brings me to speak of the grey cases. The anoxæmia of the grey cases is far worse than that of the blue, because in the blue cases there is probably an excess of CO_2 in the blood, as well as a deficiency of oxygen, whereas in the grey cases it is the other way round. In the grey cases the breathing is not so much stimulated: mere anoxæmia without excess of CO_2 does not stimulate the breathing very much, and the dissociation curve of oxyhæmoglobin is altered, so that the hæmoglobin is holding on more tightly to the oxygen. The anoxæmia is thus much more dangerous and formidable in the pale cases, where there is not much blood in the capillaries, and no excess in the large veins, and where the supply of blood to the heart is probably diminished owing to the absence of CO_2. I think the anoxæmia accounts for what Dr. McNee referred to, the presence in various organs—the brain, &c.—of hæmorrhages. These are found in CO poisoning, and I think you would expect them in these grey cases, though we cannot yet speak dogmatically on the matter.

The presence of severe anoxæmia seems to be the main source of danger in these gas cases; and if by adding oxygen to the inspired air we can so increase the alveolar oxygen pressure as to enable sufficient oxygen to penetrate through the œdematous liquid to the blood, we hold death at bay until the lungs have time to recover.

Dr. J. A. Ryle.

I saw a large number of gas-poisoning cases of all types in the early, acute stages; furthermore in January of last year, having been sent to Cambridge to work with Dr. G. H. Hunt and Dr. C. M. Wilson, I was brought in contact with some of the late results of gassing. It is with these late results that we should, I think, be particularly concerned on account of the pension problems which they raise.

I would suggest a classification of the sequelæ of gas-poisoning into four groups. In the first group—fortunately a small proportion of the total—are the cases of gross pulmonary damage with gross physical signs, that is to say, chronic bronchitis, re-awakened tuberculosis, and other cases not specifically described except by the French, who called them "pseudo-tuberculosis," but which I prefer to regard as cases of bronchiectasis following "mustard-gas" poisoning. Hæmoptysis and chronic cough with purulent sputum are the usual features of this type of lesion. Dr. C. M. Wilson, who had much experience of "mustard gas" cases at Boulogne, showed clinically and at autopsy the occurrence of acute bronchiectasis following poisoning with "mustard gas." It is probable that a certain number of these cases survived the acute stage. Dr. G. Marshall, who is in charge of the tuberculosis department at Guy's, tells me that he has seen cases of this type, that he regards them as having bronchiectasis, and that he has seen them with clubbing of the fingers.

In the second group of cases there is also structural damage, but it is not so superficially obvious. These cases, for lack of gross signs, have often passed into the "D.A.H." group. On inquiry you will find a history of broncho-pneumonia or prolonged initial invalidism following gassing, and on careful examination you will find signs of emphysema, as evidenced by diminution or complete absence of præcordial dullness and the presence of apical bulging on forced expiration against resistance. Naturally they show a poor exercise tolerance.

In the third group I would place the "effort syndrome" cases with none of these physical signs of structural damage. I do not know if they are to be regarded as the result of any toxic or organic effect of gas. My impression is that a large number of "D.A.H." cases exist in which no history can be obtained of general infection and in which there is no definite evidence of focal infection or other factors such as have been described by Dr. Lewis as the more important factors in predisposing to or producing "D.A.H.," but in which the "D.A.H." has followed upon some crisis in the man's experience in France. That is to say, they followed upon prolonged fear under bombardment. And I think there is an exactly similar group following the fear of gas attacks—fear as bad as that caused by bombardments. Whether it is to be considered as a purely functional condition, or as some disturbance of the sympathetic nervous system, or of the endocrine glands, I do not know.

The fourth group is also a very important one, and it is a big one, and, I think, must have accounted for a considerable percentage of the cases of disability following gassing. It includes the functional conditions: blepharospasm, vomiting, aphonia, &c., which, in most of the cases, followed mustard-gas poisoning. This vomiting has often led to a diagnosis of chronic gastritis, and many of the men have been treated for gastric symptoms because of the repeated vomiting. In most of these cases I believe the condition to be functional. Dr. Hurst and his colleagues have come across such cases and have cured them by ordinary methods of re-education and persuasion. We had not at Cambridge much real opportunity for classing into groups, because we had so small a number of cases to deal with, but it occurred to me that some such classification as this might ultimately be adopted, as it is so important to recognize the various types of disability, for purposes of pension assessment.

Dr. T. I. Bennett.

I would like to ask one or two questions in regard to difficult problems which arose at the end of the war in connexion with gas-poisoning, as to which one has often wished to hear an explanation. This is the first opportunity I have had of asking.

The difficulties arose chiefly in connexion with the "Blue Cross" gas-poisoning, to which but little reference has been made to-night, yet they are the most interesting cases we had to do with in the later stages of the war. I should feel greatly indebted if Sir Wilmot Herringham, or some other speaker, could give us some information concerning the gastro-intestinal symptoms which we saw in connexion with the Blue Cross cases in the summer of 1918. I think there are here to-night several observers who will agree with me that at about the time we were told the Germans were using a slightly different compound—a cyanarsene compound, instead of the phenyl-arsene—we got cases closely resembling those of dysentery, except that perhaps they had more vomiting, sometimes with hæmatemesis, and a few days later there was marked pyrexia. We were never able to follow those very far: were any of the subsequent gastric symptoms in men who suffered from gas-poisoning due to an organic cause, namely, poisoning from Blue Cross gas?

Sir WILMOT HERRINGHAM (in reply).

There was very little known about the Blue Cross cases, and I do not remember having seen any cases with intestinal symptoms in the hospital from that kind of poisoning. The most interesting developments were the nervous: these were very curious, and were, at one time, even alarming. They were chiefly, however, of the nature of paralyses and anæsthesia, and I believe they quickly passed off, for the most part. There was some loss of reflexes. There were no sequelæ of Blue Cross gas-poisoning which did not pass off fairly soon: I do not think any man died, or was even seriously ill, from the effects of this gas.

Colonel Cummins has told us about the actual position in the organization of the anti-gas work during the war. That is a matter which it is difficult for us, civilians, to understand. And when he says it is a matter of general staff work, I sincerely hope he does not mean that scientific investigation would be in any way dependent upon the General Staff. It is not that their goodwill would be lacking, but their intelligence is lacking.

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War Section.

President—Surgeon Rear-Admiral Sir ROBERT HILL, K.C.M.G., C.B., C.V.O., R.N.

Scurvy: With Special Reference to Prophylaxis in the Royal Navy.[1]

By Surgeon Rear-Admiral BASSETT-SMITH, C.B., C.M.G., R.N.

IN bringing up the subject of scurvy for discussion, I know well that it is one which has been very fully investigated and ably treated by a large number of distinguished persons, but it is of particular interest to all those who have to maintain the health and well-being of our sailors.

In the days of the old sailing ships, with voyages of extended duration, the danger of scurvy was much greater than it is now, little was known of the ætiology of the disease, and catering difficulties were much more marked. Dr. Lind and Sir Gilbert Blane, the pioneers of naval hygiene, were both very active in studying and endeavouring to eradicate this scourge of the Navy. Fresh fruit and vegetables, as prophylactic and curative agents for scurvy, had been known to the Dutch as far back as 1564, and these had been successfully employed in the fleet by Sir Robert Hawkins in 1600, but the use of orange and lemon juice had been more or less forgotten until Dr. Lind insisted on their value. He, however, recommended *preserved* juice for use on long voyages. The fresh fruit juice was concentrated by *heat* to a thick syrup; as this was done in glazed earthenware vessels soluble lead salts were formed and Lind drew attention to this danger which he referred to as "Death in the Pot." Trotter strongly opposed the use of this form of preserved juice as it was said to undergo fermentation and become mouldy. Unfortunately its issue to the crews in Cook's famous voyage of 1773-74 did not prevent scurvy breaking out. It was Gilbert Blane's teaching and powerful influence which brought Lind's recommendation into official use. Sir Gilbert Blane in 1757 pointed out the importance of cleanliness, sufficient ventilation, and absence of damp in ships, and the need of a supply of lemon juice for the prevention of scurvy. It was, however, not until 1796, when Blane was one of the Commissioners of the Navy Board, that the use of lemon juice was ordered as an essential part of the naval dietary, and this quickly resulted in the practical disappearance of scurvy from the service afloat. In 1782, two-thirds of an ounce of lemon juice per man, *per diem*, protected the whole ship's company of H.M.S. *Suffolk* during a voyage of twenty-three weeks without touching port. In a discussion on the probable causes of the disease, which he attributed to the lack of a "certain element which is necessary for growth and repair," Blane foreshadowed the present deficiency theory.

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[1] At a meeting of the Section, held May 10, 1920.

From that time to the present, lime juice has been a standing ration but its efficacy has disappeared. The reason for this has been ably shown by Mrs. Alice Henderson Smith. As originally prepared the lime juice was made from sweet limes, *Citrus medica*, with lemons, imported chiefly from Spain. In 1793 war stopped these supplies, but in 1802 delivery was resumed and scurvy, which had obtained a temporary hold, was again almost eliminated. About 1860, by the development of the cultivation of limes in the West Indies, a large quantity was made available, and the contracts entered for the Navy caused these, the sour lime, *Citrus medica var. acida*, to supersede the sweet limes and lemons formerly in use, and for a time the new lime juice was believed to be better than the old. In Ross's Polar expedition the original lemon juice had been issued, but Sir G. Nares in 1875 was provided with the new West Indian lime juice. The general conditions of the two expeditions were very similar but while the former escaped scurvy, Sir George Nares's men suffered severely from it in spite of the regular issue of the lime juice, thus demonstrating the failure of the new preparation as a prophylactic. The sister ship in the latter expedition, the *Investigator*, was supplied with lemon juice, and for twenty-seven months after leaving home she had few cases, though the crew suffered great hardships. In three and a half years she had only three deaths from scurvy whereas Nares had the same number in one year.

The evidence is clear that lemon juice is far more effective than lime juice in preventing scurvy and this has been fully confirmed by the large number of laboratory experiments carried out at the Lister Institute, London, and elsewhere, which have determined exactly their relative values for the purpose. Lemon juice and sweet limes have therefore been found to repair a deficiency which, if not rectified, induces scurvy, and the consumption of these juices is curative for the same disease. This is in accordance with modern teaching which attributed the condition to a deficiency of a certain accessory factor known as scurvy vitamine. This factor is found in living vegetable (and secondarily in animal) tissue; in largest amount in fresh fruit, green vegetables, and growing pulses, and, to a less extent, in roots and tubers. Young vegetables contain more than old. It is present in small amount in fresh meat and milk. It has not been found in yeast, fats, dry cereals and pulses, but exists to a certain extent in canned tomatoes. The antiscurvy food factor is sensitive to high temperatures and if exposed to them it rapidly loses its efficacy; the presence of alkali is also detrimental.

Recent History.—Much has been written of the prevalence of scurvy, during and since the war, among the combatant forces and in the civil populations chiefly affected in war areas. Of the former those in Mesopotamia suffered very severely and Colonel Willcox, in his excellent communications,[1] has described the reasons for the outbreak very fully. The high incidence amongst the native troops at Kut, in comparison with that of the Europeans, is explained on dietary grounds; the latter had fresh meat to supplement their meagre diet and the former were more susceptible owing to diminished vitality from preventable causes. He states that the knowledge we then possessed for the prevention of scurvy and beri-beri was not fully appreciated and acted upon by those in authority, i.e., that the food must have the necessary accessory factors as well as sufficient calories as estimated by the protein, carbohydrates, and fat. The fact that over 11,000 cases of scurvy occurred in the last six months of 1916 indicates the importance of the subject. The following table shows the amount of scurvy in the Indian troops and beri-beri in the British :—

Year	Scurvy (Indian)	Beri-beri (British)
1916	11,445	104
1917	2,197	84
1918	825	51

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[1] See *Proceedings*, xiii (Sect. Therap.), p. 7.

The crews of British men-of-war were also affected to a small extent, and as has been shown repeatedly by the endemic presence in the Persian Gulf of ship beri-beri, this is a question of some importance, more particularly in times of extra strain as was the case in these years. Much has been done to prevent these outbreaks by increasing and supplementing the diet, but climatic conditions in that area always make the provision of efficient food supplies difficult.

In other war areas, Serbia and the remainder of the Balkans, famine conditions have necessarily given rise to an enormous number of scurvy cases. In Northern Russia it is very prevalent each winter, particularly in the prisons. Even on the Western Front the cases of incipient scurvy were very numerous, due in great part to the use of tinned provisions.

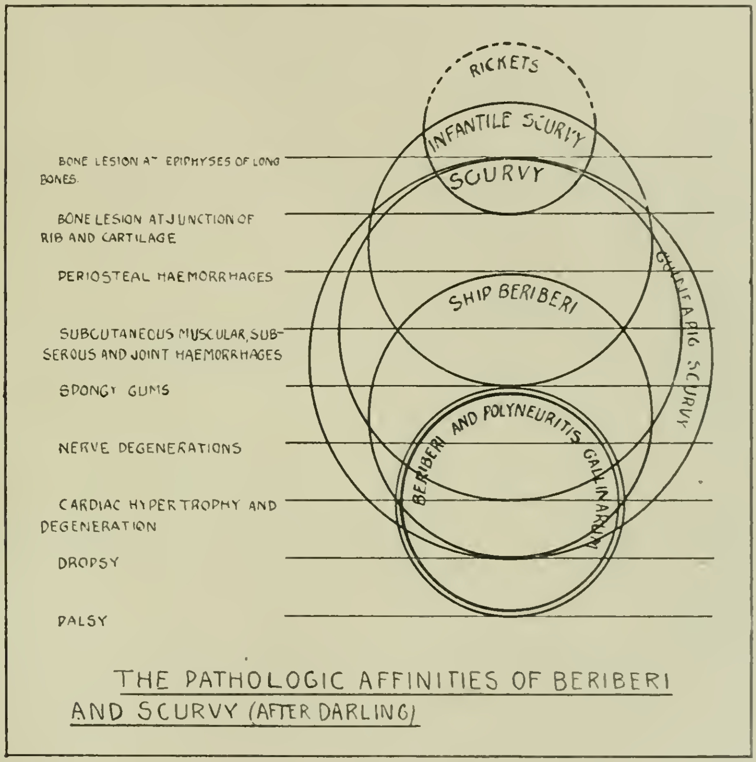
The post-war hardships in the civil populations have induced scurvy and the gallant fight being made by noble-minded individuals of all classes and peoples throws a glimmer of sunshine on what is otherwise a dark and tempestuous horizon. The work in Vienna, by Dr. Chick and others, to ameliorate the sufferings of the infantile population, is particularly noteworthy. From a scientific point of view such an overwhelming mass of cases has increased our knowledge of the clinical features of scurvy enormously and many new facts have been ascertained.

By Japanese investigators the conditions produced in guinea-pigs on a deficiency diet is considered to be similar to that found in ship beri-beri, a disease which undoubtedly links up human scurvy and beri-beri.

The ætiology, thanks to the work of British, American, French and other observers has now been placed on a sure foundation following the elaborate investigations that have, for years, been carried out in establishing the ætiology of beri-beri as a deficiency disease. The close relationship of beri-beri, ship beri-beri, scurvy, and rickets was well shown in a diagram by Darling in 1915 which I here reproduce; it shows the gradation from polyneuritis avium at one end to that of rickets at the other.

He recognized several varieties of beri-beri—infantile beri-beri, asylum beri-beri, wet-and-dry beri-beri (which Vedder and Clark look upon as distinct), the Brazilian type, endemic dropsy, and endemic peripheral neuritis. Darling, with Surgeon-General Gorgas, spent much time in investigating the endemic scurvy found among the miners in the Rand of South Africa. The points of similarity in the cardiac condition with that of beri-beri were often very marked in the concentric hypertrophy of the right heart and degeneration of the vagus nerves. The disease appeared to depend upon the following factors: Over-milled corn as a chief article of diet, over-cooked corn, over-boiled meat, insufficient vegetables.

Clinically, cases were seen of every degree of severity, from those who had spongy gums only to those with excessive hæmorrhages.


RICKETS
INFANTILE SCURVY
SCURVY
SHIP BERIBERI
GUINEA PIG SCURVY
BERIBERI AND POLYNEURITIS GALLINARUM
BONE LESION AT EPIPHYSES OF LONG BONES
BONE LESION AT JUNCTION OF RIB AND CARTILAGE
PERIOSTEAL HAEMORRHAGES
SUBCUTANEOUS MUSCULAR, SUBSEROUS AND JOINT HAEMORRHAGES
SPONGY GUMS
NERVE DEGENERATIONS
CARDIAC HYPERTROPHY AND DEGENERATION
DROPSY
PALSY

THE PATHOLOGIC AFFINITIES OF BERIBERI AND SCURVY (AFTER DARLING)

Fig. 1.

Some cases showed marked rickety symptoms with extreme destruction of the chondrocostal junctions, others were similar to beri-beri with cardiac degeneration and changes in the vagus nerves. Personal and racial factors had an unknown influence on the resultant symptoms: a deficiency diet in a tropical African negro miner causes severe scurvy symptoms and in a Cape Colony African labourer mild scurvy, a diet that in some African negroes may cause scurvy will in others produce neuritis. In all cases the knee-jerks were exaggerated. (Those who have experience in ship beri-beri have often been puzzled by the persistence of knee-jerks in these cases also.)

The Rand scurvy, besides showing the cardiac changes, differs from the ordinary forms found in time of poverty and famine in that it does not so readily yield to treatment. Darling sums up in the following words: "The striking excentric hypertrophy and dilation of the right heart, with extensive fatty degeneration of the muscle, and the severe degeneration of the vagus nerves, in cases of scurvy seen in the Rand, furnish new and additional facts which show the intimate relationship between scurvy and beri-beri as to ætiology. The affinities between these two diseases and certain other cachexias lend emphasis to the opinion that they are all the result of the continued use of a one-sided and deficient diet."

Table I.—Showing Affinities and Pathologic Features of Scurvy, Beri-beri, &c.

Bone lesions at epiphyses of long bones	Rickets	Infantile scurvy	*	Guinea-pig scurvy			
Bone lesions at junction of ribs and cartilage	Rickets	Infantile scurvy	Scurvy	Guinea-pig scurvy			
Subperiosteal hemorrhages		Infantile scurvy	Scurvy	Guinea pig scurvy			
Joint, subserous, subcutaneous and muscle hemorrhages		Infantile scurvy	Scurvy	Guinea-pig scurvy	Ship beri-beri		
Spongy gums		Infantile scurvy	Scurvy	Guinea-pig scurvy	Ship beri-beri		
Nerve degeneration			Scurvy	Guinea-pig scurvy	Ship beri-beri	Beri-beri	Polyneuritis gallinarum
Cardiac hypertrophy and degeneration			Scurvy	Guinea-pig scurvy	Ship beri-beri	Beri-beri	Polyneuritis gallinarum
Dropsy				Guinea-pig scurvy	Ship beri-beri	Beri-bêri	Polyneuritis gallinarum
Palsy						Beri-beri	Polyneuritis gallinarum

The necessary diet for the maintenance of health, besides sufficient protein, carbohydrates, fats, salts, &c., must contain the accessory food factors; the antineuritic water-soluble B; the fat-soluble A; and the antiscurvy factor. It is with the last that we are here concerned.

Recognizing that this cannot be supplied by the preserved lime juice from West Indian sources as at present made, it has been my object to provide an efficient, portable, and palatable substitute for the use of ships as occasion requires, and for such expeditions as may be undertaken in Polar exploration, &c. For the last six months I have been experimenting at the Royal Naval College, Greenwich, with guinea-pigs, which are extremely susceptible to scurvy, to obtain such an antiscorbutic preparation to replace or add to the lime juice ration. The value of these experiments with guinea-pigs in relation to human scurvy has received abundant proof by experiments, using monkeys, carried out on similar lines by Barnes and Hume, Chick, Hume and Skelton, and others.

Much of the work done at Greenwich has not been original but was necessary before passing on to the methods of prevention and cure. The use of dried orange juice has been largely employed by Givens and McClugage, who found that in the fresh state it was equal to that of lemons, and that it retained its antiscurvy properties for a considerable time, but in their preparation a certain amount of heat was employed which diminishes the efficiency of the product. In the investigation carried out at Greenwich various methods of preparing the juice were tried, evaporation at 60° C., de-acidification, impregnation of filter paper, rapid drying, &c.

The final laboratory method evolved, and which has been continuously in use is as follows: The juice of fresh lemons is roughly filtered through muslin and then through filter paper under reduced pressure by means of a water pump. The filtered juice is evaporated, *in vacuo* over sulphuric acid at ordinary temperature (13·5° to 15·5° C.). The residue of non-crystallizable syrup is worked up into as stiff a paste as possible with a mixture of anhydrous lactose 97 per cent., gum tragacanth 3 per cent. The paste is cut up into sections each containing the juice of half a lemon. These are rolled, faced with the mixture, and pressed to assume the lozenge form. These tablets have been stored at 15°, 30°, and 37° C. for months, and have been employed in the experiments. The average yield of juice from one lemon is about 48 c.c., and each tablet contains the equivalent of 24 c.c. of unfiltered juice. The whole process takes about five days; no heat whatever is used.

Two series of experiments were carried out:—

(1) Using a basal diet of bran and oats, 60 c.c. of milk heated in a steam sterilizer for one hour and a quarter, and water *ad lib.* In this there was abundant water-soluble anti-beri-beri, and fat-soluble growth

factor. The control animals died between the thirtieth and fiftieth day with all the usual signs of guinea-pig scurvy and great loss of weight. The addition of Navy lime juice did not give any protection.

(2) In the second series Navy biscuit was used as the basal diet, to which was added steamed milk, 60 c.c., and lime water; on one day per week the biscuit was substituted by oats to supply the anti-beri-beri vitamine. Growth was well maintained for over a fortnight, then the weight rapidly decreased, scurvy signs were manifested, and death followed. Control animals with 20 grm. of fresh cabbage or 10 c.c. fresh de-acidified lime juice grew normally. Animals were given one-fifth of a tablet (= 4·8 c.c. fresh lemon juice), in solid form, by hand feeding. There was generally a loss of weight at first, but when they became accustomed to the diet the animals grew and remained healthy for over three months.

A further series was carried out to find the minimum amount of tablet which would act as a preventive dose. The daily amount was reduced to one-tenth (= 2·4 c.c. fresh lemon juice). In two animals this was sufficient, but in two others signs of paralysis appeared and they had to be placed on normal diet again; this was followed by recovery.

Keeping Properties.—Animals were also given tablets which had been stored for three months and more, and no loss of protective power was noticed. Tablets kept at 37° C. became dark in colour and softer, due to caramelization of the lactose, and this appears to affect to a slight extent the vitamine properties. In no instance were the tablets found to have become mouldy whether kept at room temperature or blood heat.

Therapeutic Action.—If administered to an animal following scurvy signs, which were not too marked, a cure was effected (fig. 8, p. 65), but if diarrhœa had set in, or a great loss of weight had occurred, the animals died.

As they have been so fully reported elsewhere, it is unnecessary to describe the scurvy symptoms in the animals or the post-mortem findings, such as muscular, mucous, periosteal and intestinal hæmorrhages, fragility of bones, disorganization of joints, &c., with general atrophy of the intestinal tract, but the condition of the suprarenal bodies, as emphasized by McCarrison, deserves further mention. He has shown that in guinea-pigs these organs are enlarged, hæmorrhagic, and have a reduced adrenalin content, and with this there is a tendency to secondary bacterial blood infections (found once in my cases). In my experiments the changes in the suprarenals of the scorbutic animals were very marked. There was marked congestion of the whole gland, with diffused or circumscribed hæmorrhages and fatty degeneration of the cells chiefly affecting the cortical areas. The weight of the glands was roughly three times that found in healthy guinea-pigs.

The reduction of the normal secretion is, according to McCarrison, an early factor in the scurvy syndrome, and influences very clearly the metabolic changes in the animal—and he concluded that the evidence, so far available, points to the dependence of the functional perfection of the adrenal glands upon the adequate supply, in the food, of the accessory food factors of the various classes.

COMPARATIVE RESULTS.

Scurvy animals	ANIMAL		SUPRARENALS	
	Original weight	Weight at death	Weight (mgrm.)	Milligrams per 100 grm. of body weight at death
No. 1. (1) Died on forty-eighth day; hæmorrhages and joint signs. Suprarenals, local and diffused hæmorrhages	340	235	270 225	115 96
No. 2. (4) Died on forty-fourth day; hæmorrhages and joint signs. Suprarenals: hæmorrhages and degeneration of cells in cortex	360	235	237 200	101 85
No. 3. (5) Died on fifty-third day; mucous and muscular hæmorrhages. Suprarenals: diffuse hæmorrhages and slight fatty changes	340	270	255 225	94 83
No. 4. (8) Died on forty-fourth day; joint signs. Suprarenals: intense hæmorrhagic condition of whole gland, fatty changes in cortex	320	240	185 175	77 73
No. 5. (31) Killed on twenty-seventh day; hæmorrhages and joint signs; ophthalmia. Suprarenals: extreme fatty degeneration of cortical cells, slight hæmorrhages	370	260	220 182	85 70

Five Scurvy Animals—Suprarenal Bodies.
Average weight per 100 grm. body weight at time of death, 87·9 mgrm.

Six Normal Animals—Suprarenal Bodies.
Average weight per 100 grm. body weight at time of death, 25·8 mgrm.

Many observations have shown that the onset of scurvy is very gradual. Dr. J. R. Comrie, in an account of scurvy in Russian prisons, stated that it might be four and a half months, but generally it is as long as six months, before symptoms are manifested. In the prescurvy stage, owing to the deprivation of antiscurvy factors, the metabolism of food is incomplete and a *positive* agent is added, which acts like a toxin, causing changes in the adrenals and their secretions, favouring the hæmorrhagic conditions which are such marked features of the declared disease.

The use of this concentrated preparation has shown that it has effective antiscurvy properties for animals. We may, therefore, with confidence, apply its use to man under conditions of service which preclude the regular provision of fresh fruit and other known preventive substances, such as when on detached duty in the Persian Gulf, West Coast of Africa, and in Arctic exploration. It could also be employed in the mercantile marine. Though it is not yet possible to say how long the activity of the retained antiscurvy vitamine will remain effective, it is probable that this will be of extended duration if the preparation is kept dry and cool. Dr. Chick, in her admirable work at Vienna, has found that 5 c.c. of lemon juice is a prophylactic dose for infants, therefore 24 c.c. would probably be ample for an adult (half lemon). The tablets dissolve fairly readily if added to water containing a small quantity of bicarbonate of soda, and it is suggested that these dissolved tablets, containing antiscurvy accessory factor, should be added to the ordinary non-active pleasant tasting lime juice at present issued.

For general service, when large quantities are required, the non-heated concentrated syrup, as obtained before being mixed with lactose, might be kept in bottles, and the equivalent of 24 c.c. of fresh lemon juice be given per man, after solution in water to which sugar has been added at the time of issue. It is probable that this syrup would keep without mould formation, and this aspect is at present under consideration.

In this investigation I am greatly indebted to Mr. R. C. Frederick for his able assistance in preparing the tablets used and to Sick Berth Steward A. Coules for his careful attention in feeding the animals. Finally I have to thank you for your sympathetic hearing of what is, I feel, a very incomplete treatment of the subject.

Diet			Number of animals	Length of experiment (days)	Results as regards—	
Special ration	Amount (grm.)	Basal diet			Growth	Occurrence of scurvy
Dried lemon juice =4·4 c.c.	One-fifth tablet	Bran and oats, heated milk	1	161	Good	Complete protection
Fresh lemon juice de-acidified	10 c.c.	Bran and oats, heated milk	1	120	Good	Complete protection
Fresh milk thirty-two days, then tablet added	60 c.c.	Bran and oats, water	1	110	Very good	Slight protection; scurvy appeared thirty-second day; cured by one-fifth tablet
Cabbage	20 grm.	Bran and oats, heated milk	1	114	Very good	Complete protection
None	—	Bran and oats, heated milk	7	29 to 52	None	Scurvy, five died; one cured by dried juice, one by cabbage
Dried lemon juice =4·4 c.c.	One-fifth tablet	Navy biscuit, heated milk, lime water	5	60 to 120	Good	Complete protection
Fresh lemon juice de-acidified	10 c.c.	Navy biscuit, heated milk	1	160	Very good	Complete protection
Old dried lemon juice at 15° C. =4·4 c.c.	One-fifth tablet	Navy biscuit, heated milk, lime water	2	112	Very good	Complete protection
Old dried lemon juice at 15° C. =2·2 c.c.	One-tenth tablet	Navy biscuit, heated milk, lime water	4	60 to 100	Very good	Two, complete protection, two showed signs and were treated
Old dried lemon juice at 37° C. =4·4 c.c. (1) (2)	One-fifth tablet	Navy biscuit, heated milk, lime water	2	70 to 100	Fair	(1) Complete protection; (2) slight scurvy signs, and loss of weight, cleared up on juice being doubled on thirty-first day
Navy lime juice de-acidified	10 c.c.	Navy biscuit, heated milk	1	37	None	Scurvy, died thirty-seventh day
None	—	Navy biscuit, heated milk, lime water	2	28 to 42	None	Scurvy, died twenty-eighth and forty-second day
Cabbage	20 grm.	Navy biscuit, heated milk, lime water	1	110	Very good	Complete protection

BIBLIOGRAPHY.

Barnes and Hume. *Biochem. Journ.*, November, 1919.
Chick and Dalyell. *Wien. klin. Wochenschr.*, December, 1919.
Chick and Delf. *Biochem. Journ.*, November, 1919.
Chick, Hume, and Skelton. *Lancet*, 1918, ii, 734.
Comrie, J. R. *Edin. Med. Journ.*, 1919.
Darling. *Journ. Amer. Med. Assoc.*, February, 1915.
Delf and Tozer. *Biochem. Journ.*, December, 1918, xii, 416.
Givins and McClugage. *Amer. Journ. Dis. of Child.*, July, 1919.
Heiss and Unger. *Biochem. Journ.*, June, 1919.
McCarrison, R. *Indian Journ. Med. Research*, vi, 275, 550.
Smith, A. Henderson. *Journ. Roy. Army Med. Corps*, 1919, xxxii, 93, 188.
Walshe, F. M. R. *Brit. Journ. Dis. of Child.*, 1918, xv, 258.
Willcox, W. H. *Proc. Roy. Soc. Med.*, xiii (Sect. Therap.), 7.

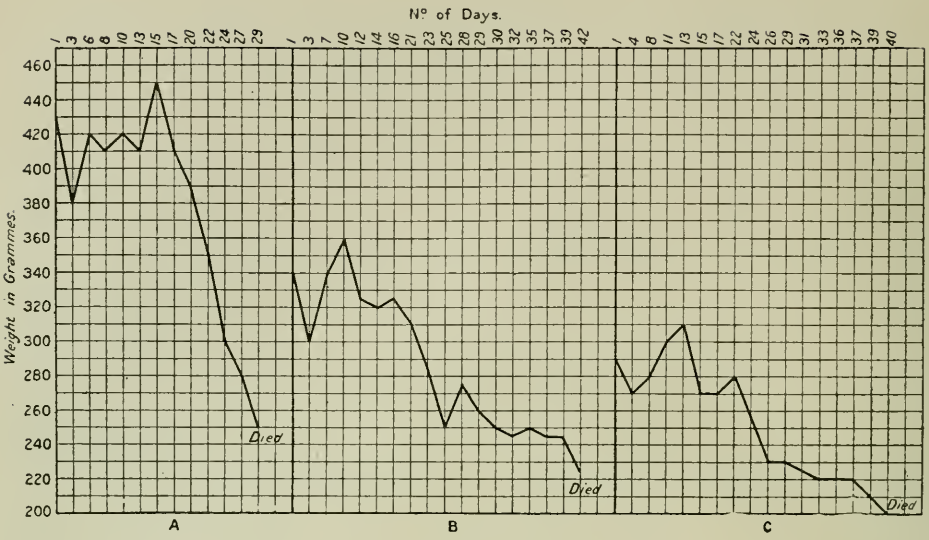


FIG. 2.

A—No. 23. Diet : Navy biscuit, 30 grm. ; heated milk 60 c.c. ; lime water, 20 c.c.
B—Composite Chart. Nos. 1, 2, 4, 7, 8. Diet : Bran and oats, 40 grm. ; heated milk 60 c.c.
C—No. 16. Diet : Navy biscuit, 30 grm. ; heated milk, 60 c.c. ; Navy lime juice, 10 c.c.

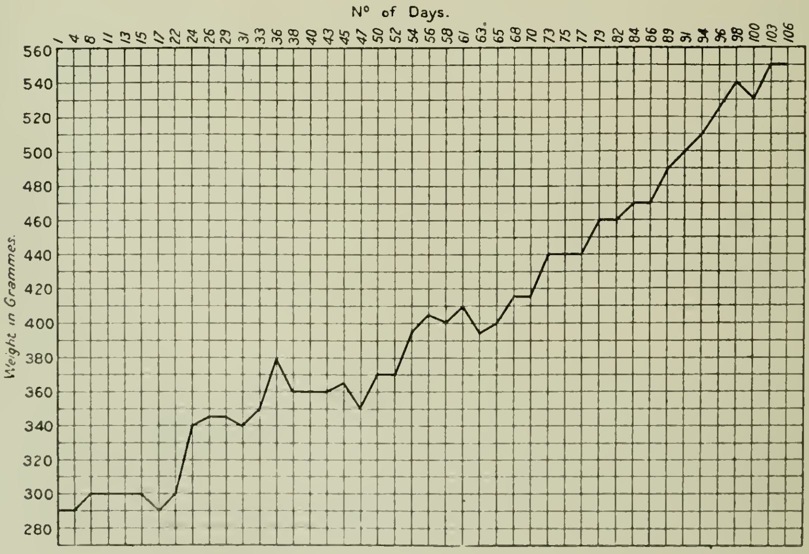


FIG. 3.

No. 15.—Diet : Navy biscuit, 40 grm. ; heated milk, 60 c.c. ; raw cabbage, 20 grm. daily.

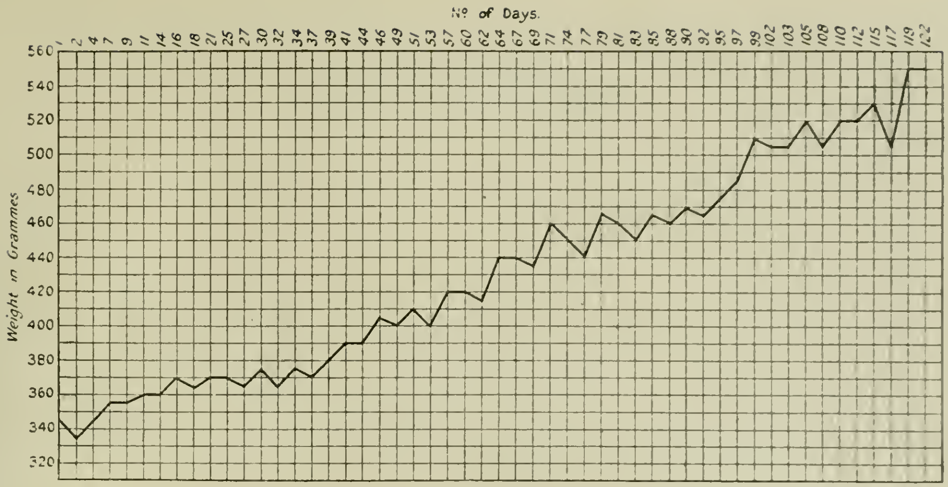


FIG. 4.

Composite Chart. Nos. 17, 18, 19, 20.—$\frac{1}{5}$ tablet dried juice. Diet : Biscuit, 30 grm. ; heated milk, 60 c.c. ; lime water, 20 c.c.

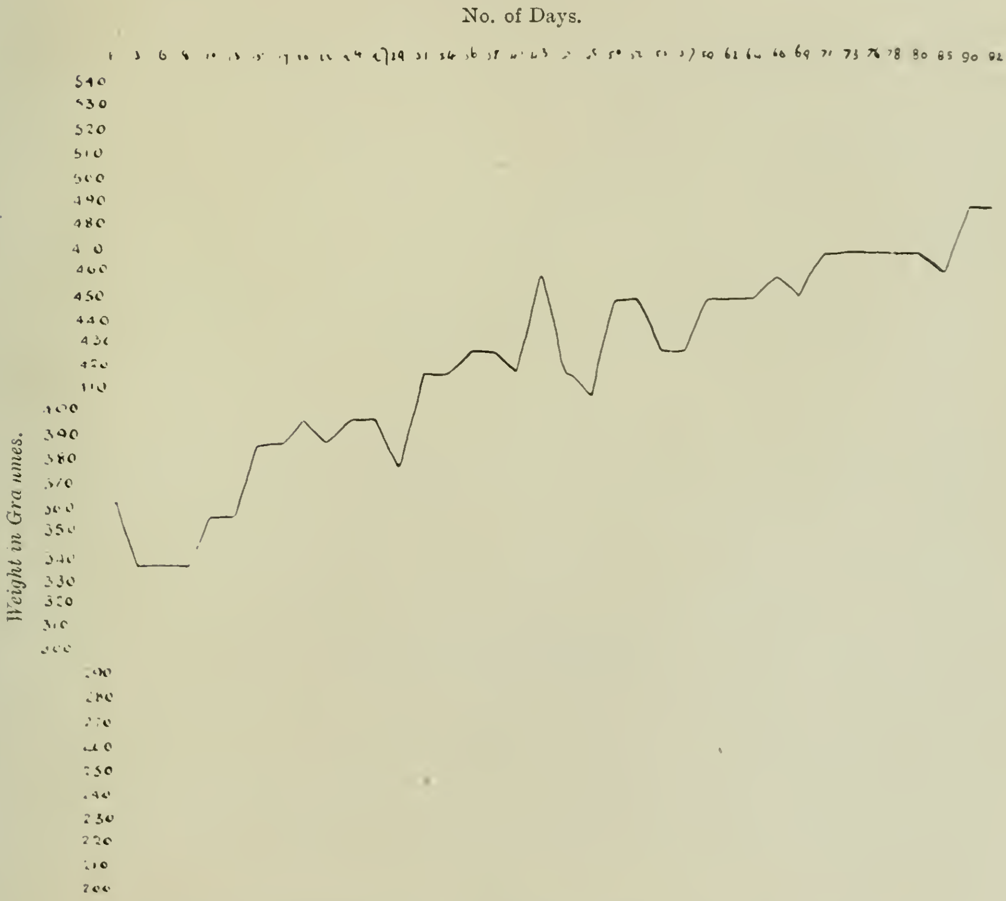


FIG. 5.

Biscuit, 30 grm. ; milk, 60 c.c. ; lime water, 20 c.c. ; old room temperature, dried juice, $\frac{1}{5}$-tablet.

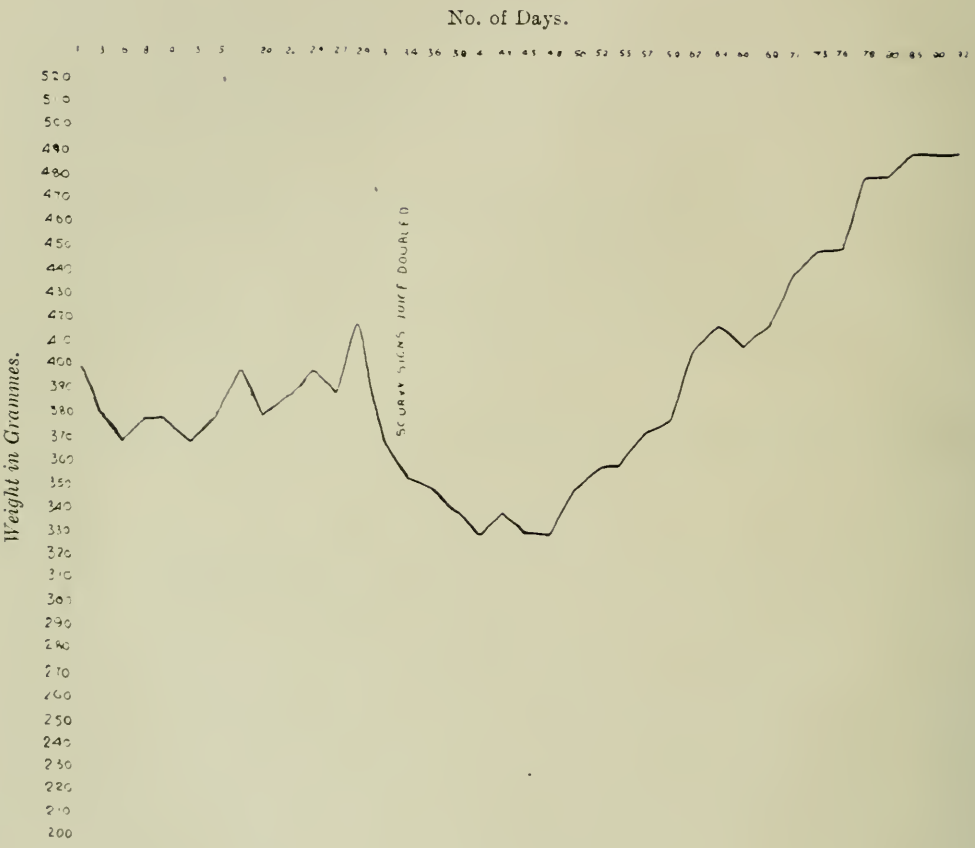


FIG. 6.

Navy biscuit, 30 grm.; heated milk, 60 c.c.; lime water, 20 c.c.; old dried juice at 37° C., $\frac{1}{5}$-tablet.

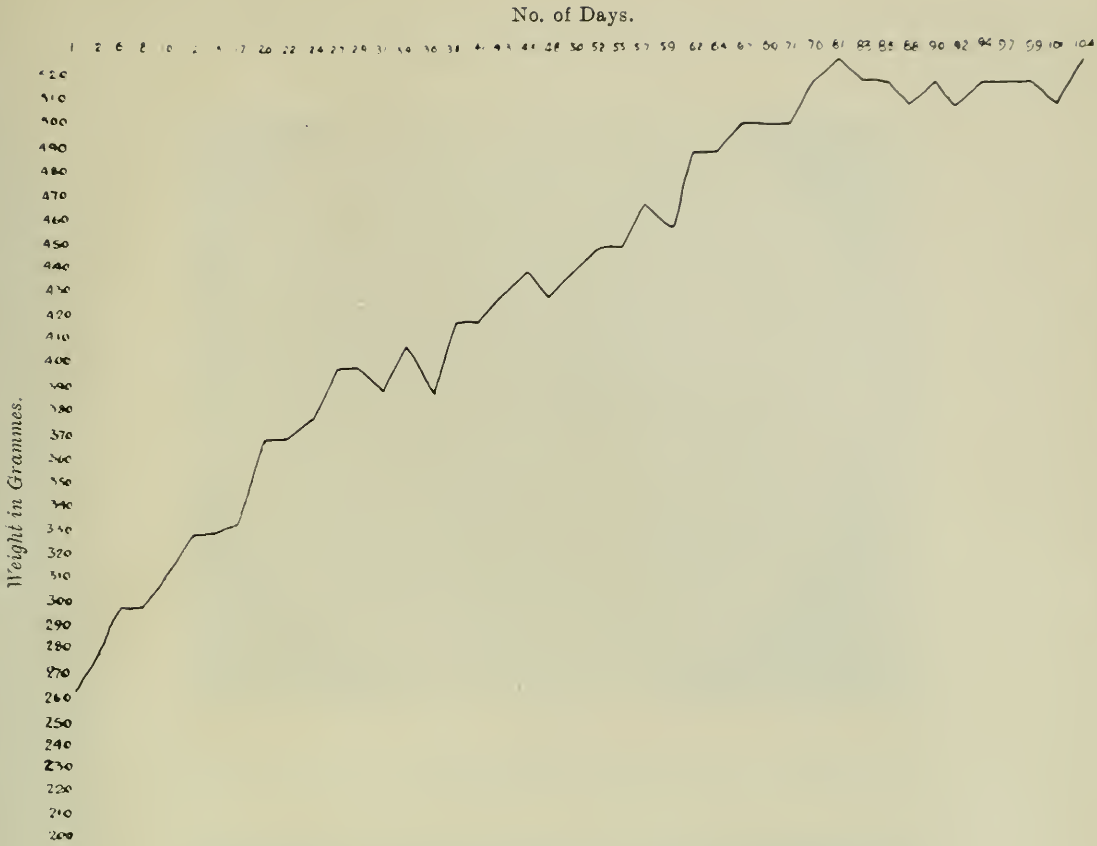


FIG. 7.

Navy biscuit, 40 grm.; heated milk, 60 c.c.; old dried juice at 15° C., $\frac{1}{10}$-tablet; lime water, 20 c.c.

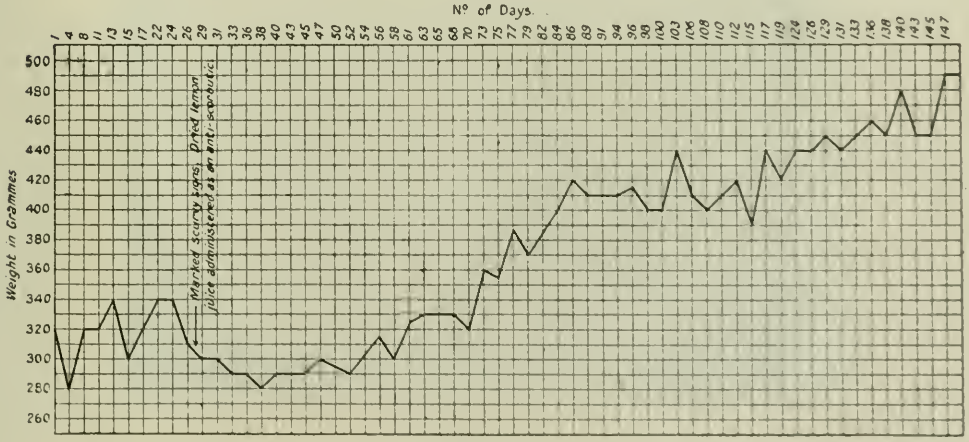


FIG. 8.

No. 3.—Diet: Bran and oats, 40 grm.; heated milk, 60 c.c. $\frac{1}{3}$ tablet dried lemon juice on twenty-eighth day, when marked scurvy signs were present.

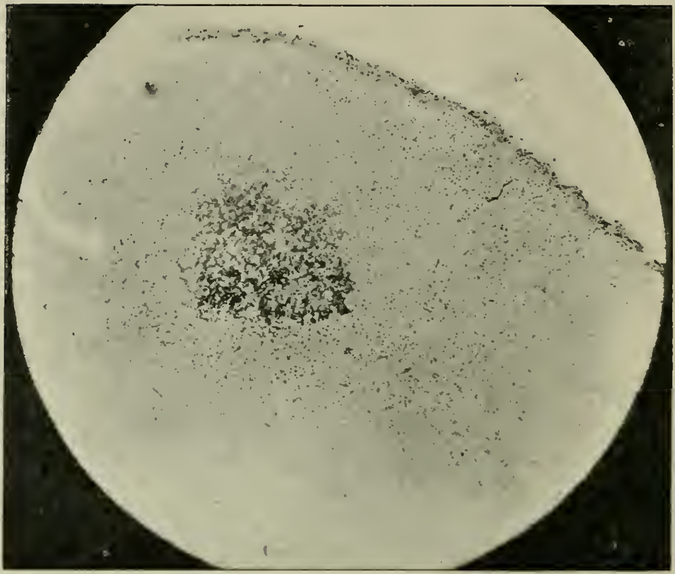


Fig. 9.

Guinea-pig No. 5. Died on fifty-third day; diffused hæmorrhage in the cortex of the suprarenal.

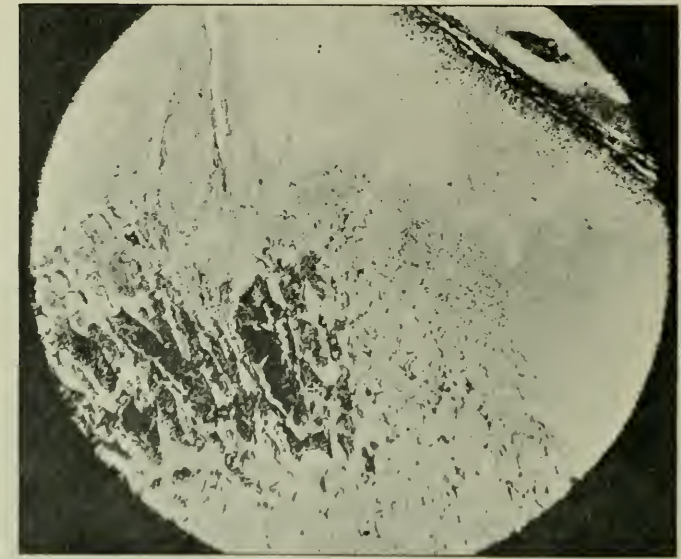


Fig. 10.

Guinea-pig No. 8. Died on forty-fourth day; intense congestion of suprarenal with extensive hæmorrhages in cortex.

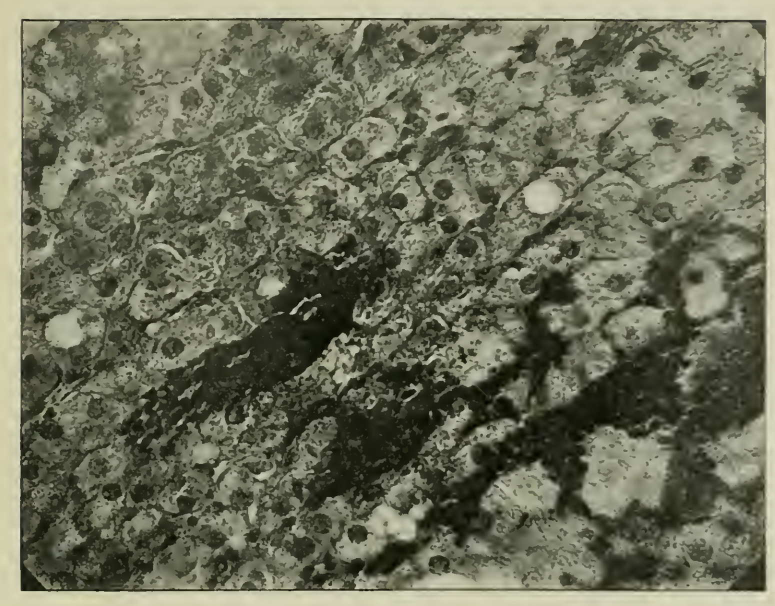


FIG. 11.

Guinea-pig No. 23. Died on thirtieth day; cortical hæmorrhages and fatty degeneration of suprarenal.

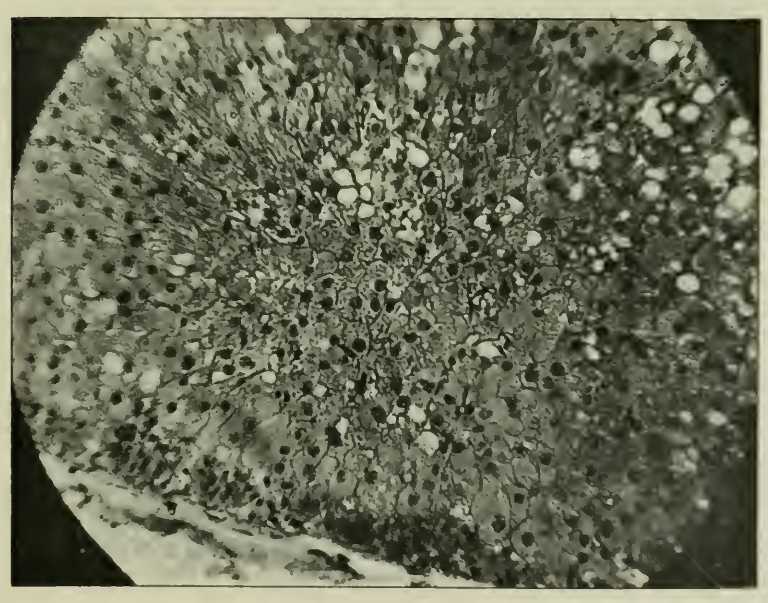


FIG. 12.

Guinea-pig No. 8. Died on forty-fourth day; very marked fatty changes in cortex, with slight hæmorrhages.

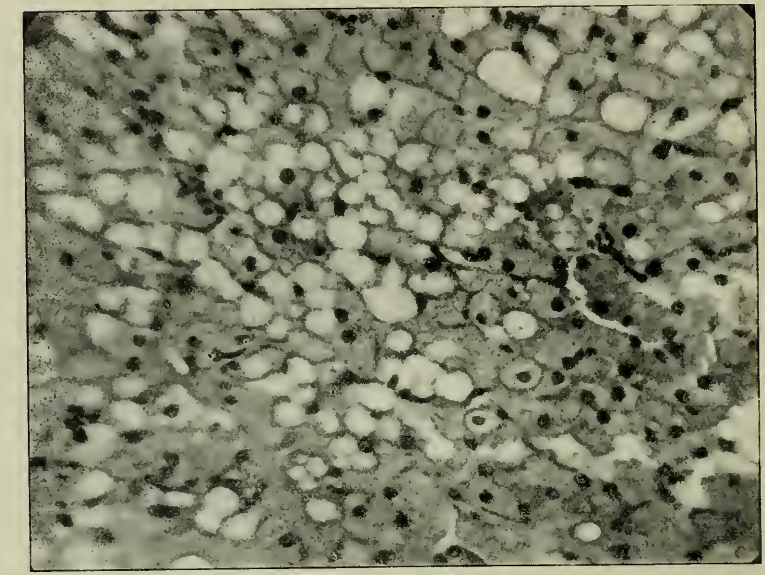


Fig. 13.

Guinea-pig No. 31. Killed on twenty-seventh day; intense fatty degeneration of suprarenal. (Under a higher magnification.)

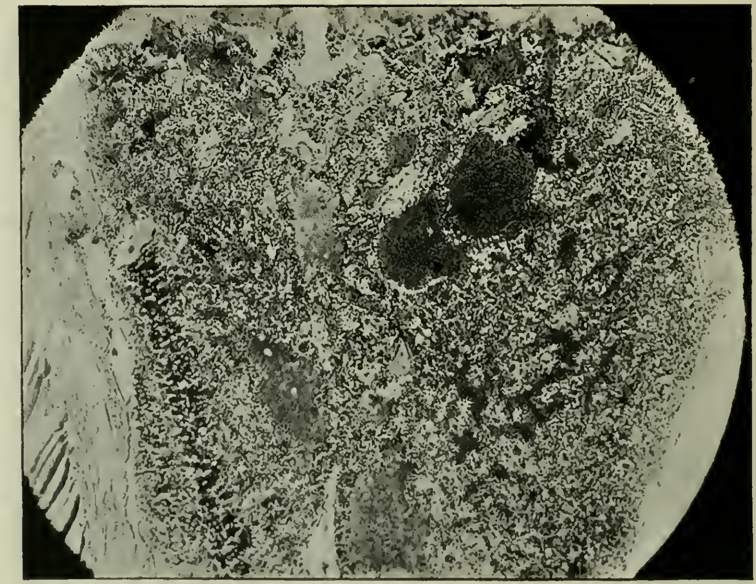


Fig. 14.

Guinea-pig No. 8. Died on forty-fourth day; intense hæmorrhages into medulla near epiphysis.

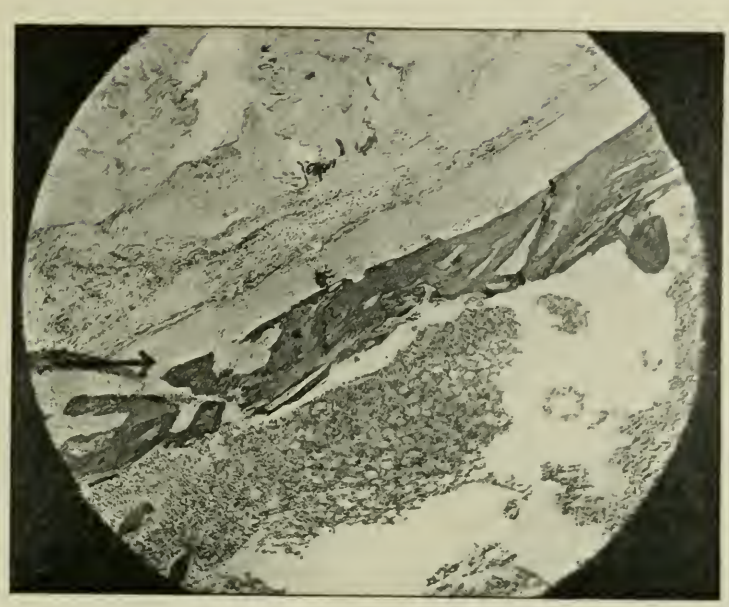


FIG. 15.
Guinea-pig No. 1. Died on forty-eighth day; sub-periosteal hæmorrhage.

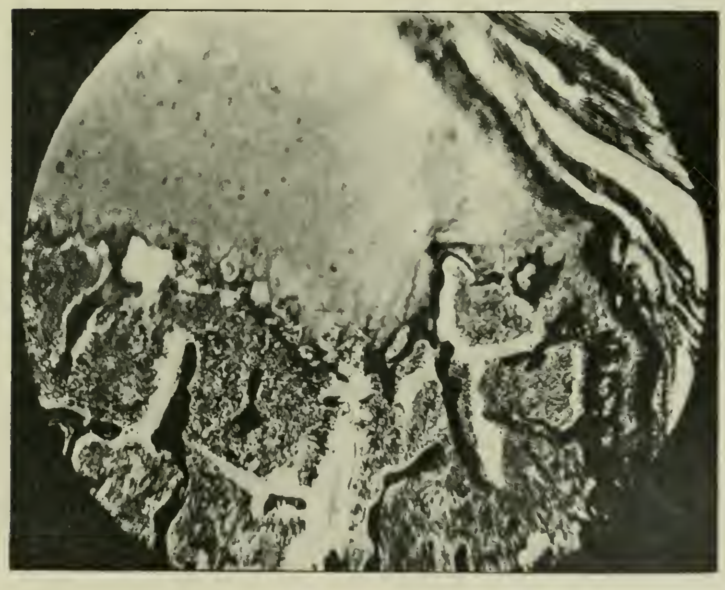


FIG. 16.
Guinea-pig No. 8. Died on forty-fourth day; costo-chondral junction, showing irregular line of ossification.

DISCUSSION.

Major-General Sir WILLIAM MACPHERSON: Scurvy in Mesopotamia, notwithstanding what Dr. W. H. Willcox has written on the subject, was not altogether due to want of recognition of its causes, but to the impossibility of getting fresh vegetable and other foods up to troops operating in certain areas. These foodstuffs became decomposed in consequence of climatic conditions before reaching the troops, and it was these troops which suffered most. As regards the resemblance between scurvy and beri-beri this was pointed out by myself in a report which is published in the official reports on the Russo-Japanese War. I observed thousands of cases of beri-beri amongst the Japanese, followed by some 20,000 cases of scurvy amongst the Russians in Port Arthur. The resemblance of the two cases clinically was so marked that it formed the subject of a special report. The cause amongst the Japanese was the use of highly milled and overcooked rice; and amongst the Russians the absence of fresh food and vegetables during the siege of Port Arthur.

Colonel S. L. CUMMINS: Rear-Admiral Bassett-Smith's paper has a great practical value. All who have heard or who will read the paper will recognize what this will mean to men of the naval and military and other forces, &c. Not only that, Rear-Admiral Bassett-Smith has come with his paper already in a practical form, and the method worked out and prepared ready for immediate adoption.

Proceedings of the Royal Society of Medicine.

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SUPPLEMENT

(Vol. XIII, No. 1, November, 1919).

NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

The Nervous Heart: Its Nature, Causation, Prognosis and Treatment. By R. M. Wilson, Captain R.A.M.C., and John H. Carroll, Major, M.C., U.S.A. Pp. vii + 136. Price 6s. net. London: Henry Frowde, Hodder and Stoughton, 1919.

"This book is an attempt to view the problems of heart disease, and especially functional heart disease, from a new angle—that of the nervous system." Thus the authors define the object of their book, but at first sight one does not consider that the neurogenic aspect of heart disease is exactly new. What they have set out to describe is the action and reaction of the vagus depressor system and of the sympathetic system in the production of circulatory control and circulatory disturbance. "Life is an expression of the interrelation of pressor and depressor activities, of sympathetic and vagus depressor activities." Dealing with functional diseases they find that at least 30 per cent. of all cases of irritable heart in soldiers are due to the infection of trench fever, the remaining percentages being distributed among diseases of which "rheumatism" and dysentery are probably the most important. It is, therefore, suggested that in these diseases a toxin of unknown nature acts upon the vagus depressor mechanism, increasing its irritability. The reader will find numerous tracings of the pulse and respiration curves to illustrate the authors' theories, but he will have to learn a new terminology and new views on the mechanism of the circulation before he can grasp the various vagal and sympathetic reactions. Breathlessness, giddiness, præcordial pain, and vasomotor phenomena are dealt with in the light of these reactions. As regards the treatment of functional heart disease the first thing to do, when possible, is to remove the underlying infection. Palliative treatment consists in lowering the irritability of the vagus depressor system, or increasing the resources of the sympathetic system.

Constipation and Allied Intestinal Disorders. By Arthur F. Hurst, M.A., M.D.Oxon., F.R.C.P. Second edition. With 56 illustrations; pp. xx + 440. Price 16s. net. London: Henry Frowde, Hodder and Stoughton, 1919.

The first edition of Dr. Hurst's work was published in 1909. Since then the author has found it necessary, as a consequence of his extensive radiographic studies of the alimentary canal and of his association with surgeons in a field hitherto restricted to the physician, to modify his views so substantially that the present production is a new work rather than second edition. The scope of the present monograph is remarkably comprehensive— 429 pages devoted to a subject which the standard text-books on medicine dismiss in three or four. The author explains that although the book is founded on his own investigations, it epitomizes a thorough study of all the literature that has ever appeared. Indeed, opinions and statements of nearly 350 authorities are actually quoted, and this, we understand, does not comprise the entire number consulted. Dr. Hurst defines constipation as a condition in which none of the residue of a meal taken eight hours after defæcation is excreted within forty hours, a definition which embraces all clinical varieties. For practical purposes an individual may be considered to be constipated if his bowels are not open, at least, once in forty-eight hours. Three main varieties of constipation are defined. Colic constipation or delay in the colon; dyschezia; and a third class due to deficiency of fæces. The group defined as dsychezia is conveniently separated from the first-named as designating a normal passage through the colon but with inadequate final evacuation when the pelvic colon has been reached. This term was originally employed by Robert Barnes to denote painful defæcation, but for the word in its present connexion, Dr. Hurst, as is well known, was originally responsible although he ascribes to Chevalier the credit of having recognized in 1819 the form of constipation in which purgatives did no good but for which enemata were necessary. Colic constipation is described as due to deficient motor activity of the intestines and to conditions which necessitate an excessive force to carry the fæces to the pelvic colon. Deficient motor activity is due as a rule to a constitutional weakness of the intestinal musculature but certain diseases contribute to such a weakness, in particular chlorosis and the acute specific fevers. It may also be due to diminished reflex activity either from an insufficient stimulus or deficient excitability of the intestinal mucous membrane or to general nervous depression as in hypochondriasis. Finally, it may be due to inhibition of motor activity from a central cause, a reflex cause—e.g., visceral pain, or because the sympathetic (the inhibitory nerve to the viscera) is stimulated, as in lead poisoning. A fourth group of cases comprised under the consideration of deficient motor activity is constituted by enterospasm, or, as it is more generally known, spastic constipation. The second large group of causes of colic constipation are referable to obstruction either from peculiarity of the fæces or from narrowing of the intestinal lumen with, as would be expected, the consideration of a large number of conditions often demanding surgical treatment. In this connexion Dr. Hurst objects that a true ileal kink, if it ever really leads to ileal stasis, probably does so not directly but indirectly by causing absence of relaxation of the ileo-cæcal sphincter (achalasia) just as gastric adhesions may upset the pyloric mechanism. Turning to dyschezia, Dr. Hurst reiterates the futility of employing purgatives since activity of the intestines is in uncomplicated cases normal. The chief causes of this variety of constipation are first, inefficient defæcation as from habitual disregard of the call to empty the bowels, weakness of the voluntary muscles of defæcation, unfavourable posture in the process of defæcation, and weakness of the reflex. As a second group of causes of dyschezia the various conditions leading to obstruction are again under consideration (as in colic constipation); in this instance, of course, the rectum and the pelvi-rectal flexure are the parts of the bowel concerned. Gynæcological causes play a very important part in the production of dyschezia, by their influences upon the pelvic portion of the alimentary canal, and even enlarged prostate must not be forgotten. The third class of cases of constipation is a small one, in which the condition is ascribed to deficiency of fæces. These cases include those in which insufficient food residues reach the colon—generally because insufficient food is for some reason ingested—and those in which the peculiarity of excessive digestion and absorption occurs, the condition originally described by Goodhart in 1902 as "greedy colon." Dr. Hurst destroys the popular fallacy that bismuth salts can cause constipation, this idea having evidently arisen from the astringent effect of bismuth in certain forms of diarrhœa only, namely, those in which there is excess of sulphuretted hydrogen from abnormal putrefaction. The symptoms of constipation are next considered. These are due either to reflex causes or to the mechanical pressure exerted by fæcal accumulation. Intestinal intoxication Hurst considers to be due to abnormally long retention of fæces rather than to any increase in bacterial decomposition. The author points out that the results of constipation as given by Sir Arbuthnot Lane include almost every disease from cancer of the breast and exophthalmic goitre to adenoids and flat-feet. He reviews the work of Lane and his assistants at considerable length, and many of their results he considers to be due to imperfect radiographic technique. The association of constipation with hyperchlorhydria deserves a passing word, for there appears to have been a tendency to confuse cause and effect. Dr. Hurst considers that the excess of HCl softens cellulose and renders it more susceptible to bacterial solution so that vegetable foods are abnormally digested. Thus, in his view, constipation is not a cause of duodenal ulcer but the result of hyperacidity associated with the ulcer. Again, constipation he regards as the result of chlorosis, and not (as is traditional) its cause, although the absorption of toxins greatly aggravates many of the symptoms in chlorosis. Approximately the last hundred pages are devoted to treatment; the most suitable foodstuffs are first considered and then the indications for purgatives with the special properties of each. Surgical treatment receives a critical survey. Apart from definite organic obstruction of the intestine for which surgery is unquestionably indicated, the author is clearly unenthusiastic in his reception of the various operations which have during the last ten years been recommended for the relief of constipation. With more persistent and scientifically adjusted medical treatment and greater accuracy in diagnosis to aid in the selection of the operation suitable to the particular condition found, the number of such operations and their mortality percentage would be reduced while the number of successes would be increased. A final chapter deals with constipation in infants in which the author acknowledges his association with Dr. H. C. Cameron.

SPAS AND HEALTH RESORTS OF THE BRITISH ISLES: THEIR MINERAL WATERS, CLIMATE, AND THE TREATMENT TO BE OBTAINED. With a Section on Curative Institutions. By THOMAS DAVY LUKE, M.D., F.R.C.S.Edin. With 32 full-page illustrations and a sketch map; pp. xii + 318. Price 7s. 6d. net. London: A. and C. Black, Ltd., 1919.

This consists mainly of information gleaned about the various spas, climatic resorts, and marine stations of the British Isles. As a dictionary of reference it should be useful to medical practitioners, for it contains in a convenient and accessible form most details which might be asked for at the present day by patients who formerly visited German health resorts. It is impossible to compile a work of this kind without some inaccuracies—as the author himself points out in the preface. Too much reliance must not be placed upon the sunshine records claimed by some of the resorts. There are a few short chapters at the commencement of the book referring to spa treatment, special diet cures, climate, &c., which, although of a sketchy character, are of interest. A list of hydropathic institutions and sanatoria is given at the end of the book. The illustrations are good, but are not in every instance in apposition with the descriptions of the places.

TRENCH FEVER: A LOUSE-BORNE DISEASE. By Major W. BYAM, R.A.M.C., Captains J. H. CARROLL, U.S.R., J. H. CHURCHILL, R.A.M.C.(T.), LYN DIMOND, R.A.M.C., V. E. SORAPURE, R.A.M.C., R. M. WILSON, R.A.M.C. and LL. LLOYD, R.A.M.C.(T.). With an Introduction by Lieutenant-General Sir T. H. GOODWIN, K.C.B., a Foreword by Major-General Sir DAVID BRUCE, K.C.B., F.R.S., and a Summary of the Report of the American Trench Fever Commission by Lieutenant R. H. VERCOE, R.A.M.C. With charts; pp. xvi + 196. Price 10s. 6d. net. London: Henry Frowde, Hodder and Stoughton, 1919.

This book is a record of the experiments carried out by the authors, at Hampstead, for the War Office Trench Fever Committee, and of their conclusions following on the results of these experiments. The first chapter deals with the manifestations of the acute disease and the symptoms are detailed in the order of frequency as found to occur in 200 consecutive cases. Five different types of fever are described as occurring in the experimentally infected cases. The next chapter records the experiments dealing with the mode of transmission. As a result of these investigations they state "we are of opinion that trench fever is conveyed by the excreta of infected lice; that the excreta may enter through the broken skin; that rubbing and scratching promote infection, but that the bites of lice may possibly cause a sufficient lesion to enable the virus to enter the body." This is a most important finding and is in accord with the findings of the Commission of the American Red Cross Research Committee on Trench Fever. It gives us the key of the problem of how best to attack this disease. We know its disabling results in war but have yet to learn its potency for ill-health in peace. The pathology, the question of immunity, and the disease in its chronic form are all dealt with. Prophylaxis, always of first importance, but more especially so in this case, where as yet no specific treatment has been discovered, is discussed at length, and the findings of many experiments recorded. There are four appendices; three being tabulations of the experiments, and the last a Summary of the Report of the Commission of the American Red Cross Research Committee on Trench Fever by Lieutenant R. H. Vercoe, R.A.M.C., which is most useful, when read in conjunction with the findings of the authors. The book will be of value to all interested in this disease and the interest will be enhanced in that the knowledge it contains will be fresh to the majority of its readers.

A WOMAN DOCTOR: MARY MURDOCH OF HULL. By HOPE MALLESON, B.A. With a Preface by L. B. ALDRICH-BLAKE, M.S., M.D.Lond. With four portrait illustrations; pp. xiii + 231. Price 7s. 6d. net. London: Sidgwick and Jackson, Ltd., 1919.

This is a sympathetic life of Dr. Mary Murdoch of Hull (1864-1916) who was Scottish by descent on both sides, and, in the words of her biographer, had "the imagination, the high mental energy, the quickness of wit and interest in ideas characteristic of the race which is popularly called Celtic." Her father was described by his daughter as "one of God's saints, which is rare in a lawyer; and his Latin and scholarship were a by-word in the North of Scotland forty or fifty years ago." She began her medical work in 1888 in London, and after qualifying in 1892, held some resident and other appointments before settling down for her life's work in Hull in 1896. During the next twenty years she led a busy and unselfish life as a successful practitioner and a willing helper in many public and private schemes concerned with the welfare of women and children. One of the secrets of her success was her splendid personality; the poorer people called her "our Lady Murdoch"; wherever she went affection and gratitude went with her, and she had the capacity of always appearing to have plenty of time. Her superabounding energy had to contend with the handicap of gastric ulcer, and of one occasion, when she was pulseless and kept under morphine for some days, she wrote: "My own death was quite sudden and only lasted a few hours at the most. Three other times I have been at the border, but only over it once, and just held back by the skirt." She played a prominent part in public life, not only in Hull but in connexion with Woman's Suffrage and the National Union of Women Workers, and spoke and wrote much on public health matters. There are two appendices containing obituary notices and her lectures and addresses, the first of which is the October Address to the London School of Medicine for Women in 1904, and a short preface by Dr. Louisa Aldrich-Blake.

BARBED-WIRE DISEASE: A PSYCHOLOGICAL STUDY OF THE PRISONERS OF WAR. By A. L. VISCHER, M.D.Basle, M.R.C.S.Eng. Translated from the German, with Additions by the Author. With an Introductory Chapter by S. A. KINNIER WILSON, M.A., B.Sc., M.D.Ed., F.R.C.P.Lond. Pp. 84. Price 3s. 6d. net. London: John Bale, Sons and Danielsson, Ltd., 1919.

An agreement made between Great Britain and Germany in July, 1917, indicates an official recognition of the group of symptoms associated with captivity under the now familiar term "barbed-wire disease." It contains a paragraph to the effect that prisoners of war, who have been at least eighteen months in captivity and who are suffering from barbed-wire disease, shall for the future be recognized as suitable for internment in Switzerland. It is the causation and description of this group of symptoms with which Dr. Vischer is concerned. An extensive introductory chapter by Dr. Kinnier Wilson occupies fully one quarter of the book; it forms an important amplification of the author's views, and fills the gap resulting from his inability to utilize the English literature of life in war prison camps. Dr. Wilson states that, for the purposes of his introduction, he has read every available book of English origin on the subject, and we gather that English writers agree with the German and French in complaint of the absence of privacy with its resulting irritability. It has long been accepted as a commonplace that people who are inevitably and unavoidably closely associated, get on each other's nerves. Such a circumstance is invariably mentioned by explorers, during long sea voyages, in institutions and monasteries, and even to some extent during ordinary military service. But whilst all of these contribute certain essential circumstances which conduce to the peculiar psycho-neurotic symptom-complex of a prisoner of war, counterbalancing elements which are generally present in the others are entirely lacking in the prisoner. Thus, factors common to all internment, civil or otherwise, such as restrictions, rules, regulations, limited space, monotonous food, and the absence of means for sexual intercourse, are supplemented in the prisoner of war by complete absence of opportunity for privacy, by ignorance of duration of the incarceration, by irregularity of communication from home, and "more than anything else, by the barbed wire winding like a red thread through the mental processes." The chief symptoms which constitute the syndrome are irritability, difficulty in concentrating, restlessness, failure of memory, moodiness, depression and unpleasant dreams, distrust and delusions of persecution. Dr. Vischer is of opinion that "probably very few prisoners who have been over six months in camp are free from the disease and that many will return to their homes with a damaged mentality." This is not in accordance with Dr. Wilson's experience of returned *English* prisoners, and he is disposed to lay much weight upon reaction to surroundings depending on the prisoner's character, temperament, and disposition. To the superior capacity of the Englishman "to make the best of it" and to preserve an invincible spirit in the face of the most appalling circumstances, of physical sufferings and mental torments combined with a sense of humour, must be ascribed the salvation, moral and mental, of many a British soldier.

AUTOBIOGRAPHICAL REMINISCENCES OF SIR HERMANN WEBER. Written privately for the Family. With Annotations and a List of his Medical Writings by his Son FREDERICK PARKES WEBER. With 21 illustrations; pp. xlii + 124. London: John Bale, Sons and Danielsson, Ltd., 1919.

To his friends—and all those who met the late Sir Hermann David Weber rapidly passed into this category—these memoirs will have a very peculiar attraction as those of an exceptionally lovable character; the only drawback is that they practically stop about 1866. Born on December 29 or more probably 30, 1823, at Holzkirchen, a small village in the north of Bavaria, he characteristically chose his profession on account of the effect of the visits of Dr. Wiegand, of Fulda, on his mother when ill of bronchitis, and the desire to give similar help to others. He worked first at Marburg on the preliminary subjects under Bunsen, Fick, and Carl Ludwig, and later at Bonn under his uncle, Friedrich Nasse, whose name is perpetuated in Nasse's law, to the effect that hæmophilia is propagated entirely by unaffected females. After holding the post of first assistant in the medical clinic for two years he came in 1851 to London as Resident Medical Officer to the German Hospital, Dalston, and soon afterwards became a member of the select "Medical Society of Observation," thus beginning his extensive friendships with the leading members of the profession in London. In 1853 he was appointed physician to the German Hospital, married in the following year, and became a naturalized British subject in 1866. Although the reminiscences do not go further than 1866, there is a special chapter on some medical friends the contents of which, incidentally, refer to events of a much later date. Among the numerous interesting points in these thumb sketches of his friends special attention may be directed to Sir Hermann's opinion that Addison had a considerable share in Bright's description of renal disease, and that Bright, in contrast to his colleagues Addison and Gull, rarely expressed a decided opinion and generally hesitated in difficult cases. There is a great charm about this simple frank narrative, and though it may be difficult for those who knew the late Sir Hermann Weber to form an unbiassed opinion on the question whether or not the publication should be confined to a comparatively narrow circle, there does not appear to be any reason why the larger public should not have the pleasure of reading the autobiography of one who was for so long such a prominent figure in the medical life of London. The illustrations are well done and Dr. Parkes Weber has added a number of footnotes giving details, resembling those in the epitome of the Dictionary of National Biography, of the various persons mentioned in the text.

MEDICAL RESEARCH AND HUMAN WELFARE: A RECORD OF PERSONAL EXPERIENCES AND OBSERVATIONS DURING A PROFESSIONAL LIFE OF FIFTY-SEVEN YEARS. By W. W. KEEN, M.D., LL.D. The Colver Lectures, 1917. With portrait and 6 plates; pp. xi + 160. Price $1.25. London: Skeffington and Son, Ltd., 1919.

This pleasantly written volume contains the second Colver Lecture at the Brown University which Professor Keen entered at the age of 18, in 1855, and two photographs of the lecturer, in uniform, taken in 1917 and in 1861 when he took part in the old horribly fatal surgery of the Civil War and cannot remember that he ever saw a wound that was not infected. The present state of surgery and of medical practice is contrasted throughout with the conditions existing when the lecturer first entered the profession, and the improvement due to medical research is pointed out with statistics to emphasize the moral that a small outlay on scientific investigation insures an enormous economy both in human life and pecuniary expenditure. Carrel, who, in what may be called "mathematical surgery," can now predict to a day when a wound will be completely healed with almost the accuracy of an astronomer in predicting an eclipse, is the type of the new surgeon who restricts his actual surgical work so as to carry on researches in bacteriology, physiology, physiological chemistry and physiological physics. Pasteur's epoch-making advances are set out. and the lecturer says that just as Sir Humphry Davy, when asked what was his greatest discovery, instantly replied "Michael Faraday," so might Pasteur in similar circumstances have answered "Joseph Lister." As an example of the need for medical research reference is made to a fatal case of tetany after thyroidectomy, before the connexion between the parathyroids and tetany was known, and remarks "what a chapter could be written on the calamities of unavoidable ignorance." The lessons of the war and the life-saving influence of medical research among the belligerents are fully noticed.

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SUPPLEMENT

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NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

AUTO-EROTIC PHENOMENA IN ADOLESCENCE: AN ANALYTICAL STUDY OF THE PSYCHOLOGY AND PSYCHOPATHOLOGY OF ONANISM. By K. MENZIES. With a Foreword by Dr. ERNEST JONES. Pp. viii. + 96. Price 4s. 6d. net. London: H. K. Lewis and Co., Ltd., 1919.

An introductory chapter explains psycho-analysis as standing in the same relation to the soul that surgery does to the body, and the principle underlying psycho-analysis is given in the words of Dr. Ernest Jones whose writings as well as those of Dr. Havelock Ellis and Freud are frequently quoted throughout. The subject of masturbation is then considered in four chapters devoted to its psychology, pathology, ethical considerations, and treatment. It appears that an anonymous work entitled "Onania, or the Heinous Sin of Self-Pollution," published in the eighteenth century and ascribed to Tissot, first asserted that masturbation gave rise to very serious results; but the opinions of Sir James Paget, Forel, Brill and other authorities are quoted to correct the alarmist statements still made both by some well-intentioned writers and by interested quacks. The author agrees with most modern writers that the principal evil results are psychical and not physical, and are manifested in heightened self-consciousness, oversensitiveness, timidity and exhausting mental conflict. The section on treatment therefore deals with mental rather than physiological means; the individual should be surrounded with an atmosphere of sympathy and encouragement rather than of condemnation, being encouraged to analyse freely and aloud in the presence of his confidant the feelings of distress and remorse that attend on his failing, and the general aim should be the withdrawal of over-emphasis from the habit, to which end it would appear inadvisable and sometimes dangerous to extort the promise of an immediate cessation of the practice, as psycho-analysis has shown that the abrupt breaking of long-continued masturbation may be followed by the appearance of a neurosis.

THE FUTURE OF MEDICINE. By Sir JAMES MACKENZIE, F.R.S., M.D., F.R.C.P., LL.D.Ab. and Ed., F.R.C.P.I.(Hon.). Pp. 288. Price 8s. 6d. net. London: Henry Frowde, Hodder and Stoughton, 1919.

No detailed analysis of the contents of this latest book by Sir James Mackenzie can give a prospective reader an adequate conception of its worth. A book, which is in the main a criticism, will have its own critics without in any way detracting from its value which lies in its power to stimulate thought almost as much as in the facts or figments it may contain. The first section of the book is under the title of "Criticism," and the author opens his subject with an exposition of the difficulties that beset the path of the investigator in his attempt at the solution of the problems of medicine. He contends that these difficulties have been enhanced rather than lessened by the present trend of medical teaching, and that by want of co-operation between workers in the various fields of research we are tending to lose our way in a maze of blind alleys, with the result that not only the student but more advanced workers find themselves bewildered and aghast at the apparent complexity of the problems confronting them. This apparent complexity is aptly illustrated by the setting out at length on pp. 35-39 of the syllabus recommended by the Professor of Clinical Medicine at Johns Hopkins University. Its perusal leaves one breathless, but with the strong conviction that study along these lines could only end in disaster. Part II. is devoted to the narrative of the author's personal experiences. He relates his early dissatisfaction with the lack of knowledge requisite for the solution of the many problems that confronted him, and shows how the recognition of the causes of this limitation enabled him to succeed in solving some of the problems in that branch of medicine to which he later devoted himself. This success was attained not by elaborate details of technique but by the recognition of principles which could be applied to other branches of medical research equally fruitfully. That laboratory instruments are necessary in the study of certain problems, and aid in their solution, the author readily admits, but pleads that they should be used as aids to, and not as substitutes for, our faculties of sight and touch. In the last section, entitled "Constructive," suggestions as to the methods of training and research which are likely to advance our knowledge are given. The unique position of the general practitioner as an agent for the furthering of medical knowledge is pointed out and stress is laid on the necessity of granting him every assistance at the outset of his career, in order that he may be able to use his opportunities to the full. Unfortunately the present-day tendency seems to be to forget the fact that the human organism is a unit and maintains and expresses itself as such, and the practitioner's activities are being curtailed by those who would divide up the body into the watertight compartments of specialism.

SURGERY OF THE LUNG AND PLEURA. By H. MORRISTON DAVIES, M.A., M.D., M.C.Cantab., F.R.C.S.Eng., Hon. Captain R.A.M.C.(T.) With 2 coloured plates and 79 other illustrations ; pp. xxiii + 259. Price 25s. net. London : Shaw and Sons, 1919.

This is a very clear and well illustrated account of the latest surgical methods applied to the treatment of diseases of the lung and pleura. The book is based for the most part on the author's own experience and throughout leaves the impression that he has tried and proved the various procedures and only advises when thoroughly convinced himself. The first three chapters deal with anatomy, the physiology of intrapleural pressures, and some special points in diagnosis and prognosis. One chapter is devoted to diseases of the pleura and another to injuries of the lung and pleura. In Chapter VI a rather sketchy account of bronchoscopy is given. The last seven chapters deal with surgery of the lung under the headings—gangrene and abscess, tuberculosis, bronchiectasis, streptotrichosis, hydatid disease, tumours, and emphysema. So far as we are aware this is the only thoroughly up-to-date book in the English language dealing with lung surgery. The subject has been comparatively neglected and it is largely due to the author's enthusiastic work that lately attention has been focused on this branch of surgery. The author recommends aspiration with oxygen replacement as the first line of treatment in pneumococcal empyema, and later pleurotomy with immediate suture of the wound and oxygen replacement. Temporary drainage is recommended only when fever due to toxic absorption persists. Four different surgical procedures applicable to pulmonary tuberculosis are described—nitrogen displacement, rib mobilization, section of the phrenic nerve, and displacement by foreign bodies. The indications for performing these operations are given, but very few case-records are inserted. The book is well produced, and contains a large number of radiograms illustrating the various conditions. Operations are described but not figured. Both physicians and surgeons will find much in this treatise to stimulate thought and to challenge their acceptance of old-fashioned methods of treatment. The chest has always been regarded as the peculiar province of the physician but this book opens up a new field to the surgeon and should lead the physician to collaborate more often with his surgical colleagues.

William Howard Lister. By Walter Seton. With a Foreword by Lieutenant-General Sir Ivor Maxse, K.C.B., C.V.O., D.S.O. Portrait. Pp. 93. Printed for Private Circulation by Philip Lee Warner, Publisher to the Medici Society, Ltd., London, 1919.

This is a sympathetic record of the short life of Dr. W. H. Lister, D.S.O., M.C., of University College, who, like so many young medical men, gave his life for his country. In his thirty-one years he had many experiences and was remarkable for his enthusiasm and vitality. Early in his student days he was the moving spirit in the protest against the action of the anti-vivisectionists, which underlay the destruction of the bronze statue of the Brown Dog on a drinking fountain close to Latchmere Gardens, Battersea. He was one of the best known undergraduates of his time in the University of London, and spared no pains in schemes for advancing its corporate spirit; thus he was a keen member of the committee and later Vice-President of the Union Society of University College, Assistant Honorary Secretary of the University of London Athletic Union, and did all that was possible to start a University Boat Club. He threw himself into the establishment of University College Hall and energetically interviewed all who were likely to help, as is shown in the following episode: "The idea suddenly occurred to him on April 1, that he would make a personal appeal on behalf of the Hall to Mr. Joseph Storrs Fry, of Bristol, a close friend of his grandfather's. He set out by the next train, and found Mr. Fry, who received him kindly and listened to him patiently. Mr. Fry told him that he had not any money to put into the Hall scheme, whereupon Lister immediately offered to lend him the necessary funds to enable him to apply for shares. Mr. Fry did not accept the offer, but prayed with his visitor, who then departed to Buxton somewhat disappointed!" Another evidence of his intense interest in London University is his evidence before the Royal Commission on University Education in London. In 1912 he went out as a dresser to the Balkan War, and qualifying in the next year saw more of the world as Medical Officer on R.M.S. *Cobequid*, and did heroic service in its wreck. Directly on the outbreak of war he joined the R.A.M.C. and was severely wounded in October, being recommended for the V.C. His record during the war was a marvellous one for gallantry; he was invalided eight times, decorated by the King on three occasions, and was made a Fellow of University College under the clause in the College statutes which provides as one ground for election "distinction in public life." Shortly before his death on August 9, 1918, from a bomb, he wrote "I only pray that I may be able to do my duty without shrinking even if with fear." It is only fitting to add that this memoir is charmingly printed and bound.

The Practitioner's Manual of Venereal Diseases, with Modern Methods of Diagnosis and Treatment. By A. C. Magian, M.D. With 61 illustrations; pp. viii + 215. Price 10s. 6d. net. London: William Heinemann (Medical Books), Ltd., 1919.

Designed as a practical handbook for the general practitioner this book sets forth in detail the signs, symptoms, and treatment of the three venereal diseases. In the case of gonorrhœa and syphilis, a short summary of the disease is followed by chapters which deal in detail with their various manifestations and their appropriate treatment. The necessary bacteriological and serological examinations are explained with practical instructions. There are copious illustrations, chiefly of instruments and apparatus used. The questions of cure and subsequent marriage are fully dealt with, and at the end of the book are reprinted specimen pamphlets to be given to all persons under treatment for venereal diseases.

The Exact Diagnosis of Latent Cancer: An Enquiry into the True Significance of the Morphological Changes in the Blood. By O. C. Gruner, M.D. With 19 illustrations; pp. vii + 79. Price 7s. 6d. net. London: H. K. Lewis and Co., Ltd., 1919.

The author claims that it is possible to detect the presence of cancer, even if only in its incipient or precancerous stage, by an examination of the blood alone, and to decide whether an operation for the removal of a cancer has or has not been completely successful. He lays stress upon the investigation of live as well as of dried blood. The usual routine examinations are regarded as far too meagre. Not only colours, numbers, and differential counts are noted, but the consistence, colour, and texture of fresh blood; the consistence, attitude, construction, agglutinability, contour, and types of aggregations of red disks and other refinements, are regarded as of great importance. The suspended matter, fibrin content; the size, shape, construction, contours, motility, agglutinability, and intimate structure of the white cells and of their nuclei; their mode of death, as well as associated changes are all subjected to examination and classification. Interpretations, methods of procedure and of recording are gone into and illustrative cases are given. The thesis is illustrated by 19 figures.

INTERNAL MEDICINE: A WORK FOR THE PRACTISING PHYSICIAN ON DIAGNOSIS AND TREATMENT. By NATHANIEL B. POTTER, M.D., JAMES C. WILSON, M.D., and Professor NORBERT ORTNER. In three volumes, with complete Desk Index in separate volume. With 14 coloured plates and 427 other illustratons; pp. xli + 2333. Price £3 15s. net the four volumes. Philadelphia and London: J. B. Lippincott Co., 1919.

This work, which has now reached its fifth edition, represents a complete treatise on internal medicine in three volumes. It stands, as it were, midway between the elaborate "Systems" of Medicine and the ordinary one volumed text-book, and is suited more to the needs of the practitioner than of the student. The first volume deals with methods of examination and the diagnostic value of symptoms; the second describes diseases systematically in the same way as the usual text-books, whilst the third volume is devoted entirely to therapeutics, which are discussed much more fully than in the ordinary systematic treatise. There is an elaborate index, which has a separate volume to itself. The illustrations in the first and second volumes are numerous and clear, and in the present edition the whole subject-matter has been thoroughly revised and the newer knowledge in medicine and therapeutics incorporated.

THE BREAST: ITS ANOMALIES, ITS DISEASES AND THEIR TREATMENT. By JOHN B. DEAVER, M.D., LL.D., Sc.D., and JOSEPH MCFARLAND, M.D., Sc.D., assisted by J. LEON HERMAN, B.S., M.D. With 8 coloured plates and 277 other illustrations. Price 40s. net. London: William Heinemann (Medical Books), Ltd., 1918.

The whole subject of diseases of the breast needs reconsideration from an original standpoint and in the light of modern scientific knowledge. The old terms must be ruthlessly discarded and a fresh classification adopted, for at the present time there is a superfluity of nomenclature and a maddening system of cross division. Professors Deaver and McFarland show that no less than twenty-four different names have been given to cystic disease of the breast and at least fifty-four to mammary cancer. Statistics are useless with such an exuberant nomenclature, whether they deal with the frequency of different forms of disease or with the even more important questions of recurrence and mortality, for no two writers discuss the same conditions under the same heading. Professors Deaver and McFarland have not entirely divested themselves of the old hindrances to progress, but they have produced a comprehensive book which is of great service to those interested in the subject and have made the way easy for further advance by means of their excellent bibliographical references. The microscopic and naked eye appearances of the different conditions are carefully described and the descriptions are elucidated by many well executed illustrations, eight of which are coloured. As a second edition is certain to be required it is well to note that greater care is required of the proof reader; thus on pp. 587, 589 axillary is spelt "axiliary"; whilst scirrhus is sometimes spelt "schirrus" and at least once "sirrhus." Fig. 215 on p. 596 serves no useful purpose and should be omitted. It would perhaps be too much to ask that some better synonyms should be found for "periductal," "Bosselation," "matricial," "metastizes," and "prognosing." But the book is so good and useful that verbal errors are venial.

FRACTURED FEMURS: THEIR TREATMENT BY CALLIPER EXTENSION. By MAURICE G. PEARSON, O.B.E., M.B., B.Sc.Lond., F.R.C.S., and J. DRUMMOND, M.D., M.R.C.P. Edin. With 57 illustrations; pp. xii + 92. Price 10s. 6d. net. London: Henry Frowde, Hodder and Stoughton, 1919.

The system described in this book allows of movement of the knee from the beginning of treatment with advantage to the fractured bone and to the wound in the soft parts by methods evolved during the war. In the last nine months of the war there were 5,000 new femur cases in the British Army in France. Many cases were moribund from tetanus, gas-gangrene, &c., when they reached the clearing stations. A whole system of transport and treatment had to be organized. Thomas's splint came into general use, orderlies being trained to put them on in four minutes (fig. 1, p. 10). At the clearing stations prompt and extensive operation came into use early in 1917. Fractures are considered in detail in three anatomical groups — upper, middle and lower thirds. The patient's highest resistance for early operation is rarely later than three hours after his arrival at hospital. In shell and bomb wounds the aim is to excise all damaged tissues, attached fragments of bone being removed, and the length of the limb maintained however much bone had been shot away. Fixation of fragments by a single wire brought out of the wound was used when needed. How to avoid secondary hæmorrhage is discussed. The method of using the Thomas splint is fully given, and the mechanical principles involved are explained. Every point is clearly illustrated: methods of treatment, post-operative treatment and results. The authors found Besley's traction-callipers effective.

THE AMŒBÆ LIVING IN MAN: A ZOOLOGICAL MONOGRAPH. By CLIFFORD DOBELL, M.A., F.R.S. With 5 plates; pp. viii + 155. Price 7s. 6d. net. London: Published for the Medical Research Committee by John Bale, Sons and Danielsson, Ltd., 1919.

Although dealing with the subject chiefly from a zoological standpoint, this volume well deserves the attention of medical men, and especially of pathologists who are concerned with the differential diagnosis of amœbic infections. After a short description of the best methods of examination, the author gives a brief historical survey and a general account of the present state of our knowledge. The genera and species of amœbæ are then considered in detail, and in an important synopsis (p. 19) the author recognizes only four genera (which include six species) of amœbæ which can be definitely accepted as occurring in man. They are named in strict accordance with the rules of zoological nomenclature. Of these species the author is of opinion that there is satisfactory evidence of pathogenicity only in the case of *Entamœba histolytica*. The morphology, occurrence, and effects of treatment on the individual species are then described, a short discussion of "Urinary Amœbiasis" is given, and lastly an account of amœbæ in dogs and monkeys, and of doubtful amœboid organisms. The bibliography, which has been compiled with great care and a series of excellent coloured plates, are valuable additions to the work of one who is admittedly a leading authority on the subject. A noticeable feature in this publication is the criticism, for the most part adverse, of the work of past and contemporary experimenters.

DEFECTIVE HOUSING AND THE GROWTH OF CHILDREN. By J. LAWSON DICK, M.D., F.R.C.S. Pp. 94. Price 3s. 6d. net. London: George Allen and Unwin, Ltd., 1919.

The author's pen has been guided through this little book by a praiseworthy endeavour to explain the claims of the children of the working classes for the provision by the State of better housing and a more healthy environment. To procure the latter Dr. Lawson Dick points out that there must be work for all at a living wage, with adequate provision against ill-health, unemployment and old age; and a dwelling, at once sanitary and comfortable, preferably in some outlying district, with facilities for fresh air, exercise and sunlight. Passing in review the principal stigmata of rickets, to which disease most of his observations have been directed, he arrives at the conclusion, drawn from his experience and supported by statistics, that while defective housing is the essential, deficient feeding is the contributive factor, and that, whether the food be good or bad, overcrowding and slum conditions, with the evils they entail, will inevitably produce that disease. As showing the effect of housing upon the incidence of pulmonary tuberculosis, he cites Dr. Maxwell Wilson's striking statistics relating to Edinburgh in 1912: one room, 6·9; two rooms, 5·6; three rooms, 3·5; four rooms and over, 1·4 (per 1,000 inhabitants); and shows that in 1911 one-sixth of the total population of the kingdom lived in houses of less than four rooms, adding as a corollary that "the remedy is not to build sanatoria, but to demolish slums."

APPLIED ANATOMY: THE CONSTRUCTION OF THE HUMAN BODY CONSIDERED IN RELATION TO ITS FUNCTIONS, DISEASES AND INJURIES. By GWILYM G. DAVIS. Fifth edition. With 631 illustrations: pp. x + 630. Price 30s. net. Philadelphia and London: J. B. Lippincott Co., 1918.

The fifth edition of this work contains a few additions to the text and illustrations to bring it up to date. The figures are finely reproduced and in character and number are almost sufficient for a systematic treatise on pure anatomy. The text deals with the applied anatomy of the whole body, being particularly full in relation to all surgical procedures.

THE MEDICAL REPORT OF THE RICE EXPEDITION TO BRAZIL. By W. T. COUNCILMAN and R. A. LAMBERT, M.D. With 35 illustrations; pp. vi + 126. Price 5s. 6d. net. Cambridge, U.S.A.: Harvard University Press; London: Humphrey Milford, 1918.

The expedition, the medical report of which is contained in this little volume, was organized by Dr. A. Hamilton Rice, primarily for geographical research. The main portion is devoted to a description of the general conditions of health in the three chief cities of the Amazon (Pará, Manaos, and Iquitos), and in the small towns and villages on the Rio Negro (a tributary of the Amazon) as far up as San Gabriel. The climatic conditions in the Amazon valley, the character of the land and vegetation on the river banks, and the mode of life of the inhabitants, are also briefly dealt with. It is pointed out that the insect pests, especially the micuims, the piums, and the Tucandera ants, are the real enemies of man in these regions, owing to their ubiquity, and to their particularly annoying and ferocious nature. So far as can be ascertained, however, none of these transmit disease. The chief cause of the country's lack of development, according to the authors, lies in the prevalence of malaria in *chronic* form. Mosquitoes are, however, apparently scanty in number, and the statistics as to the frequency of acute malaria in the cities is considered unreliable. After malaria, the two conditions which receive special notice are "splenic enlargement" and "ulcers." Although some of the cases of the former are probably due to malaria and to kala-azar, the authors believe that many of them belong to the obscure condition known as tropical splenomegaly. Ulcers are very common, and their ætiological differentiation is in a very elementary state. There is here ample scope for scientific investigation, if more hospital and laboratory accommodation were available. The endemic diseases of the Indians are briefly mentioned, and it is remarked that the native population is on the decrease. A short account is also given of the health of the inhabitants of Barbados and Porto Rico, and suggestions are offered for social and hygienic reforms, which are greatly needed for the proper development of those countries.

HEREDITY. By J. ARTHUR THOMSON, M.A., LL.D. Third edition. With 9 coloured plates and 38 other illustrations; pp. xvi + 627. Price 15s. net. London: John Murray, 1919.

The whole range of this most complex subject is gradually unfolded; its experimental basis considered; the theories advanced in its explanation are stated and criticized. Evolution and nurture play their part in hereditary reproduction but the influence of the development of germ and sperm—discontinuous growth of Haeckel—and the subtle effects of amphimixis are traced. Thus ancestral inheritance, with paternal and maternal influence, is studied. Huxley's prophetic utterance that the produced offspring is an organism comparable to a web knit of maternal warp and paternal woof is investigated and the parts played by the centrosome, the chromosomes and segmentation nucleus are advanced. Inheritance, though dual, is strictly multiple. Variation, an ambiguous term, may be continuous or discontinuous, and the theory that there are modifications in individuals of a type is closely pursued. De Vries' important work and the theory of mutation advance the unfolding of the schema of hereditary changes as we know them. Specific inertia and stability of specific characters, even racial inertia, hold to some extent for mental characteristics. Like need not and does not in all cases produce like, but always tends to do so. There is latent influence, i.e., recessive, and dominance fluctuating according to conditions which the author examines. Galton's and Weismann's views on transmission of acquired characters come under review. That Mendel's teaching, though modified by recent research, throws much light upon inheritance or transmission through dominating ancestors is shown. Thus the offspring is a mosaic of past generations the characters of which are determined by complex interactions. There is a full exposition of the history of the subject, of the work of Darwin, Weismann, Galton, de Vries, Bateson and other investigators; their views are collated, discussed, and summaries of conclusions arrived at are supplied, which render clear the complex problems considered. We find fertilization, theories anent development, germinal selection, determination of sex, thoroughly discussed. In later pages we are shown the relations of biology and sociology and the social significance of heredity. Much that appears in earlier chapters is more fully elaborated; a full bibliography is appended.

ANÆSTHESIA AND THE NURSES' DUTIES. By A. DE PRENDERVILLE, LL.B., M.R.C.S. With an Introduction by Sir JAMES CANTLIE, K.B.E., M.B., F.R.C.S. With illustrations; pp. xii + 100. Price 3s. 6d. net. London: William Heinemann (Medical Books), Ltd., 1919.

The introduction by Sir James Cantlie eulogizes the author of this posthumous book. The writer advances reasons why nurses should be instructed about the administration of anæsthetics, and sets himself the task of teaching the essential duties involved in the efficient carrying out of this procedure. Such whilom means of inducing insensiance to pain as the use of opium and other drugs—e.g., mandragora, alcohol, pressure upon nerves, chilling by ice and salt, and the practice of hypnotism are mentioned. A brief résumé of the history of anæsthesia also finds a place in these pages, which takes us from the days of Humphry Davy to our own. The conditions affecting the patient, whether due to age, sex, disease or brought about by operative measures, are discussed and divided into the categories which are conducive to the production of favourable or unfavourable anæsthesia. Nurses are instructed in the proper preparation for the anæsthetic and the after-care of the patient as he recovers consciousness. Succeeding chapters deal with the phenomena to be noticed during anæsthesia—how to recognize the onset of danger and how to deal with it when it arises; the preparation and cleansing of apparatus; and such special technique as is called for in the case of special branches of surgical procedure.

THE GREAT WAR AND THE R.A.M.C. By Brevet Lieutenant-Colonel F. S. BRERETON, R.A.M.C. With 9 maps, 1 plan, and 2 appendices; pp. xv + 300. Price 14s. net. London: Constable and Co., Ltd., 1919.

This, the first volume of the "Popular Medical History of the War," deals with the military operations from Mons to the Aisne, and the organization of the R.A.M.C. during the opening weeks of the campaign. Colonel Brereton has had access to official documents, and his narrative, prefaced by the Director-General A.M.S., is therefore authoritative. He describes minutely every stage of the Retreat, and the Advance on the Marne, mentioning in detail, with the aid of maps, the various phases as they developed. Although the Army Medical Department had repeatedly and urgently recommended in peace-time the provision of motor ambulance convoys, these were lacking in the critical weeks of August-September, 1914, and the absence of these for the rapid evacuation of casualties, caused the lines of the retreat to be blocked by slowly moving transport. This state of affairs was remedied none too soon, when, by September 20, a fleet of motor ambulances under R.A.M.C. control replaced the lorries, which too often were beds of agony for men gravely wounded. At the same time the author commends the establishment of casualty clearing stations, which at a later stage were brought to a high degree of efficiency. The writer's historical sense is so strong that one has to read between the lines of his unimpassioned account to realize the appalling conditions under which the R.A.M.C. had to work, and the ordeals to which they were hourly subjected. The book contains in an appendix the Official Report on the typhus epidemic at Wittenberg Camp. The Report calls for the indictment of the infamous Oberstabarzt Dr. Aschenbach, whose calculated inhumanity to the prisoners in the stricken camp won (?) for him the Iron Cross. Further volumes of this history will be awaited with interest by all arms that took the field in the first months of the war.

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NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

Sir Victor Horsley: A Study of his Life and Work. By Stephen Paget. With 12 full-page plates and 17 other illustrations; pp. xi + 358. Price 21s. net. London: Constable and Co., Ltd., 1919.

Sir Victor Horsley died in his fifty-ninth year from heat stroke at Amarah in Mesopotamia on July 16, 1917, and in little more than two years Mr. Stephen Paget's study of this great personality has been published; it is written by an obvious friend and admirer who is fully conscious of the responsibilities of his task and is most anxious to avoid the natural bias that is almost inevitable in these circumstances. The difficulties inherent in making a just and lasting estimate of a great man within a short time of his death are always great, and they are particularly so in dealing with such a many-sided man as Sir Victor Horsley, but this is certainly an exceptional biography and as far as can be judged at present a correct perspective has been attained. Lady Horsley, to whom the life is dedicated, in her prefatory note says that it would be hard to find two men more widely apart in their mental attitude than her husband and the writer of his life and that this constantly appears in the critical attitude of the biographer, but that no attempt has been made to suppress or soften it. The volume is divided into three parts, dealing with the first thirty years of his life, the remainder of his career in London, and his activity during the War; the division between the first thirty years and the rest of his life, which is gracefully described as "the summer and autumn of a life which had no winter," is comparatively easy, but the difference of the years 1914-17 is mainly one of place. He was born on April 14, 1857, the same day as Princess Beatrice and at Queen Victoria's desire was named after herself. His father, J. C. Horsley, the well known artist, came of a musical and artistic stock and was restlessly energetic, impulsive and hot tempered, but generous and quick to make amends. Mrs. J. C. Horsley was a sister of Sir Seymour Haden, the first President of the Royal Society of Painter-etchers and himself a surgeon and the son of a surgeon: she was strong-willed and extremely skilful with her hands. Sir Victor spent his boyhood in the country, where riding made him wish to be a cavalry officer but, as this was ruled out by parental considerations of expense, he decided to be a surgeon, not a physician, as "there was plenty of riding, driving and cutting about." He was a day boy at Cranbrook school and though in the sixth form he was not specially distinguished; Mr. Paget says that he ought to have gone to a public school far from home, but this might well have impaired his originality and made him more conventional. Before he was seventeen he went to University College to work for the University of London; he was at this time assertive and disputatious and early in his student career took up the temperance and anti-tobacco causes, his father as a non-smoker cordially agreed as to the evils of nicotine but was inclined to resent abuse of "God's good gift" of wine, an expression which particularly roused Victor's ire. After a brilliant student's career he qualified in 1880 at the age of twenty-three, having previously made the first of his numerous contributions to *Brain*. As a house surgeon he became attracted to cerebral surgery, and like so many distinguished men was at one time threatened with pulmonary tuberculosis. In 1882 he became surgical registrar and assistant professor of pathology at University College. From 1884 to 1890 he was professor superintendent of the Brown Institution, and there undertook three important lines of research, namely: (1) localization of function in the brain, and the pathology of epilepsy and canine chorea; (2) the thyroid gland, with special reference to myxœdema and cretinism; and (3) the protective treatment against hydrophobia, which are fully described in separate chapters. In 1886 he was elected a fellow of the Royal Society and surgeon to the National Hospital, Queen Square, where a year later he performed successfully the first operation for removal of a tumour from the spinal cord. The stirring events of the successive years are given with explanatory comments and impress the reader with his tireless energy. In 1885 he became assistant surgeon, in 1900 obtained beds, and in 1906 retired from the brilliant staff of University College Hospital, the position of which is compared to Balliol among the colleges at Oxford, and in 1902, to his great astonishment, he was knighted. As the years went on, his interest and participation in professional politics and schemes to improve the welfare of mankind steadily grew. A special chapter is devoted to his fight against alcohol, on which he had first lectured as early as 1882; it is suggested that his lectures on this subject might have been better for more frequent lighter touches, and one of those rare reliefs in which he described our three most British institutions, John Bull, Father Christmas and King Henry VIII, as examples of fatty degeneration due to chronic alcoholism is quoted. Mr. Paget places him on a par with Ambroise Paré, John Hunter, and Lister, but says that he had wider instincts, so that he could not rest in science and practice, but must also be in politics. With his ardent and fearless temperament he was impatient of expediency and inevitably trod on some sensitive corns and so gave offence; but he had the supreme gift of delight in the use of all his gifts and so was never idle, slack or at a loss for something to work at or fight for. To look at he was a man created for friendship and for happiness; he had the grand air, and the average man looked sadly commonplace by his side; his happy home life and his private practice are sympathetically described in the chapter immediately before the last section of his life, that during the War, which contains his efforts to get to the Front, his activities there, and the tragedy of his premature death.

DISEASES OF OCCUPATION AND VOCATIONAL HYGIENE. Edited by GEORGE M. KOBER, M.D., LL.D., Washington, D.C., and WILLIAM C. HANSON, M.D., Belmont, Mass. With illustrations; pp. xxi + 918. Price 32s. net. London: William Heinemann (Medical Books), Ltd., 1918.

In this comprehensive volume, by several writers, occupational diseases are dealt with in such a way as to make the book of interest to medical and legal experts and also to employers of labour. The experience is mainly drawn from America, Sir Thomas Oliver being the only contributor from the United Kingdom. The subject matter is grouped under three heads: Part I—Specific diseases of occupation: Intoxications, infectious diseases, dust diseases of the lungs, fatigue and occupation, and occupational diseases of the skin. Part II—Causation and prevention of occupational diseases and accidents. Part III—The relation of clinics, statistics, and legislation to occupational diseases. There is, in this part, a description of the Milan (Italy) clinic for occupational diseases by Dr. Luigi Devoto, the director. A useful and informing chapter is the one on "The Uses and Fallacies of Statistics." It is a book that can be very profitably studied, especially in relation to the present inquiry into the administration of the Workmen's Compensation Act. At the end of each section a comprehensive bibliography is inserted. In the last part a well drawn up list of industrial poisons, and another of "industries and poisons" will be found, together with occupational mortality and morbidity statistics.

DENTAL SURGERY AND PATHOLOGY. By J. S. COLYER, F.R.C.S., L.D.S. Fourth edition. With 920 illustrations; pp. xiv + 899. Price 32s. net. London: Longmans, Green and Co., 1919.

This is a much enlarged edition, which the increased knowledge of the last few years demands. It deals with ætiology and pathology rather than with the elaborate technique involved in dental operations, for, as the author states in his preface, "so-called conservative dentistry often retains sources of sepsis," and the general health of the patient ought to be the object aimed at. The book is truly British in character, dealing in fairness with antagonistic views, evidenced by the abundant references given, but fearless in the expression of the author's own convictions. The illustrations are abundant and well chosen. Abnormalities and irregularities of the teeth are attributed to diminution in the size of the jaws, "a characteristic developed in each generation as the result of the action of the environment" (Sim Wallace). The effects of mouth breathing and adenoids are fully discussed. The surgical and mechanical methods of treatment are extensively illustrated; 150 pages are devoted to dental caries, most of which is entirely new in the light of present day knowledge. The chemical changes. microscopical appearances and bacteriology are fully explained, and the various theories which lead up to the statement "that the prevalence of dental caries in modern races is due to the soft character of the food and the increase of carbohydrates which undergo rapid fermentation." The details of the "elaborate technique" of dental operations is not omitted in spite of the author's previous statement. The morbid anatomy and pathology of periodontal disease is illustrated also by comparative specimens. It is shown that pockets around the teeth are stagnation areas and that it is impossible to eradicate them; cures of periodontal disease are rare, but the progress of the disease is greatly influenced by the degree of resistance offered by the tissues. Treatment falls under two headings: local, by the dental surgeon and by the patient; general, by the physician or surgeon, under which the value of vaccine therapy is examined. The popular term for this disease is studiously avoided. The chapters on tumours, diseases connected with the teeth, diseases of the gums and oral mucous membrane, saliva, odontomes, various swellings and fractures of the jaws, should give the work a place in every medical library, for probably no other book contains so much reliable information on these everyday diseases of such far reaching importance.

A PHYSICIAN IN FRANCE. By Major-General Sir WILMOT HERRINGHAM, K.C.M.G., C.B. late Consulting Physician to the Forces overseas. With 9 illustrations; pp. viii+293. Price 15s. net. London: Edward Arnold, 1919.

Sir Wilmot Herringham went to France on October 20, 1914, and remained out until 1919, and for almost the whole of the time was at general headquarters with Sir Anthony Bowlby, a very happy association; he was first called to general headquarters to attend Lord Roberts in his fatal illness, and was with him when he died. His work, which was to go round the medical cases with the resident medical officers and to assist them in every way possible, was much the same as that of a hospital physician at home, except that he was not responsible for the treatment; but the most important duty of all seemed to him to be the stimulation of the medical officers to an interest in their work and the prevention of careless methods which the circumstances of war and the deficiency of scientific equipment in the clearing stations are apt to produce. While insisting on the cordiality with which the Royal Army Medical Corps received the civilian medical men, he is under the impression that at first the appointment of consultants was regarded merely as "eyewash," though eventually its utility was proved. In a general sketch of the R.A.M.C. the transformation that has taken place since his student days is outlined, and he discusses the difficulties attendant on the necessary expansion of the corps during the war. The organization in the field is described, especially the field ambulance and the clearing stations: though out of his official sphere a base hospital was visited by himself as a patient with sciatica, and there he was very much spoilt, partly an account of his rank, partly because he was a doctor, and partly in respect of his 62 years of age. To the critics of the R.A.M.C. who chiefly complained that there were more officers than were needed, he retorts that they were ignorant, sometimes amusingly so, of organization and did not realize how much larger a number of medical officers is required by a fighting force than by a civil population. Enteric fever, cerebro-spinal fever, trench fever, and shell shock are discussed in language that the lay reader can understand; the way in which medicine has been advanced during the war and the work of the Medical Research Committee are described, and he considers it certain that the German output does not compare with ours either in volume or in value. So much for the strictly medical part of the book, which, however, is on the whole rather less than that dealing with the life in France and reflections aroused thereby; thus the first four chapters, "The Surprise," "The Two Ideals," "The Two Tempers," and "The Stake," deal with the outbreak of war, and the contrast of the German and British nations and their aims. While full justice is paid to the thoroughness of German workers, the real glory of Germany is shown to be a sincere reverence for learning and for labour and a real comprehension of the dignity of both; and thus she can read us the greatest lesson for all, for while she worshipped work, we worshipped play. The last chapter repeats this note to the effect that we have lost the desire for work and the pride in working well, but the last sentence of the book ends more cheerfully in the belief that, although at present labour has lost its head, the men who saved Europe are not likely to ruin England.

MANUAL OF TROPICAL MEDICINE. By ALDO CASTELLANI, C.M.G., M.D., M.R.C.P., and ALBERT J. CHALMERS, M.D., F.R.C.S., D.P.H. Third Edition. With 16 plates and 909 other illustrations; pp. x + 2436. Price 45s. net. London: Baillière, Tindall and Cox, 1919.

This publication is so well known to all students of tropical medicine that a note on its contents is almost superfluous. Large portions of the book have, however, been rewritten, and new subjects have been introduced, so that the manual is thoroughly up to date. The mass of information contained in its two thousand odd pages is prodigious, and as a book of reference on tropical diseases, both from a pathological and a clinical aspect, it is undoubtedly of great value. The authors are aware that the volume is unwieldy, but they are of opinion that it is more convenient for the tropical practitioner than if it were divided into two parts. This point has already received notice in the various reviews which have appeared in the medical press. Much of the information contained in the book is based on the personal experience of the authors, and, as such, is naturally subject to debate. The volume is divided into three parts. The first part is of an introductory nature, and deals briefly with the history of tropical medicine, tropical climatology, foods, and diseases generally, and with the conditions which determine fitness for a tropical life. In the second part the causes of tropical diseases are discussed in detail. Practically every ætiological factor which is definitely known to have, or which may possibly have, any relation to tropical diseases, is touched upon. Chemical causes (poisons, poisonous foods, and venomous animals), physical causes (humidity, pressure, radiation, &c.), and parasitic causes are dealt with consecutively. The parasites are classified systematically, and are named in accordance with the international rules of zoological nomenclature. They include protozoa, worms, ticks, mites, &c., and the enormous and important disease-bearing class—insects. Of the vegetable parasites, the bacteria are described somewhat shortly, and chiefly from the point of view of indicating the systematic relationship of the various genera and species; an admittedly difficult task. The three chapters which deal with the fungi constitute a useful addition to the literature of the tropical practitioner who requires information on the fungal causes of disease. Professor Castellani is an authority on medical mycology, and a compilation from his pen on this subject must be worthy of every consideration for accuracy. In Part III, which forms the greater part of the volume, the authors describe at length the symptoms, treatment, and clinical features generally, of the various diseases of the tropics. They divide the subject into three sections, dealing respectively with fevers, general diseases, and systemic diseases. In the two first-named sections the diseases are classified according to their mode of origin (whether protozoal, bacterial, &c.) and are further arranged under the headings of the intermediate host or carrier by which they are transmitted to man. Among the systemic diseases, special attention is given to diseases of the alimentary tract, such as dysentery, cholera, sprue, &c., and to the skin diseases which are peculiar to tropical regions. Other diseases, however, receive ample consideration, and a useful bibliography is attached to each of them. The volume is well illustrated, and contains a number of coloured plates and a large index.

MEDICINE IN ENGLAND DURING THE REIGN OF GEORGE III. The Fitzpatrick Lectures delivered at the Royal College of Physicians, 1917-1918. By ARNOLD CHAPLIN, M.D.Cantab. Pp. 141. London: Published by the author, 1919.

The reign of George III covers a most interesting period in the general intellectual life of this country when liberty of thought based on free and scientific inquiry was dispelling the fixed doctrines of traditional authority; medical thought began to move at the same time, for, apart from Sydenham and Harvey, who were a century in advance of their time, the desire to appeal to facts ascertained by experiment had little influence on Medicine until the end of the eighteenth century; during the first half of this century Medicine was under the sway of various "systems" founded for the most part on metaphysical speculation and retarding rather than advancing a just conception of the subject. But early in the reign of George III Morgagni's great work, translated into English by Alexander in 1769, began to exert its influence in Britain and inspired Matthew Baillie and others. Dr. Arnold Chaplin describes at some length the conditions of the medical profession; in 1782 there appear to have been 4,459 medical practitioners in England and Wales with an estimated population of nearly eight millions, or one medical man to every 1,782 persons, as compared with one to 1,440 at the present time. Nor has the scale of remuneration altered much; physicians charged a guinea, which then had a far greater purchasing power, and a relatively larger number of physicians than now secured a handsome competence in those days. The College of Physicians, the Corporation of Surgeons, and the Society of Apothecaries had control over the London district, but in the provinces there were a large number of ignorant and irregular practitioners; this danger to the public weal led to the passing of the Apothecaries Act in 1815, and thus to an immense advance in the regulations for the control of the medical profession, for it at once placed the education of about three-quarters of those intending to practice under the control of the Apothecaries Society. The state of medical education in London and at the universities, the hospitals, the medical societies, and the medical press are discussed, and an account of the long drawn out contest between the Royal College of Physicians and its licentiates, in which William Charles Wells, the famous author of "An Essay on Dewd," took part, is given. A specially attractive feature of these lectures lies in the biographies of contemporary physicians, such as Thomas Young, Sir George Baker, William Heberden the elder, and especially of Wells, who, after a dire struggle with penury and ill-health, desired only the modest epitaph that "he had tried to extend the boundaries of knowledge." The medical work of Wells has been rather forgotten, and few now realize that he to some extent anticipated Blackall and Bright in the recognition of albuminuria, and that Darwin gave him the credit of having first enunciated the principle of natural selection. The lectures contain accounts of the illnesses of George III. Napoleon, Samuel Johnson, and Charles James Fox, and the review of Medicine during this period is completed by consideration of its relations to the State, such as the treatment of the insane, of prisoners and of paupers, the rise and effects of vaccination, the inception of sanitary science, and the development of the medical services of the Royal Navy and Army.

PSYCHOSES OF THE WAR: INCLUDING NEURASTHENIA AND SHELL SHOCK. By H. C. MARR, Lieutenant-Colonel R.A.M.C. Temp., M.D. With 60 illustrations; pp. xiii + 292 + 38. Price 16s. net. London: Henry Frowde, Hodder and Stoughton, 1919.

The title of this work is perhaps somewhat misleading, since the contents are those mainly of an ordinary text-book on mental diseases but with clinical illustrations mostly taken from Service cases. After a very brief reference to the borderland of mental disease, the anatomy and histology of "the seat of mind" is dealt with, followed by the various means of observation of mental phenomena, the classification of mental affections, malingering and feigned insanity. Neurasthenia and psychasthenia are then discussed, and lastly the various psychoses. An appendix on the examination of the cerebro-spinal fluid is added, as well as some pages on mental case-taking. The book is fully illustrated. It is at once evident that the author is not in any way in sympathy with modern psychiatric trends of thought, and Bolton's theories on mental pathology are seemingly accepted without question. There are many points to which exception might be taken. Much doubt might be expressed concerning some of the writer's nosological grouping. Hysteria is regarded as a symptom-complex of neurasthenia, and the so-called functional psychoses come under the heading of "mental enfeeblement (adolescent)." The statement that "in insanity, loss of mind and wasting of cerebral tissue go hand in hand" can surely hardly be supported, while statistics by other psychiatrists in no way confirm the author's findings, and certainly not his statement that 42·31 per cent. of the soldiers admitted to mental hospitals were found to have been weak-minded from birth! Much in the book is very far removed from modern teaching, and this naturally detracts greatly from its value.

THE ESSENTIALS OF CHEMICAL PHYSIOLOGY. For the use of Students. By W. D. HALLIBURTON, M.D., LL.D., F.R.S. Tenth edition. With coloured plate and 71 other illustrations; pp. xi + 324. Price 12s. 6d. net. London: Longmans, Green and Co., 1919.

As the title implies, this handbook mainly deals with the chemical composition of the body and the part played by its various constituents in carrying on the phenomena of life, but it necessarily also includes the closely allied subject of physiological chemistry, which is a branch of organic chemistry and treats of the composition and reactions of physiological substances. The work consists of two courses—the one an elementary and the other an advanced course, the order in which the subjects are treated being the same in both. The instruction is partly practical, partly theoretical, and is clear and up-to-date; thus vitamines and war margarine are duly considered. There is, in addition, an appendix containing descriptions of various laboratory instruments and of certain methods of research which should always be shown in demonstrations, though there may be practical difficulties in allowing each student to perform the experiment. There is much to interest the qualified man; under blood the difficult subject of immunity is described in a refreshingly lucid manner and in the simplest language. There is a useful index.

MENDELISM. By REGINALD CRUNDALL PUNNETT, F.R.S. Fifth edition. With 8 plates and 52 other illustrations; pp. xv + 219. Price 7s. 6d. net. London: Macmillan and Co., Ltd., 1919.

Gregor Mendel's work, published in 1865, opened up new lines of thought, and although almost unnoticed for nearly forty years, led to a vast number of experiments, while it provoked much constructive criticism. The present book deals with the problem which Mendel set himself to solve, and the contributions made to this end by subsequent workers. The association of Mendelism with the many questions raised by the study of heredity is obvious, but, until the work done with the fruit-fly Drosophila revealed the intimate relation of the chromosomes of the living cell with the phenomena of heritage, researches seemed mere patchwork, and their knitting together into a homologous whole a matter of almost insuperable difficulty. As in Darwin's pronouncements working hypotheses were requisite in post-Mendelian schemata. Professor Punnett details the apparent rifts in the orderly procession of reasoning from hypothesis to experiment, and from experiment to proof. We are introduced to Mendel's work with *Pisum sativum* and kindred plants, while succeeding pages reveal how the interaction of factors explains many difficulties. Reversion, dominance, variation in wild forms and domestic varieties are considered. One of the most puzzling questions at issue is sex, and to this two long chapters are devoted. The principles of Mendelian heredity are involved in economical procedures, such as breeding of cattle and floriculture, and it is believed that they control the problems which man has to face. Practically all these aspects find exposition in Professor Punnett's volume, and much that was vague when Mendel left his work is now shown to be clear and convincingly true.

Diseases of the Ear in School Children: an Essay on the Prevention of Deafness. By James Kerr Love, M.D., F.R.F.P.S.Glas. Pp. viii + 94. Price 5s. 6d. net. Bristol: John Wright and Son, Ltd., 1919.

In continuation of the work with which his name has been so long associated, Dr. Love here gives some further fruits of his experience in dealing with cases of ear disease. He holds that "nearly all the middle-ear disease of later life, both suppurative and non-suppurative, commences during the earlier years of the school period," and he shows the importance, and, incidentally, the convenience, of aural school clinics in providing the necessary safeguards. An account is given of the clinics established by the Glasgow School Board and the Secondary Education Committee of the County of Dumbarton. A chapter is devoted to the part played by syphilis in causing deafness, and Dr. Love suggests that the effects both of this disease and of measles are apt to be under-estimated. He inclines to the opinion that hereditary deafness is dependent on the existence of one or more recessive Mendelian characters, and finds in the fact that "infectious diseases or wrong marrying"—both preventable conditions—include most of the causes of deafness ground for hoping that the disease is capable of being almost entirely eliminated. Some general considerations of treatment are scattered through the book, and it may be noted that a conservative attitude is taken up in regard to the mastoid operation, though it is held that cases following the exanthemata should not be allowed to leave hospital until by this or other means all discharge from the ear has been arrested.

The Pituitary: a Study of the Morphology, Physiology, Pathology, and Surgical Treatment of the Pituitary, together with an account of the Therapeutical Uses of the Extracts made from this Organ. By W. Blair Bell. With 190 illustrations; pp. xx + 329. Price 30s. net. London: Ballière, Tindall and Cox, 1919.

The first 78 pages consists of the anatomy and comparative anatomy of the pituitary body and contains original work on the comparative anatomy from the lowest to the highest vertebrates. The next 145 pages deal with the histology and physiology of the gland. Experiments by the author are described showing the effect of the complete removal of the pituitary body in dogs, the partial removal of the anterior and posterior lobes, and the removal of the anterior and posterior lobes separately. The inter-relation between the other ductless glands and the ovary and uterus are discussed. with experiments by the author, with illustrations. A full account is given of the diseases of the pituitary and the associated symptoms, including effects on the optic tract, followed by the medical and surgical treatment of pituitary lesions. The last 26 pages are devoted to the therapeutical uses of the gland in general medicine, obstetrics and surgery. The work is admirably arranged and profusely illustrated and forms a valuable addition to the literature of the subject.

The Forms of Alcoholism and their Treatment. By Hugh Wingfield, M.A., M.D., B.C.Cantab. Pp. vi + 76. Price 5s. net. London: Henry Frowde, Hodder and Stoughton, 1919.

In considering the problems of alcoholism it is pointed out that drunkards, although regarded by the laity as simply vicious, are weaklings and victims, more than offenders, whose excesses are often due to environment or compulsion. In some cases hereditary tendency dominates while resistance is recessive. Sir Archdall Reid's views are quoted as suggestive of an acquired protection against alcoholism among habitual drinkers due in part to natural selection and in part to dying out of the unprotected. The effects of alcohol on the individual are next considered. The excessive euphoria and paralysis of the will makes for drunkenness. A careful examination is made of the forms of alcoholism in pseudo-dipsomania, possibly the primitive type, in which the craving is only excited by drink, the bouts are ended by physical disability; chronic sober alcoholism, when the patient regularly drinks to excess, but does not become drunk, and craving supervenes only on withdrawal of the drug; chronic inebriate alcoholism resembles the last of these, but the patient is never sober, and—true dipsomania

The factors making for alcoholism are: (1) euphoria, craving for the pleasure associated with drink; (2) dysphoria, a craving to counteract the misery of depression; (3) varieties of natural tolerance; and (4) partial paralysis of the inhibitory mental functions. To these must be added a fifth, viz., the condition provoked by complete abstention after prolonged continuous excess. The varieties of alcholism and their etiological factors are minutely discussed. Jauregg's theory of an anti-alcohol toxin is regarded as a useful working hypothesis, valuable as a guide to treatment. Compulsory restraint with segregation is discountenanced by the writer, who at some length describes mode systems of combating the drink disease. He insists upon recognition of the cause and type of each case before imposing alcohol restriction and the use of drugs. Hypnotic suggestion he regards as useful in suitable cases.

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SUPPLEMENT

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NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

The Urethroscope in the Diagnosis and Treatment of Urethritis. By Major N. P. L. Lumb, O.B.E., R.A.M.C.(T.C.). With 40 original illustrations; pp. xii + 51. Price 10s. 6d. net. London: John Bale, Sons and Danielsson, 1919.

Thanks to the illustrations, this book enables everyone in possession of a urethroscope to obtain a sound knowledge of the various conditions of the urethra and its diseases. The manipulation of instruments during observation and operations is first explained, then normal and abnormal appearances, finally there are indications when not to use as well as when to use the urethroscope. There are clinical notes and illustrations of progressive changes in the course of typical cases. Coloured plates similar to these should be accessible whenever instruction in venereal disease is given.

Vital Statistics: An Introduction to the Science of Demography. By George Chandler Whipple, Professor of Sanitary Engineering, Harvard University. Pp. xii + 517. Price 18s. 6d. net. New York: John Wiley and Sons, Inc. London: Chapman and Hall, Ltd., 1919.

The aim and scope of this book is well summed up in the statement in the preface that it makes no claim to be an exhaustive treatise or a compendium of facts, but is merely a guide to the study of vital statistics, and is written for students who are preparing themselves to be public health officials and for public health officials who are willing to be students. To such the book should be very valuable, as it gives a detailed and complete account of the elementary principles of statistical methods, including the tabulation, diagrammatic and graphical representation of data, the computation of birth-rates and death-rates, with their analyses and standardization, the construction of a life table, the theory of probability, correlation, &c., as well as repeated warnings against, and illustrations of, the common and not infrequent fallacies which are so liable to appear in the treatment of statistical data by the unwary. At the end of each chapter exercises and questions relating to the subject matter are given, and with these are associated in many cases references to books or papers from which full information on the subject may be obtained. A noteworthy feature of the book is the account of a plotting paper devised by Dr. Allan Hazen, called an arithmetic-probability paper, and somewhat analogous to the semi-logarithmic paper in which the percentage scale is so spaced that any set of figures which follows the natural law of probability plots out as a straight line, thus greatly facilitating the use of the theory of probability in statistical work. On p. 177 is to be found a very interesting and striking series of graphs compiled from the statistics of

Sweden from 1750 to 1900, illustrating the change in the age distribution of a population from year to year, where there is much variation in the birth-rate, a high birth-rate giving rise to a peak in a certain age-group in any one year causes a peak in a higher age-group a few years later, and this increase can be traced in the graphs throughout the whole period of life. The volume contains a useful list of references to works on the subject of statistics, as well as a table of logarithms for numbers up to 1,000. It should serve admirably the purpose for which it has been designed.

VENEREAL DISEASES: A PRACTICAL HANDBOOK FOR STUDENTS. By C. H. BROWNING, M.D., D.P.H., and DAVID WATSON, M.B., C.M. With an Introduction by Sir JOHN BLAND-SUTTON, F.R.C.S. With 25 coloured plates and 51 other illustrations; pp. xv + 336. Price 16s. net. London: Henry Frowde, Hodder and Stoughton, 1919.

The authors supply the essentials of knowledge necessary for students in the prevention and limitation of the ravages of venereal diseases. The response to early treatment is contrasted with the refractoriness manifested in later stages, and the information given is detailed in such a way that students at venereal clinics in which laboratory work is carried out will be enabled to correlate manifestations and carry out thorough treatment. The book indicates what amount of knowledge should be imparted to students in order to remove defects in teaching how to treat early syphilis and gonorrhœa in surgical lectures and departments. Secondary and cutaneous manifestations are considered to belong to the province of teachers of skin diseases, whereas later manifestations are to be regarded as appertaining to the departments of general medicine. Although teachers have individual methods of imparting instruction a merely superficial perusal of the ideas of others will enable them to make several diseases better understood by the profession as well as by the public.

DISEASES OF THE DIGESTIVE ORGANS: WITH SPECIAL REFERENCE TO THEIR DIAGNOSIS AND TREATMENT. By CHARLES D. AARON, Sc.D., M.D. Second edition. With 156 engravings, 48 Roentgenograms, and 9 coloured plates; pp. xlvii + 818. Price $7.00. Philadelphia: Lee and Febiger, 1918.

Professor Aaron's text-book is not restricted to disorders and diseases of the digestive system, but is a more general treatise upon the alimentary canal and its adjuncts and his selection is based upon an anatomical rather than upon a physiological or clinical differentiation. Thus in addition to the ordinary details relating to digestion and its disorders, ulcers of the intestine as a whole—catarrhal, tuberculous and typhoidal—all receive attention, and a chapter is devoted to the animal parasites of the intestine. Like most American works, the usual luxuriousness in production with prodigality of illustrations is to be noted. Yet the coloured illustrations portray such familiar details that one would have thought their expense was hardly justified, more especially as in one particular instance the coloration depicted by alizarin as an indicator in the analysis of test meals, is distinctly unconvincing. Whilst on the subject of test-meals it may be noted that the only method described in analysis is Töpfer's. The more elaborate methods which are common in this country do not seem to find favour in America. Dietetics in all its aspects, and diseases directly attributable to improper or diseased food are considered in great detail. It is interesting to note that of 274 cases of trichinosis investigated in America by Stiles, 208 were those of Germans. Intragastric technique is dealt with from every point of view, including lavage, massage and electricity, although the author quotes Freund to the effect that the influence of electricity on gastric secretion is absolutely negative. In all, 241 pages are devoted to investigation and general treatment before the formal description of diseases is undertaken. The author regards hyperchlorhydria as a condition in which the quantity of gastric juice is normal, but the percentage of free hyperchloric acid is higher than normal, or in other words, that hyperchlorhydria is distinct from hypersecretion. In the treatment of gastric ulcer he recommends the subnitrate of bismuth in preference to other salts, because of "the nascent nitric acid which is liberated and acts as a stimulant, astringent, and antiseptic." All the specific tests for carcinoma of the stomach are described in detail, and gastro-enteroptosis is very exhaustively considered, although it may be remarked that in these days of food restrictions the provision of lavish quantities of butter and cream recommended is hardly likely to be ratified. Duodenal exploration and alimentation, which are comparatively unrecognized in this country, are mentioned on several occasions, and recommended in the treatment of hepatic cirrhosis as well as of gastric ulcer. The technique is fully described ; and, since from the illustration appended the operation is being performed by a nurse, we conclude that it does not demand the peculiar skill which is generally regarded as associated with the practice. In the consideration of primary atonic constipation the condition with which we have long been familiar as "greedy colon" is described as a recent observation of Adolf Schmidt, Strasburger and Lohrisch. The author is a warm advocate of greater liberality than is customary in the treatment of typhoid fever, and the diet he permits might in comparison with the old régime be regarded as almost a banquet. He is also strongly in favour of the Ochsner method of delaying operation in cases of acute appendicitis, and states that it is entitled to the greatest consideration, and has been greatly misunderstood by the profession.

THE PHYSIOLOGY OF MUSCULAR EXERCISE. By F. A. BAINBRIDGE, M.A., M.D.Cantab., D.Sc., F.R.C.P., F.R.S. With 22 diagrams; pp. ix + 215. Price 10s. 6d. net. London : Longmans, Green and Co., 1919.

The author's object in this monograph is to present the subject in such a manner as to promote a closer connexion between the laboratory worker and those directly concerned with the health and well-being of the community ; thus, a fuller realization, and a more direct practical application by employers and administrators, of the physiological principles underlying the capacity of the body to perform muscular work would undoubtedly increase the output and improve the health of industrial workers. As muscular exercise is as much a nervous as a muscular process, its beneficial effects are seen in the psychical as well as in the physical life of the individual. Muscular exercise presents three problems : (1) The changes in the skeletal muscles, whereby the transformation of energy constituting muscular movement is effected ; (2) the nature of the adjustments occurring elsewhere in the body in order to provide the muscles with the oxygen and food-stuffs necessary for this purpose ; and (3) the means by which these are co-ordinated together in muscular exercise. These problems are considered in eleven chapters, each of which has a convenient summary. The discussion of the evidence as to the relative importance of carbohydrate and fat as the source of energy in muscular contraction shows that carbohydrate takes the first place, although there is reason to believe that fat is also utilized for this purpose to some extent. The rise of temperature during exercise, due to the increased oxidation of fats and carbohydrates, increases the efficiency of the circulatory and respiratory adjustments, and thus promotes the more efficient execution of muscular work. The causes of the rise of general blood-pressure and of dilatation of the muscular vessels, such as acid and other metabolites, are discussed. The share taken by adrenalin in bringing about the circulatory and other changes occurring during exercises, even when this is carried out under emotional stress, is comparatively small. The chapter on "Training" contains a physiological explanation of "second wind." The two concluding chapters are devoted to the after-effects of exercise. There is no direct evidence that exercise is ever harmful provided the individual is physically sound and in good health ; when bad effects follow, and these can almost always be referred directly or indirectly to the heart, as in effort-syndrome and overstress of the heart, there is a definite cause such as infective disease or a sedentary life. The view that the respiratory symptoms in effort-syndrome depend upon a sufficiency of buffer-salts in the blood is not accepted.

ESSENTIALS OF PHYSIOLOGY. By F. A. BAINBRIDGE, F.R.S., M.A., M.D.Cantab., D.Sc.Lond., F.R.C.P., and J. ACKWORTH MENZIES, M.A.Dunelm, M.D.Edin. Third Edition. With 179 illustrations; pp. viii + 484. Price 12s. 6d. net. London : Longmans, Green and Co., 1919.

This text-book of the Essentials of Physiology is designed primarily to meet the requirements of students preparing for pass examinations. The present (third) edition has been thoroughly revised and a few new figures added.

MANUAL OF DISEASES OF CHILDREN. By JAMES BURNET, M.A., M.D., M.R.C.P.Edin. Second edition. With plates and other illustrations; pp. ix + 416. Price 8s. 6d. net. Edinburgh: E. and S. Livingstone, 1919.

In the preface to this second edition the author says that "in connexion with children's diseases statements are handed on from author to author without any attempt being made to verify or refute them by personal observation and investigation." This statement is so generally true that it applies even to the work under consideration, and the therapeutics of childhood would be much simplified if all mere traditions were abolished. The association of rheumatic infection and heart disease in childhood is so intimate that it seems a pity to deal with them in separate sections. The important subject of infant feeding is discussed very fully, and the advice given on the knotty problems involved bears the mark of experience and judgment. Diseases of the alimentary system occupy a large section and the minor as well as the major maladies of this group are duly considered. New sections have been added on the infectious fevers and diseases of the skin, the nose, the throat, and the eye. Several types of disease are illustrated by good photographs.

THE OLIVER-SHARPEY LECTURES ON THE FEEDING OF NATIONS. A STUDY IN APPLIED PHYSIOLOGY. By ERNEST H. STARLING, C.M.G., M.D., F.R.C.P., F.R.S. Pp. ix + 146. Price 5s. net. London: Longmans, Green and Co., 1919.

These lectures deal with the food requirements of a Nation, as based upon the physiological needs of the average "man" in energy and protein, and with the methods, administrative and other, by which the required amount of food may be produced or distributed. The effects of deficiency of food on national health are also briefly reviewed. Tables are appended showing the average composition and calorie value of the more important foods and the average yearly quantity of the total food consumed in the United Kingdom before the War.

ESSAYS ON THE SURGERY OF THE TEMPORAL BONE. By Sir CHARLES A. BALLANCE, K.C.M.G., C.B., M.V.O., M.S., F.R.C.S., with the assistance of CHARLES DAVID GREEN, M.D., F.R.C.S. Two volumes, with plates and figures; pp. xxxvii + 612. Price £5 5s. London: Macmillan and Co., 1919.

Sir Charles Ballance's contributions to the surgery of the temporal bone are so well known and so highly appreciated by the profession that anything coming from his pen on this subject possesses more than ordinary interest. His recently published Essays on "The Surgery of the Temporal Bone" reflect the greatest credit on his studiousness, his extensive knowledge and his method of expression. The book before us is a *magnum opus* in every sense. It is lavishly illustrated, beautifully printed and neatly bound. Begun before the War, the Essays have been gradually compiled as time and opportunity in a busy and strenuous life afforded. In some respects this intermittency has been a misfortune, as the years which have elapsed since the Essays were first commenced have added a good deal to the sum of human knowledge and additions here and there would have made certain of the chapters more up-to-date, a fault, however, which the author will doubtless rectify in a second edition, which we have no hesitation in saying will soon be called for, if it has not been called for already. In a masterly preface, the author lays stress upon the half-hearted way with which aural surgery is regarded by many large and important British hospitals even to-day. The inadequacy of many aural departments in this country, the small number of beds assigned to the aural surgeon, the want of sufficient assistance, and in some places, efficient assistance, and the outstanding fact that aural surgery is not made a compulsory subject in the medical curriculum are blots on our acquaintance with the practical importance of diseases of the temporal bone. This great work, if read and mentally digested by the members of the General Medical Council, should serve to impress upon them the immense importance of making the study of diseases of the ear compulsory. The general historical survey as detailed in Chapters I and II is a mine of interesting information. Admirably compiled, it forms in itself an erudite review of what has led up to our present knowledge of the frequent and often fatal ravages of temporal bone sepsis. Chapter III dealing with important anatomical details is of extreme value to the student, and is replete with reference to the work of British and foreign authors. The chapter devoted to pathology is conspicuous from the wealth of information it contains. The paragraphs devoted to the consideration of thrombosis of the sinuses and of the diploëtic venules, a subject with which the author's name will for all time be linked, stands out prominently as a masterly contribution to the subject. To do anything like adequate justice to the various Essays would require a much more detailed criticism than is possible in a necessarily short review, Those interested in the practice of aural surgery will be well repaid by reading and inwardly digesting each chapter separately. In the reviewer's opinion, Chapters VIII, IX, X and XII are the best exposition of the particular subjects dealt with it has been his good fortune to read. The author, eminent as he is as a general surgeon, will doubtless go down to posterity as one of the foremost and most original of aural surgeons. The two handsome volumes should be in the possession of every library, and in the hands of every practising aural surgeon. In the compilation of the Essays, the author has been ably assisted by Mr. Charles David Green, to whom our thanks and congratulations are also due. The publishers, Messrs. Macmillan and Company, are to be highly commended for the general excellence of their work, and more particularly upon the excellence of the illustrations. Let it suffice to say that the work is a truly noble contribution to British surgery, and will ever remain a permanent memorial to the industry and intellectual capacity of one of the foremost surgeons of our day.

THE ART OF ANÆSTHESIA. By PALUEL J. FLAGG, M.D. Second edition. With 136 illustrations; pp. xviii + 367. Price 18s. net. Phildelphia and London: J. B. Lippincott Co., 1919.

The book presents the views on anæsthesia and analgesia current in the United States. It deals largely with the administration of ether by the methods now commonly adopted in this country: the open, the closed, intrapharyngeal, intratracheal, intravenous, and colonic etherization. Some of them are fully described; some receive a more cursory notice. The nitrous-oxide and oxygen method is extolled for major surgery, but it is pointed out that for various types of operation the addition of ether is advisable. Chloroform is dealt with very briefly; quoting the pronouncement of the Committee on Anæsthesia of the American Medical Association, the writer insists: "The use of chloroform as an anæsthetic for major surgery is no longer justifiable." Various forms of apparatus, many somewhat complicated, are described, but a chapter on emergency anæsthesia supplies hints for makeshift and simpler apparatus. Local analgesia and spinal anæsthesia receive some attention and the anoci-association plan is fully treated, and commented upon favourably. The opening chapters present a good deal of valuable information on general anæsthesia, posture, symptoms and signs of anæsthesia, how to avoid dangers and how to cope with them when they occur.

HANDBOOK OF ANÆSTHETICS. By J. STUART ROSS, M.B., Ch.B., F.R.C.S.E. With an Introduction by HY. ALEXIS THOMSON, C.M.G., M.D., F.R.C.S.E., and Chapters upon Local and Spinal Anæsthesia, by WM. QUARRY WOOD, M.D., F.R.C.S.E., and upon Intratracheal Anæsthesia, by H. TORRANCE THOMSON, M.D., F.R.C.S.E. With 54 illustrations; pp. xii + 214. Price 7s. 6d. net. Edinburgh: E. and S. Livingstone, 1919.

After giving a brief but comprehensive review of the physiological actions of the agents employed in narcosis the author deals with shock and anæsthesia and includes Crile's well-known views upon the subject. A useful chapter on anoxæmia follows, which takes in its purview the causes, prevention and treatment of asphyxia. Various methods of employing recognized anæsthetics are described and the question of the advisability of permitting rebreathing, which involves the views of Yandell Henderson on acapnia, are duly set forth. A section is devoted to the knowledge to be derived from the clinical observation of the patient and its practical bearing. The usual anæsthetics are discussed and routine as well as less common methods of employing them are detailed. Such important matters as the correct posture of the patient, the choice of the anæsthetic, dangers incident to anæsthesia with appropriate treatment, are all considered. Intratracheal insufflation is described by Dr. H. Torrance Thomson, while the subjects of local and spinal anæsthesia are epitomized by Dr. W. Quarry Wood. Three appendices deal with ether evaporation, percentages of ether in open methods, and the action of anæsthetics upon the blood.

NAVAL HYGIENE. By JAMES CHAMBERS PRYOR, A.M., M.D., Medical Inspector, United States Navy. With 153 illustrations; pp. 508. Price 12s. 6d. net. London: Wm. Heinemann (Medical Books), Ltd., 1919.

This manual, described as of an elementary character, for the United States Naval Medical School, is published with the approval of the Surgeon-General, U.S. Navy, and by permission of the Navy Department. It deals with hygiene of naval men in ships, submarines, in the air and ashore, is written tersely, and has 153 illustrations. After a short historical section, the development of naval architecture and its influence on naval hygiene are considered. Ventilation, heating, light, and he water supply next receive attention. The chapters on food and its inspection are illustrated by coloured figures of meat and contain much information; thus Buddeizing is a process of sterilizing milk recently employed along the Baltic: a small quantity of hydrogen peroxide is added to the milk, which is then subjected to a temperature of 122° F. for twenty minutes, thus driving off the peroxide and leaving the taste of the milk unimpaired. An account is given of the sick bay and the hospital ship. The special chapter on heat cramps contains the author's observations; under certain conditions not thoroughly understood, men in the fire rooms are attacked by painful cramps and show albumin in the urine; the treatment consists in hot baths, atropine, and, if necessary, morphine. In describing prophylaxis against venereal disease it is stated that while perhaps falling short of the extravagant claims of enthusiasts, carefully carried out personal prophylaxis undoubtedly reduces the incidence of disease by its antiseptic effect and by its educational results. The chapter on the disposal of the dead contains directions as to embalming and is followed by a glossary of nautical terms, and by an appendix on the physical examination of recruits which is illustrated by a number of finger prints.

LECONS DE PATHOLOGIE DIGESTIVE (QUATRIÈME SÉRIE). Par M. LOEPER. Pp. viii + 298. Price 11 fr. Paris: Masson et Cie, 1919.

This series of lectures on the pathology of the alimentary canal, the fourth that M. Loeper has published, is naturally based in great measure on data collected during the war. Although the war has not brought out any very remarkable discoveries in connexion with the alimentary canal it has greatly increased the incidence of the known forms of gastro-intestinal disease, and has multiplied the toxic and infective causes of abdominal disorder. A chapter is devoted to gastritis due to poisonous gases, of which the compound chlorine and ypérite are the most irritating. The immediate effects of gassing are vomiting and hæmatemesis, but the delayed effects are important and frequent, especially in chlorine gas poisoning, in which about 30 per cent. of the men exposed are thus affected, the indigestion being either painful or flatulent in type. As in other forms of "dyspepsia," the author shows by cytological examination of stomach washings that there is gastritis to account for the symptoms. The dyspeptic conditions caused by explosions may be indirect and due to disturbance of the brain and medulla, the vagus and sympathetic being involved, or due to direct action of force on the abdominal contents—namely, the viscera or their nerve supply. Another form of dyspepsia rendered prominent by the war is that due to suprarenal insufficiency; this is characterized by gastric atony and constipation; hypodermic injections of adrenalin are followed by improvement, increase in the gastric hydrochloric acid and more active peristalsis. Attention is called to cæcal atony as a sequel of enteric fever; the symptoms are morning looseness of the stools, splashing as in a dilated stomach, and attacks suggesting appendicitis. The premonitary diarrhœa of a malarial paroxysm is described, and several chapters are devoted to the effects of dysentery. In various intestinal affections irritation of the cœliac plexus plays an important part in causing pain and diarrhœa alternating with constipation, the outcome of spasm and atony. From the spread of inflammation to the nerve fibres a persistent cœlialgia may result, and in carcinoma of the colon severe colic may be due to the extention of either inflammation or carcinomatous infiltraticn to the sympathetic.

MEDIAEVAL MEDICINE. By JAMES J. WALSH, K.C.St.G., M.D., Ph.D., Sc.D., Litt.D. With 8 illustrations; pp. xii + 221. Price 7s. 6d. net. London: A. and C. Black, Ltd., 1920.

In this book Dr. Walsh tells sympathetically the story of the medical sciences from the fifth to the fifteenth centuries. After a short account of early mediaeval medicine he deals with the great schools of Salernum and Montpellier and shows how much modern medicine owes to their teaching. He then tells of the great surgeons of Italy, France, Flanders and England, and draws attention to the fact that they often attained by the method of trial and error similar results in the treatment of wounds to those which have been recently obtained by more scientific means. The later chapters are devoted to the neglected subject of surgical specialities in mediaeval times, and Dr. Walsh is able to prove that much attention was devoted to the eyes, the teeth, the ear and the diseases of women. There is a chapter on hospitals and another on the treatment of the insane in the middle ages. Dr. Walsh has produced a very readable book on a difficult period in the history of medicine. He does not, however, write for the student and has contented himself in popularizing what was well known to the historian of medicine. There are a few errors to be corrected—e.g., Paynel's translation of the Regimen was published at least as early as 1530; St. Bartholomew's Hospital and the Priory of St. Bartholomew in Smithfield were distinct foundations from the beginning and both came into existence early in the twelfth century. There is a misprint in the second line of the Latin quotation on page 50. These small blemishes do not detract from the interest of the book for the general reader. There are eight well-executed illustrations.

MONOGRAPHS OF THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH. No. XI: TOTAL DIETARY REGULATION IN THE TREATMENT OF DIABETES. By FREDERICK M. ALLEN, M.D., EDGAR STILLMAN, M.D., and REGINALD FITZ, M.D. With 62 charts; pp. vi + 646. New York: Rockefeller Institute, 1919.

The work of Dr. F. M. Allen on diabetes has been divided into three parts. An account of the first portion, in which the results of his experiments on diabetic animals were compared with diabetes in man, was published in 1913. The present volume deals with the second portion and describes a series of clinical investigations in which the principles of treatment elaborated from his experimental investigations have been applied to patients in the hospital of the Rockefeller Institute. A preliminary outline of the third part, devoted to a study of the pathology of diabetes, is also included. The present monograph comprises a record of 76 out of 100 diabetic patients which have been studied in conjunction with Drs. Stillman and Fitz. As the title denotes, the principle underlying the treatment adopted has been a regulation of the total diet, not merely of the carbohydrate ingestion alone. It is claimed that the results obtained demonstrate the practical value of this system of dieting and sustain the theoretical considerations from which it originated, for, although an actual cure, in the sense of a restoration of the normal powers of food assimilation, has not been attained in any instance, an amelioration of the condition and a prolongation of life and usefulness has been secured when the patient has been content to follow the dietetic restrictions laid down for him. The mistakes incident to a new method have reduced the general results below the theoretical ideal, but, considering the grave character of most of the cases selected for treatment and their probable fate under former systems of dieting, the measure of success secured has been encouraging. The first 65 pages of the work are devoted to a history of diabetes. The general plan of treatment adopted by the authors is then described; this chapter being divided into sections dealing with general measures, treatment up to the cessation of glycosuria, emergencies and complications, treatment following glycosuria, ideals of diet and laboratory control, and practical management of diets. A detailed record, with numerous charts, of the cases investigated follows. In the succeeding chapters the effects of pancreas feeding, exercise, and fat in the diet are discussed. The following section is devoted to a critical survey of the results obtained, the reasons for failure in the treatment, the causes of death, complications, and prognosis. A short discussion of the ætiology and pathology of diabetes concludes the work.

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SUPPLEMENT

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NOTES ON BOOKS.

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Diseases of Women. By Thomas George Stevens, M.D., B.S.Lond., F.R.C.S.Eng., M.R.C.P.Lond. Second edition. With 202 illustrations; pp. xv + 443. Price 20s. net. London: University of London Press, Ltd., 1919.

Mr. Stevens's book is relatively small, but the type is good, and one can pay the diagrams no greater compliment than by truthfully saying that one knows what they represent without reading the description, quite a rare merit in diagrams and illustrations. The subject matter of the book is an important one, dealing as it does with the defects and diseases of the female generative organs. It opens with a chapter on diagnosis, and shows that a combination of anatomical, physiological and pathological knowledge, if reinforced by clinical experience, and especially by *tactus eruditus*, will suffice to solve the problems of gynæcology. To help the student (are we not all students?) there are chapters on these subjects, followed by two on development and on malformations. Chapter VI deals with menstruation, its cycle, variations, and other characteristics. Is the author justified in saying that "Very few women can be said to be normal, mentally or physically, during menstruation?" Some observers of much experience would certainly question this dictum. The chapter closes with a much needed caution that all over-prolonged or irregular hæmorrhage at the menopause demands careful investigation. Chapter VII gives an account of the anomalies of menstruation. Attention is drawn to absence, irregularity, excess of menstruation, and to unduly painful conditions. Chapter VIII treats of the "infections of the generative system." It gives to gonorrhœa the prominence it deserves, and sketches the treatment to be adopted. The immediate symptoms and the later consequences of the gonorrhœal infection are carefully distinguished. Next come the infectious following labour and abortion, many of which are septic in origin. Among these infections are cited the streptococcic, staphylococcic, the *Bacillus coli communis*, &c., the most frequent and the most dangerous being probably infection with the streptococcus, but all play an evil part in puerperal sepsis, and so do other micro-organisms on occasion Infection with syphilis is also discussed and many other infections of the vulva and vagina, such as furunculosis. In Chapter IX there is information as to the pathological effects of parturition, some of which, of course, were included in the last chapter. This part of the subject is of equal importance to all medical practitioners and by no means concerns the gynæcologist only. Chapter X is a long one and deals with another common and universally interesting part of the subject, for displacements of the uterus, falling of the bladder and rectocele, are treated (or maltreated?) by us all. Chapter

XI sheds new light on, or rather it emphatically calls attention to, the much neglected subject of "disturbances of the sexual functions." It is wonderful how many otherwise good books on gynæcology fail to devote time and space to this most interesting and important part of the subject. The same remark might well be made with regard to "The urinary system in relation to gynæcology." Information about this was vainly sought by students until quite recently, but it is presented to us in Chapter XIII. Chapter XV gets back to the beaten track and discusses "new growths." Many students have a vain belief that displacements and new growths are practically all the troubles of importance to woman. They should read Mr. Stevens's book to get a fairer perspective. Chapters on preparations for operation and the after-care of operation cases close a most valuable book—one that is a pleasure to read and to review.

THE VENEREAL PROBLEM: A DESCRIPTION OF THE VENEREAL DISEASES: THEIR HISTORY; THEIR PREVALENCE IN GREAT BRITAIN; THEIR EFFECT UPON THE NATIONAL LIFE; THE FACTORS CAUSING THEIR PROPAGATION AND SPREAD; AND A DISCUSSION OF METHODS FOR THEIR CONTROL AND ERADICATION. By E. T. BURKE, D.S.O., M.B., Ch.B.Glas. With 6 illustrations; pp. xv + 208. Price 7s. 6d. net. London: Henry Kimpton, 1919.

Captain E. T. Burke brings a vivid interest and much experience to bear on his work in the important subject of venereal diseases. He writes in a simple, readable manner, and lays the subject before the public clearly and adequately. No one can write on a subject so important and so much discussed in a way to please all readers; but although Captain Burke is fully convinced that his own views are justified, he tacitly admits that other people are entitled to advocate theirs. The book is small, handy and well printed. It has a table of contents and an index which increase its value to busy people. He deals with venereal diseases as they concern the individual and the State. He briefly describes syphilis and gonorrhœa, and points out the very serious consequences of gonorrhœa, especially in woman, involving, as it does, some 75 to 80 per cent. of her pelvic troubles. Comparing syphilis with tuberculosis, scarlet fever, diphtheria, pneumonia, typhoid and influenza, we are shown that in 1910 it came fourth in killing power, tuberculosis being easily first with 51.317 deaths, pneumonia second with 39,760 deaths, but influenza, which stood third with 6,504 fatal cases, was not so wonderfully more disastrous to the individual life than syphilis, with 5,555 deaths. It must be remembered that whereas people die, or recover, from influenza, the secondary and tertiary states of syphilis lead to more crippling, and to more unemployment; and it must not be forgotten that it is a racial poison destroying the partner and the offspring of the sick man as no other disease has power to destroy them. Captain Burke pleads for the prophylaxis of purity, and offers sound advice as to the safeguards afforded by regular employment, athletics, adequate and suitable amusements, and, above all, by early marriage. He points out the dangers of regular prostitution and the still greater powers for evil of the amateur or clandestine prostitute. Captain Burke also recognizes the part played by alcohol in the acquisition and dissemination of venereal diseases, the obfuscated judgment of the alcoholic leading to both these evils, while alcoholism makes the process of cure more difficult and uncertain. Finally notification, education and prophylaxis are carefully discussed, and here we find how long is the road and how uphill is the climb to national purity and safety—still *adsit spes fiat res !!*

PAINLESS CHILDBIRTH AND TWILIGHT SLEEP. By CECIL WEBB-JOHNSON, M.B., Ch.B., Captain, R.A.M.C.(T.F.). Foreword by COMYNS BERKELEY, M.C., M.D., F.R.C.P. Second edition. Pp. xviii + 169. Price 10s. 6d. London: Butterworth and Co., 1919.

There are two "forewords" to this book, one by Captain V. Green Armitage and one by Mr. Comyns Berkeley. The former was selected because he was "the only man of experience in India who had seriously studied the subject and obtained good results by closely following the proper technique." Doubtless by this time Dr. Green Armitage no longer stands alone in this respect. In his opinion, "IF THE CORRECT AND ATTESTED TECHNIQUE is carried out, the doctor within easy call, and the nurse of accredited skill, the advantages of twilight sleep far outweigh the disadvantages, and this is particularly the case in primiparæ." Mr. Comyns Berkeley was selected to write the foreword to the second edition because "he was neither a reckless enthusiast nor a bigoted opponent, but one who saw the dangers and pitfalls to be avoided and readily acknowledged the benefits to be gained." In Mr. Berkeley's opinion "there are certain disadvantages of which the patient is most probably unaware, but of which any doctor professing to use this method of treatment must be conversant. For instance, in a majority of cases labour is prolonged, the necessity for forceps extraction is increased, the percentage of ruptured perineums is higher, and the failure of occipito-posterior positions to rotate naturally, therefore necessitating operative interference, is greater. In other words, the general adoption of 'twilight sleep,' except on lines similar to those laid down in this book and with the conduction of labour with the same antiseptic and aseptic technique as is now obligatory on a surgeon when operating, would inevitably lead to an increased mortality and morbidity rate of the mother and child." The whole book leads irresistibly to the conclusions reached by Captain Green Armitage and Mr. Comyns Berkeley. One rises from its perusal more convinced than before that great as may be the advantages that may accrue to suffering womankind by a perfect application of the Freiburg method of procuring forgetfulness of the pains of childbirth, the price to be paid may be too heavy. The author, Captain Webb-Johnson, is an enthusiast, and would, if possible, annul the sorrow in which women bring forth children, but he sees clearly that long and special training is necessary to make the scopolamine-morphine amnesia (forgetfulness) safe. He admits that the drugs must be pure and the technique perfect if the process of labour is to be rendered pleasant while not increasing its danger. Captain Webb-Johnson very properly points out the distinction between "painless childbirth" and "twilight sleep," and tells us candidly that while, in his opinion, *amnesia* is compatible with safety, *analgesia* is not, with the means at present at our command. The book contains chapters dealing with the drugs, with the correct technique, and with the criteria on which variations of the treatment are founded. At the end of the book are specimen charts of the character and results of labour under twilight sleep, and evidently the author hopes that his readers may try the Freiburg method and tabulate their results. This is the way in which some more generally practicable a method may eventually be evolved.

THE AFTER-TREATMENT OF WOUNDS AND INJURIES. By R. C. ELMSLIE, M.S.Lond., F.R.C.S.Eng., Brevet Major R.A.M.C.(T.). With 144 illustrations; pp. vi + 319. Price 15s. net. London: J. and A. Churchill, 1919.

The author has arranged his subject matter in two parts. In the earlier chapters he describes the principles which govern the after-treatment of wounds and injuries, and includes mal-union, delayed union, and non-union of fractures; stiff and flail joints; injuries of nerves, muscles, tendons, and skin. In the later chapters he has followed a regional classification, and deals with the injuries of specific regions, the upper and lower extremities and the spine. Many helpful examples of actual cases are given. Considerable space is devoted to the normal anatomy of the peripheral nervous system: it is a question whether this is necessary in a book of this character. Whilst the author acknowledges his indebtedness to the teaching of the principles of orthopædics by Sir Robert Jones, many of whose methods he has employed, he strongly supports the radical operation described in Professor Broca's book for the treatment of chronic osteomyelitis. The chapter devoted to amputations impresses the principle that the site of amputation must be determined by the functional utility of the final stump. The last three chapters deal with plaster of Paris technique, splints and surgical appliances and physiotherapy. Three principles are strongly emphasized upon the reader throughout the book: the necessity of a knowledge of the pathological condition, a clear appreciation of the mechanics of the part, and a constant aim at the restoration of function. The work is the result of an extensive personal experience of cases, mainly at the Special Military Surgical Hospital at Shepherd's Bush, and will prove a valuable guide to surgeons employed in reconstructive surgery.

PENSIONS AND THE PRINCIPLES OF THEIR EVALUATION. By LLEWELLYN J. LLEWELLYN, M.B.Lond., and A. BASSETT JONES, M.B.Lond., Temporary Captain R.A.M.C. With a Section on Pensions in Relation to the Eye, by W. M. BEAUMONT. Pp. xxvii + 702. Price 30s. net. London: William Heinemann (Medical Books) Ltd., 1919.

The main object of the authors of this book is to express in grades of incapacity, for purposes of pension, the effects of injury and disease incurred in military or naval service. They have accordingly surveyed a very large number of such conditions which disable or partially disable a man, and have set out the various factors in each case which should influence the examiner in determining the actual grade of incapacity resulting from the injury or disease. In addition, the book contains a number of chapters dealing with the history of State-provided pensions, recent legislation, procedure in administration, and other subjects. We learn that provision of some sort or other for the disabled soldier dates from Anglo Saxon times, when every "ceorl" who was liable to serve had "five hides of land" and certain dignities. Under the feudal system the barons made grants to their retainers. Edward the Fourth made grants for battle service to certain individuals, but the first statute for providing for maimed soldiers and mariners dates from the reign of Elizabeth. The authors describe the later evolution of the pension system up to the issue of the Royal Warrant of 1918. With an organization changing so frequently in its details, and growing so rapidly, as the Ministry of Pensions, it is unavoidable that some sections of this book should quickly get out of date.

THE PRINCIPLES OF GYNÆCOLOGY: A MANUAL FOR STUDENTS AND PRACTITIONERS. By W. BLAIR BELL, B.S., M.D.Lond., &c. Third edition. With 392 illustrations; pp. xv + 660. Price 38s. net. London: Baillière, Tindall and Cox, 1919.

Dr. Blair Bell's book has reached its third edition, a fact which shows that it ministers to a real need of medical practitioners and students. The arrangement of the contents of the book are unusual and have been criticized, but there is much to be said for the convenience of considering gynæcological ailments from the pathological rather than from the regional standpoint. Chapter I deals with the evolution of the female generative organs and traces their evolution from the simple structure of the lamprey and running through the amphibiæ, reptilia, to the mammalia. The subject is interesting not only in itself but because it illustrates the morphology of the human organs. Chapter II is concerned with the development of the female genital organs. The importance of the subject is very great, as Dr. Blair Bell says: "The normal progress or development is of great importance, for only by a knowledge of it can we explain the malformations, and certain other pathological conditions, due to developmental defects, which we may meet in our clinical work." Accordingly we find a careful description of the gradually advancing normal development, together with the possible arrests and perversions; the whole is well illustrated by diagrams that are clear and so lettered as to be readily understood. In Chapter III we have the anatomy of the female genital organs, beginning with their naked-eye appearances and relations. The illustrations are drawn from various sources, as may best illustrate the text—Kelly, Cunningham, Cullen, Moritz, and other authors are thus quoted. The value to the student of good, clear, and well-lettered diagrams cannot be exaggerated. In Part II we find the physiology of the female genital organs, including the consideration of puberty, menstruation, reproduction, and the menopause. This portion of the book is short, and what we have given us in it makes us wish there were more. Part III deals with case-taking and the examination of the patient. Dr. Blair Bell cautions his readers not to "rush" their patients; haste is the enemy of accuracy and the destruction of that confidential relation that must exist between doctor and patient if real information is to be gained by the one and real benefit by the other—alas! how difficult it is to secure the time in which this desideratum can be attained. Part IV is devoted to derangements of the normal anatomical conditions, and we are led on through malformations, injuries, and malpositions to Part V, in which are described the disorders of puberty, menstruation, and conception. We are reminded of the various types of menstruation, of the various errors of menstruation, and of the causes of these peculiarities, excesses, and defects. One of the duties of the physician is to teach women that each one has her own physical personal peculiarities, and that it is vain to expect accurate conformity to any supposed physiological standard. Under the disorders of conception we find a section on sterility, a much neglected and an important subject on which the profession and the public are in need of careful instruction based on experience. Abortion and extra-uterine conception receive brief notice, no doubt because they belong more rightfully to a treatise on obstetrics. Part VI is devoted to infective and parasitic diseases of the genital tract. As would be expected, the first infection described is that due to the gonococcus. There is a large field awaiting careful exploration in the infective and parasitic lesions of the female genitalia, and a rich reward will accrue to those practitioners who study and treat them with the care they demand. Part VII is devoted to the important subject of tumours, and naturally is relatively long. It deals with tumours of all kinds affecting each and all of the female genital organs—one cannot but feel the difficulty of dealing with this large and deeply important subject within the narrow limits necessarily assigned to it in a relatively small book in which much space is rightly devoted to other branches of gynæcology. Part IX deals with operative procedures, and is founded almost exclusively on the author's experience and methods. There is always an element of fascination in seeing how another surgeon works and how he meets the difficulties to which we all are liable. The beauty of the illustrations and the clearness of the type enhance the pleasure of reading the book.

MIND AND ITS DISORDERS: A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS OF MEDICINE. By W. H. B. STODDART, M.D., F.R.C.P. Third edition. With illustrations; pp. xx + 580. Price 18s. London: H. K. Lewis and Co., 1919.

Dr. Stoddart's book is well known and mainly requires notice here because, in his recently published third edition, there is ample evidence of what the author states in his preface—i.e., that he has fundamentally changed his attitude towards mental disease, and from practical experience has become convinced of the truth of Freud's theories. Psycho-analytic doctrines and conceptions therefore are found, though the theoretical considerations of the materialistic school are by no means neglected. While the description of the various maladies has been altered but little, there has been an endeavour to indicate the psychical mechanisms underlying them as far as possible without entering into great detail. Some chapters have been entirely rewritten and others enlarged. The chapters on normal psychology and the psychology of the insane are naturally added to, especially in relation to memory, unconscious association, emotion and action; the sexual and herd instincts; dreams; delusions and hallucinations. As in the second edition, the task of expounding the principles of psycho-analysis within the scope of ten pages is attempted. It is curious to note that in discussing the causation of insanity, notwithstanding his pronounced Freudian views, Dr. Stoddart makes so little mention of mental conflict as the basic factor in the ætiology of the functional psychoses. We are surprised to hear that neurasthenia is always due to partially or completely repressed auto-eroticism, that the renunciation of masturbation is largely causative, and that psycho-analysis is its only cure. It was thought that the cessation of this habit tended towards the production of anxiety neurosis (upon which a new chapter is added). At any rate, if the mechanism is purely psychical, why is neurasthenia not brought under the heading of the psychoneuroses? Exophthalmic goitre is now classified under the neuroses, certainly not without good grounds, though perhaps it is anticipating too much to state that the disease is curable by psycho-analysis. In dealing with the psychoses it is noted that the classification has been completely re-modelled, but the old conception of anergic stupor is still retained as though it were a disease entity instead of only a symptom. The paraphrenia of Kraepelin is accorded a special chapter, and the results of recent investigations in the treatment of general paralysis have been added. The work is certainly an excellent up-to-date text-book. We might take this opportunity of suggesting that normal psychology should not form part of a psychiatric text book and that the preliminary pages devoted to it can never be sufficient groundwork for the student of the abnormal. Psychology must previously be studied more extensively elsewhere.

Science and Life. By Frederick Soddy, M.A., F.R.S. Pp. xii + 229. Price 10s. 6d. net. London : John Murray, 1920.

This volume is a collection of essays and addresses, some of them previously published, written or delivered between 1915 and 1919 while the author was a professor at Aberdeen; in addition an appendix contains some articles and reports bearing on definite charges made by the author as to the financial treatment of science by the Carnegie Trustees for the Universities of Scotland and the University of Aberdeen. His contention is that the funds of the trust are not in general applied to the purposes for which they were intended, but are used for general University needs and to provide buildings and endowments for Art subjects, instead of the promotion of scientific study and research. In his presidential address to the Aberdeen Scientific Society on November 3, 1916, on "The Future of Science, and what Bars the Way," he brings a forcible indictment against the older Universities, where a culture that reached its zenith before the birth of Christ still struggles to retain its complete ascendency over the human mind and by opposing science acts as a clog to national reconstruction. In an address on some of the relations between "Science and the State," he points out that scientific research must not be regarded as a hobby or part-time occupation of the leisure hours of the busy teacher but as a serious business distinct from teaching: there should be research professorships, a need to which further reference is made in an article "To the new Launch," contributed to the first number of *The Crucible*, a magazine started by the science students of Aberdeen. There are several articles on chemistry, on radium and allied subjects, and special attention may be directed to the lecture before the London Chemical Society on "The conception of the Chemical Element as enlarged by the study of Radio-active change."

The Microscopic Anatomy of the Teeth. By J. Howard Mummery, D.Sc.Penn., M.R.C.S., L.D.S.Eng. With 243 figures in text; pp. viii + 382. Price 25s. London : Henry Frowde, Hodder and Stoughton, 1919.

Since the days of John Hunter and Sir John Tomes many histologists have contributed to our knowledge of this fascinating subject. The author of the present volume has finally summed up this knowledge and with the addition of his profound original work has reached a high degree which he modestly attributes to "modern perfected methods" but the work displays throughout the true, scientific spirit and marvellously accurate methods. Reviewing the evolution of teeth, he passes to a complete chapter on the development of mammalian teeth. The chapter on enamel, "one of the most difficult substances for microscopical examination," demonstrates the painstaking methods employed. The arching of the enamel prisms and the grooves into which they fit, is illustrated by many slides and diagrams, most of them original. This is specially shown in the teeth of the elephant. Plate III has several diagrams of teased enamel showing that "these prisms have a concavo-convex connexion and are deeply grooved posteriorly." It seems more practical to consider the dental pulp, the formative organ of the dentine, before the dentine itself, which is the order chosen by the author. A diagram on p. 221 illustrates the author's view of "the probability that both sensory and trophic nerves are distributed to the pulp and dentine." It is said also that recent researches appear to demonstrate that a lymphatic system is present in the dental pulp. Referring to Black's analyses of dentine, the author says that "although the amount of salts in the dentine may be approximately the same, the manner in which they are bound together in the dental tissue may vary considerably." The classification of Sir Chas. Tomes is that preferred in this work, viz., orthodentine, plicidentine, vasodentine, osteodentine. The minute anatomy of these is amply illustrated from the author's own preparations. On the calcification of dentine, the view is held by the author and others "that the cells of the pulp secrete a material which calcifies, they themselves not entering into the calcification of the substance." In a chapter on cement it is stated "that absorption of the temporary teeth begins in the cement and is not due to pressure from beneath." The chapter on the periodontal membrane shows that "the rich supply of blood-vessels and nerves to the membrane would fully account for its great sensibility in inflammatory conditions." The original work on the tooth follicle is perhaps the most fascinating in the book. The much discussed theories of Nasmyth's membrane are reviewed, but it is shown that "the forces which govern the process of eruption are still but imperfectly understood." Such are but a few of the salient points in a work which demands careful study. It will be valued by biologists and comparative anatomists as well as by dental students old and young who desire to prove their position or build on a sure foundation.

Practical Histology. By J. N. Langley, Sc.D., LL.D., F.R.S. Third edition. With illustrations; pp. viii + 320. Price 10s. 6d. net. Cambridge: W. Heffer and Sons, 1920.

The general plan of this handy book is the same as that adopted in former editions, the essential part of it being printed in large type and this representing in general the course of practical histology for elementary students in Cambridge; whereas many useful notes and the appendix are printed in smaller type, these being intended for advanced students and to aid assistants in preparing demonstration specimens. In the general course some changes have been made to meet small difficulties which the experience of the classroom has shown to be felt by students, and in order to bring the description of what is to be seen in the specimens in accord with present nomenclature. Important additions have been made in the appendix, particularly with regard to the central nervous system. Thus a brief but quite sufficient description is given of the Cajal and Bielschowsky methods as well as of some of their recent modifications for neurofibrils, of Cajal's gold chloride and Rio-Hortega's carbonate of silver methods for neuroglia, of Rio-Hortega's modifications of Achucarro's tannin methods, &c. This renders Langley's work a practical book of consultation, which every physiological and histological laboratory ought to possess.

The Philosophy of Conflict, and other Essays in Wartime. Second Series. By Havelock Ellis. Pp. 299. Price 6s. 6d. net. London: Constable and Co., Ltd., 1919.

This is a series of essays on various subjects, most of them unconnected with war. The title essay, "The Philosophy of Conflict," emphasizes the part played by struggle in the upward course of civilization altogether apart from war. "The world is cemented with blood and sweat; without pain and fortitude—that is to say without struggle and conflict—there would have been no world at all." It is the toils and struggles of civilization which create the spirit of heroism; war merely gives it scope, a fruitless scope which does not advance human progress. There is no place to-day either for the pacifist or the militarist, since both are under the delusion that, with the ending of war, struggle would vanish from the earth. Other essays are on "Eugenics in Relation to the War," "Birth Control and Eugenics," and "War and the Sex Problem." Biography is represented by essays on Luther, Herbert Spencer, Rodo, and Cowley. Perhaps of most interest to medical men will be the essay "Psycho-analysis in Relation to Sex," in which the author expresses his high opinion of the work of Freud.

An Introduction to Child Psychology. By Charles W. Waddle, Ph.D., Department of Psychology and Education in the Los Angeles State Normal School. Pp. xvi + 317. Price 6s. net. London: George C. Harrap and Co., 1919.

A small book on a very interesting and important subject. All that pertains to the child is a portion of the subject which engrosses the attention of all thinkers of the present day. The first chapter supplies us with an "historical background of the scientific study of children." It tells us, all too truly, how deep has been the guilt of the human race in all ages and in all races towards little children. The indictment is painful but unmistakably just. This is followed by a description of several methods of child study, including the well-known "intelligence" tests and Binet-Simon method. The third chapter goes somewhat carefully into the application of the principle of evolution to the study of child life—including a consideration of Darwin's theory—and the view that the ovum, the embryo, and the fœtus recapitulate in brief—but with inversions and omissions—the gradual development from the amœba to the primate. The author does not think this bears pressing but feels that there is more in the post-natal development of the individual which recapitulates the moral and mental history of the race. Mr. Waddle lays much stress on the influence of heredity, It is of course true that heredity is a great factor but surely we may hope that mentally and morally environment is the more powerful. The latter part of the book deals with the more novel and interesting subjects of the peculiar linguistic development of children. First is considered the manner in which speech commences in children and later the indications of degrees of intelligence and normal development afforded by the age at which the function is fully developed. In Chapter VIII is considered the subject of children's drawings—nearly all children enjoy drawing and revel in colouring—but few of these early blossoms mature and "set fruit." Mr. Waddle says: "Surely an activity which has played such a rôle in the life of our primitive forebears ought to fulfil its promise of tremendous value as a culture medium for children." With regard to the moral nature of children and their delinquencies—these delinquencies are, it is remarked, largely dependent on feeble-mindedness—surely a strong argument in favour of continuing guardianship and supervision and not of sending the feeble-minded out into the world to work ill to themselves and their neighbours and to procreate an increasing number of similarly afflicted individuals. A very comprehensive bibliography, a glossary, and test questions are provided for the use of students.

ESSENTIALS OF SURGERY: A TEXT-BOOK FOR STUDENT AND GRADUATE NURSES, AND FOR THOSE INTERESTED IN THE CARE OF THE SICK. By ARCHIBALD LEETE MCDONALD. M.D. With 46 illustrations; pp. ix + 265. Price 8s. 6d. net. Philadelphia and London: J. B. Lippincott Co., 1919.

The book opens with an account of the principal varieties of pathogenic bacteria, their distribution in the body, and the means of destroying them under certain circumstances. Their portals of entry are described and the means whereby the body is able to defend itself against their invasion. Chapter III elaborates the description of various forms of bacilli and cocci and sketches the pathological changes they may produce and the line of treatment to be adopted in some surgical infections. Chapter IV is devoted to the subject of tumours, or new growths—but is limited to eight pages, including illustrations. Indeed the whole book is rather of the nature of an "appetizer," and will probably stimulate the nurse's desire to know more of the subjects touched on by the author. In the fifth chapter are brief statements as to wounds, hæmorrhage, surgical operations and anæsthesia—a formidable collection of subjects for one chapter. Under the heading of surgical operations are considered contra-indications, the preparation of the patient, after-care and post-operative complications. Here, as is very fitting, we have instructions as to "positions" and various devices to promote the comfort and safety of the patient. Chapter VI, on bones and articulations, gives us a sketch of the structure of bones and of the infections, diseases and accidents to which they are liable. A brief account of vascular and nervous symptoms follows in Chapter VII and the remainder of the book is a succinct, but all too brief, résumé of the anatomy, accidents, and surgical diseases, of most of the regions of the body. To the book proper there is added a useful glossary of some of the most frequently used surgical expressions.

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SUPPLEMENT

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NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

Employment Psychology: the Application of Scientific Methods to the Selection, Training and Grading of Employees. By Henry C. Link, Ph.D. Pp. vii + 440. Price $2.50. New York: The Macmillan Company, 1919.

The application of scientific methods to the selection, training and grading of employees in industrial processes has only recently received attention, but the results already obtained are sufficient to prove the value of such procedure. In this book Dr. Link describes in detail the psychological tests which can be used in the process of selecting employees. These tests enable the workers to be classified, and each one set to that form of occupation for which he is best suited. A scientific selection replaces the previous more or less haphazard procedure, and much of the time wasted before it is realized that a person is unsuitable for his employment is saved. The book does not contain much of purely medical interest, but should be of value to employers and students of psychology.

Radio-diagnosis of Pleuro-pulmonary Affections. By F. Barjon. Translated by James A. Honeij, M.D. With plates; pp. xix + 183. Price 10s. 6d. net. Newhaven: Yale University Press; London: Humphrey Milford, 1918.

Published under the auspices of the Yale School of Medicine, Professor J. A. Honeij has rendered good service to the cause of radiology by making the work of M. F. Barjon more readily accessible to English-speaking physicians. The five principal sections of this book deal with methods of examination and technique, the radiological study of the pleuræ, bronchi and lungs, and penetrating wounds of the thorax by war projectiles. The fifty-two radio-photographs depict the conditions described in the text with accuracy and clearness. In the section on pulmonary tuberculosis, the author states that the disease, which may often seem "slight and incipient in a patient, will be proved by radioscopic examination to be more extensive and secondary to a previous attack." If the stethoscopic signs point to one apex being possibly affected, but the radiograph shows a loss of transparency upon the opposite side, the result may be considered doubtful. If, however, the stethoscopic and radiographic signs agree, the probabilities become certainties. The author does not claim that radiological examination will eliminate the difficulty that attaches to the early diagnosis of pulmonary tuberculosis, but its employment should never be neglected in doubtful cases. In establishing the indications and contra-indications for surgical treatment of the lungs the

X-rays have proved most valuable. If the radiologist ought to be a physician—and he should be according to M. Barjon—it is desirable also that the physician should interest himself to a larger extent in radiology. The bond between physician and specialist might well be drawn closer in the interests of all concerned. The book is well got up and will be found useful to radiologists and practitioners alike.

NOTES ON GALVANISM AND FARADISM. By E. M. MAGILL, M.B., B.S.Lond., D.P.H., R.C.S.I.Hon. Second edition, with 67 illustrations; pp. xvi + 224. Price 6s. net. London: H. K. Lewis, 1919.

This, the second edition of Dr. Magill's well-known little book, has been revised and additions have been made to the text. The book is intended primarily for the use of masseuses preparing for the examinations in medical electricity held by the Incorporated Society of Trained Masseuses, but it will be found very serviceable to medical men who do not propose to devote attention to the more complicated forms of apparatus. Dr. Magill begins with an introductory chapter in which she considers in outline the need of electricity. Succeeding chapters deal with static electricity in medical work, the galvanic battery, ionic medication, faradism, the dynamo, electric baths, and radiant heat and light. The book is succinctly written, and is quite sufficient for its purpose. The illustrations are clear and help the descriptions of apparatus, &c., in the text. A series of examination papers is included in the appendix.

MOSES: THE FOUNDER OF PREVENTIVE MEDICINE. By PERCIVAL WOOD, M.R.C.S., L.R.C.P., Captain R.A.M.C. Pp. xi + 116. Price 4s. net. London: S.P.C.K., 1920.

In this book the author seeks to show that Moses, the "superman" of his age, was the originator of the principal laws of sanitation that hold good at the present day. His was the master-mind that impressed upon the ancient Israelites the necessity of attention to personal hygiene, and for the proper disposal of camp refuse. The lesson to be learned from the ten plagues of Egypt is that "bad sanitation is invariably followed by disease and death." The question of food control occupied a considerable part of the Mosaic code, but his legislation on diet deals with suitability only. In the matter of the control of infectious disease our present system, though, of course, "expanded, improved in detail, in technique, in organization," remains in essentials, "precisely the same as that originally established by Moses thirty centuries ago." In his concluding chapter the author thinks that it is most probable that Moses did not "fully understand the hygienic importance of his own work," nevertheless, even his occasional failures cannot "hinder us from regarding him as the founder of preventive medicine."

THE MEASUREMENT OF INTELLIGENCE: AN EXPLANATION OF AND A COMPLETE GUIDE FOR THE USE OF THE STANFORD REVISION AND EXTENSION OF THE BINET-SIMON INTELLIGENCE SCALE. By LEWIS M. TERMAN. With an Introduction by Professor J. J. FINDLAY, M.A., M.Ed. With Test Material for the Measurement of Intelligence, by LEWIS M. TERMAN. Pp. xviii + 362. Price 6s., 3s. 6d. London: George G. Harrap, 1919.

Not being so preoccupied during its earlier years with matters arising out of the war as were the people of this country Americans were able to devote themselves to other questions, including that of tests for intelligence. As a result several works on the Binet-Simon system of testing are now coming to hand, and this book, at any rate, is worth reading. What part in the conduct of life is played by the "intelligence" which can be defined in terms of the Binet-Simon scale is doubtful and the weakness of mankind for rushing after some new thing has probably caused a quite artificial value to be placed on the tests, but they may, nevertheless, serve a useful purpose. They supply the inexperienced examiner, who does not know where to begin, with a means of learning something about his patient and they enable him to set out the fruits of his inquiry in a way which makes them comparable with other people's results and intelligible to the ordinary citizen. With the co-operation of former students at the Leland Stanford Junior University, Professor Terman has carried out a thorough revision of the tests. By the modification and rearrangement of some and the introduction of others he has added considerably to the value of the scheme, more particularly in regard to its application to the slighter degrees of mental deficiency and to older children and adults, in which respects the older scheme left a good deal to be desired. The first section of the book deals with various aspects of the problem of "intelligence." Professor Terman considers, with some show of reason, that the use of his scheme helps the teacher to avoid the pitfalls in which exponents of the older methods were liable to be entrapped—those of over-estimating the intelligence of retarded children and under-estimating that of superior children. Recognizing the importance of standardization he devotes the second part of his work to a detailed description of the application of the tests to individuals of the ages of 3 to 10, 12 and 14 years, and to the "average" and the "superior" adult. The test materials, including the "Record Booklet," which are designed to accompany the book, greatly facilitate its employment. It would be easy to quibble over various tests but no useful purpose would be served by doing so. The utility of the scheme will be determined by the extent of its adoption, not by the merits of particular procedures. One slight emendation may, however, be suggested. In mentally reversing the hands of a clock there seems no reason why a reading capable of giving an intelligible result should not be taken. At ten minutes past eight, one of the selected times, the hands are in a position which on reversal gives rise to a chronometric absurdity which would not be nearly so glaring if the time chosen had been eight minutes past.

La Réaction de Bordet-Wassermann pour le Séro-diagnostic de la Syphilis: Étude Théorique et Pratique—Méthodes Recommandées, Interprétation des Résultats. Par A.-D. Ronchèse. Préface par M. le Professor Fernand Widal. Pp. xvi + 211. Price 10f. Paris: Masson et Cie, 1919.

In taking up a book on the Wassermann reaction to-day we immediately turn to see if the writer has thrown any light upon its *rationale*. All pathologists are now familiar with the technique and the various modifications which have from time to time been advocated. As it becomes practically impossible to interpret the results of a reaction which is not understood and as clinicians are coming round more and more to the view that the interpretations which have been made in the past are fallacious, it is disappointing to find that the *modus operandi* of the reaction has not been explained. The technique and modifications are very fully described and the usual advice as to how the test should be read and applied to the clinical aspect has been given. The author is fully impressed by the value of the reaction and considers the time to be not far distant when an antigen will be prepared which will make the reaction actually specific.

Syphilis in Childhood. By Leonard Findlay, M.D., D.Sc., F.R.F.P.S.G. With 37 illustrations; pp. xii + 154. Price 8s. 6d. net. London: Henry Frowde, Hodder and Stoughton, 1919.

Considering how few are the works on congenital syphilis and that the disease is being modified by the present-day treatment, we welcome a book, which is based upon clinical experience. The author dwells at some length on the mode of infection of the child but has omitted the effect the chorionic ferments have in preventing the disease manifesting itself in the mother. Maternal infection must obviously be more common than pure paternal infection since every woman who has given birth to a syphilitic child is *ipso facto* herself infected. Being syphilitic she is liable to convey the disease to each succeeding offspring, an event, which can be avoided by treatment. A little more stress might have been laid on the treatment of the mothers and on the suckling of the infants by the mothers who had been so treated. It would also be interesting to learn the author's views as to whether the children born of syphilitic mothers, who have been properly treated, are syphilitic or not, and what happens to both these and to children who were undoubtedly syphilitic but who had been restored to health by the treatment. These are the main points, as most are familiar with the ordinary signs and symptoms of congenital syphilis. The author has included an excellent series of illustrations and interest has been added by the forty-seven case histories.

MILITARY PSYCHIATRY IN PEACE AND WAR. By C. STANFORD READ, M.D.Lond. With 2 Charts; pp. vii + 168. Price 10s. 6d. net. London: H. K. Lewis and Co., 1920.

Dr. Read had an extensive experience of mental disease before the war and was thoroughly up to date in his knowledge, being an avowed Freudian. Moreover, he had the good fortune to be given war work to which he was suited and was not utilized as a round peg in a square hole. He visited all the mental war hospitals in France and England and ultimately had charge of the mental block at Netley Hospital. From this valuable experience he has given us this present study of "Military Psychiatry in Peace and War"—mostly, of course, in war. After an interesting study of the psychology of the soldier from the recruiting office to the firing line, he compares military psychiatry before the war with that experienced during the war. This amounts to a comparison of insanity in the old standing Army with that in what was practically the whole young adult male population of the country, for these all became soldiers. In a chapter on "statistical facts and figures" a chart is given of the rise and fall of mental cases during the course of the war with an explanation of the rises and falls. Then follow chapters on dementia præcox, paranoid states, confusional states, mental deficiency, epilepsy and so forth, in their relation to military service, a few typical cases of each mental disorder being given. The recruiting system comes in for some well-deserved criticism.

DIABETIC DIETING AND COOKERY. By P. J. CAMMIDGE, M.D.Lond. Pp. viii + 222. Price 10s. 6d. net. London: University of London Press, Ltd., 1920.

The author has put together a very useful series of instructions to enable diabetic patients to have a variety of suitable and palatable foods and to indicate to the medical practitioner the lines on which the diet should be regulated. The book commences with a discussion of the necessary dietetic treatment which follows on the lines recommended by Allen with periodical starvation days. He discusses in detail the necessary restrictions in diet in order to get the urine sugar-free, and how liberty may be allowed if the analysis of the urine and blood show that it is tolerated. He insists on the necessity of restricting, in most cases, not only the carbohydrates but also the fats and proteids. Great stress is laid on the importance of watching the amount of sugar in the blood, which should fall to 0·06 to 0·08 per cent. in the morning, and even after a carbohydrate meal should not exceed 0·14 to 0·15 per cent. For accurate sugar testing he recommends a copper sulphate and iodine solution with carbonate of soda, and he titrates with sodium thiosulphate, but for general use he recommends Benedict's solution, which is more delicate than Fehling's. The estimation of sugar in the blood can be made with only 0·2 c.c. of blood, and the colorimetric test with sodium picrate-picric acid is recommended; this does not occupy much time especially when the solutions are ready for use. One of the most practical suggestions is to take standard rations of 20 grm. for protein, 4 grm. for carbohydrates and 5 grm. for fats, and to classify the various articles of diet under these headings. Each heading is subdivided into three or four groups so that *an average serving* of all the articles in any one group contain the same proportion of a ration and are interchangeable in use. By this means when the doctor has determined the suitable number of each of the rations the patient is at liberty to select his foods from the lists accordingly.

PULMONARY TUBERCULOSIS. By MAURICE FISHBERG, M.D. Second edition, revised and enlarged. With 25 plates and 100 other illustrations; pp. xii + 744. Price 27s. 6d. Philadelphia and New York: Lea and Febiger, 1919.

The new edition of Fishberg's well-known "Pulmonary Tuberculosis" gives a sufficiently comprehensive summary of modern theory and practice, presented in a singularly clear and sane manner. Neither too detailed nor too diffuse it yet affords a continued interest such as no mere compilation could ever do and is all the more valuable in that it comes from a physician of great practical experience. The theoretical basis of the book may be thus summarized: None of the known channels of infection can of itself adequately account for tuberculosis and there is something to be said for Römer's suggestion that there is probably some other mode of transmission with which we are as yet unacquainted. Infection alone will not cause phthisis and an emphatic distinction must be made between tuberculous infection and tuberculous disease. The fact that 80 per cent. of children are infected by the age of 14 and that in them the disease is largely a bacteræmia, whereas 95 per cent. of tuberculosis in adults is a local chronic disease of the lungs, is proof of the operation of other factors in phthisio-genesis. These factors—predisposition in a word—are complex, but in individual cases may consist in some natural power of resistance, massiveness of the primary dose, hereditary influences, constitutional biochemical or serological derangements of the body or of the blood, or local anatomical peculiarities. In the result phthisis is a manifestation of immunity against tuberculosis which has been acquired in the very great majority of instances in early childhood. Tuberculous infection can only occur once and phthisis evolves only in those who are predisposed to the disease. If this immunity fail then a metastatic (autogenous) reinfection takes place, not an acute miliary tuberculosis, but a local lesion, i.e., phthisis in the lung, the most vulnerable organ of the body. Fishberg discards the usual classifications of the disease and it is something of a novelty to find no mention even of such an old friend as "Turban-Gerhardt," nor does it appear that the "Astor" has yet penetrated to the ancestral home. The chapter on "Abortive Tuberculosis" should act as a wholesome corrective to pessimism, while the whole discussion on the types of the disease described by the author will well repay study. Sanatorium treatment comes in for some very plain speaking, the 5 or 10 per cent. of alleged cures being no more than is obtained by home treatment, while the cost is unjustifiable when one considers that at a moderate estimate 33 per cent. of the abacillary cases in a sanatorium are probably non-tuberculous. So radically does Fishberg differ from the accepted English creed in this respect that he would scrap existing sanatoria as such and convert them into hospitals "which admit patients on short notice, keep them for a few weeks, a month or two, until they regain their strength, and are fit for treatment in the clinics." Nor is tuberculin of more use in treatment than as a form of psycho-therapy, its efficacy depending on the intelligence of the patient and his appreciation of the theoretical aspects of the disease derived from a perusal of popular literature on the subject. A carefully written and detailed chapter on the theory and technique of artificial pneumothorax adds much to the usefulness of the book. The illustrations are excellent and well chosen and the X-ray photographs clearly reproduced in miniature. Mistakes in the text are more numerous than they ought to be and the index of authors requires drastic revision.

FUNCTIONAL NERVE DISEASE: AN EPITOME OF WAR EXPERIENCE FOR THE PRACTITIONER. Edited by H. CRICHTON MILLER, M.A., M.D. Pp. xi + 208. Price 8s. 6d. net. London: Henry Frowde, Hodder and Stoughton, 1920.

This book is intended to present to the practitioner a concise picture of the war neuroses, and the various parts of the subject, both from the theoretical and therapeutic standpoints, are dealt with by authors who have had special neurological experience in the army during the late war. Physical ætiology is taken in hand by the editor somewhat inadequately in a few pages. Dr. Riddoch, in his chapter on "Differential Diagnosis" mainly speaks of the differentiation of the neuroses and organic disease, but we fear that inexperienced physicians would find this a poor guide. Physical treatment is naturally of small importance but is sufficiently indicated by Dr. Bramwell. The mechanism of hysteria is described by Mr. Prideaux from a psycho-analytic point of view, and Captain Hadfield writes on treatment by suggestion and persuasion. Hypno-analysis does not prove quite so simple as the text asks us to believe and we greatly doubt that "telling a patient that he will not leave the room till he has divulged his repression nearly always succeeds." The explanations of repression and suppression as given by Dr. Rivers are extremely debatable. By far the best chapters in the book are those by Dr. Nicoll who deals interestingly, though briefly, with regression and psycho-analysis. The origin of many war neuroses in a "mother complex" is insisted on by Dr. Miller but there was hardly need to quote twenty-eight cases to illustrate the same point. The question of individual and institutional management follows. In conclusion Dr. McDougall summarizes the previous pages, and herein points out that the teaching of the book is not Freudian (?) though many of the authors have made use of conceptions which we owe to Freud.

ANÆSTHESIA IN DENTAL SURGERY. By THOMAS D. LUKE, M.D., F.R.C.S.Ed., and J. STUART ROSS, M.B., F.R.C.S.Ed. Fourth edition. With illustrations; pp. xii + 255. Price 8s. 6d. net. London: William Heinemann, 1919.

The authors give a general review of the use of nitrous oxide alone and when diluted with oxygen; ethyl chloride, ether and chloroform, and describe the apparatus employed and requisite technique. The design of the book is to convey the information in such a way as may render it clear to dentists. In the chapter on chloroform the open method is advocated and the arguments for and against the employment of this anæsthetic in dental surgery are weighed. Ether is stated to be less safe than ethyl chloride, the latter being extolléd for use when nitrous oxide is unavailable. Local analgesia is restricted to submucous injection, conduction analgesia not being considered. Cocaine injection is considered justifiable in dentistry and although itself a vaso-constrictor is recommended in association with adrenalin. Stovaine is also regarded favourably but its tendency to cause local necrobiosis is not adverted to. The relative safety of the general anæsthetics is carefully gone into, and lists of fatalities are given. The legal position of dentists in the matter of the use of anæsthetics is discussed. The history of anæsthesia, the choice of the anæsthetic and the common accidents under anæsthetics with the appropriate treatment are also considered.

CONVEYANCE AND DISTRIBUTION OF WATER FOR WATER SUPPLY, AQUEDUCTS, PIPE-LINES AND DISTRIBUTING SYSTEMS: A PRACTICAL TREATISE FOR WATER-WORKS ENGINEERS AND SUPERINTENDENTS. By EDWARD WEGMANN, C.E. With 367 illustrations, and 8 plates; pp. vi + 663. Price 26s. net. London: Constable and Co., 1918.

Entirely an engineering book and contains little of interest to medical men even if engaged in the preventive side of medicine. It deals exhaustively with such subjects as water consumption, loss of hydraulic gradient in aqueducts, pipes, &c., types of water-pipe with specifications and details for calculating them, types of joints and their relative efficiency, submerged and flexible pipes, valves, hydrants, aqueducts, water-meters. A useful appendix of specifications for piping and castings is appended. The examples are mostly drawn from American practice but some British examples are included.

MANUAL OF SURGERY (ROSE AND CARLESS) FOR STUDENTS AND PRACTITIONERS. By ALBERT CARLESS, C.B.E., M.B., M.S.Lond., F.R.C.S. Tenth edition. With 33 plates; pp. xii + 1562. Price 30s. net. London: Baillière, Tindall and Cox, 1920.

The increased size and price of this new edition are the result of the addition of new matter under the headings of: (1) The Treatment of Infected Wounds; (2) The Treatment of Compound Fractures; (3) Orthopædic Surgery, and (4) An Appendix on Military Surgery. The author expresses the hope that the inclusion of a large part of these new elements is temporary and that with the passing of the factors which have recently emphasized their importance, they will be eliminated without lessening the usefulness of the text-book. The Military Appendix, above mentioned, deals with war wounds and their treatment in general, followed by a more detailed consideration of wounds of the knee-joint and of the abdominal viscera. The number of illustrations has been considerably increased and the reproductions of radiographs have been collected to form a series of illustrations on art paper at the end of the volume, in place of their former scattered position throughout the text. Apart from these changes, the new edition follows closely on the well-known lines of its predecessors.

BACTERIOLOGY AND MYCOLOGY OF FOODS. By FRED WILBUR TANNER, M.S., Ph.D. With illustrations; pp. vi + 592. Price 28s. New York and London: John Wiley and Sons, 1919.

The book supplies in detail information, instruction in methods and explanations of apparatus necessary for those who propose to qualify themselves for food control, who seek to become food chemists or students of household science. The subject is at once wide and complicated, but the method adopted in the volume removes difficulties and renders complexity simple. Although essentially a matter for experts, all students equipped with a working knowledge of chemistry can master the bacteriology and mycology of foods. The writer describes bacteriological apparatus and the technique of collection, growth, staining and recognition of bacteria. The methods and principles of sterilization and disinfection are studied from the point of view of practice. Foods and their classification, with their liability to contamination, come under review. Mycology—yeasts, moulds, fungi—introduces the subject of bread and its examination. We are taught the essentials about the flora of the intestines, and the physiological and pathological bacteria of the whole alimentary tract are classified and described. The practical aspects and methods of searching for bacteria in the air, in water and in such foods as milk, milk products, eggs and egg products, meat and its products—e.g., extracts, are all carefully gone into and fully described. Indeed the practical examination of foods and the detection of unfitness occupy many sections and these, with those which deal with water hygiene, form an important part of the work. Food preservation, so serious a problem of civilized life, is studied, and the methods of detecting defects in canned foods, tomato products, with the cognate theme of food poisoning and ptomaines, are added. The writer glances more briefly, although aptly, at the methods most valuable in investigating epidemics. Appendices containing useful technical memorabilia are added.

VICIOUS CIRCLES IN DISEASE. By JAMIESON B. HURRY, M.A., M.D.Cantab. Third Edition. With illustrations; pp. xx + 377. Price 15s. net. London: J. and A. Churchill, 1919.

The interrelation of the functional activities of the organs of the body makes for physiological unity but also for reaction in disease, so that morbid departures cannot be regarded as entities, since, if one member is sick all the members suffer. Bichat taught that men died from affections of the heart, or of the lungs, or of the brain. Modern notions of pathology insist that no such differentiation between the organic systems is possible. In this volume are set forth the various facts which prove the truth of the above statement. Disease engendered in the heart may have its starting point elsewhere and its effects spread so that morbid processes invade the lungs, the kidneys, the central nervous system, and so on. In their turn such departures from the normal react upon the heart and circulation and so increase the morbus cordis. By a consideration of each system in order and the interrelation of pathological happenings with their consequences in remote systems and organs, the writer reveals how a vicious circle is established and by its revolution superinduces a series of reacting lesions, and these, unless the vicious circle is broken, end in systemic death. Although the establishment of a vicious circle in disease is commonly spoken of in medical literature the systematic study of such vicious circles has been neglected. This work undertakes to correct the omission and does so with much elaboration and meticulous care. The writer extends his view beyond the human organism—although that remains his chief preoccupation—and discusses the vicious circle of disease among the lower animals and in the vegetable kingdom. Important sections deal with ways in which the vicious circle may be broken, by the *vis medicatrix Naturæ*, by artifex whether therapeutic or surgical, and give the reasons for treatment by rational methods. An exhaustive index renders reference to the various sections of the book at once easy and satisfactory.

OCCUPATIONAL AFFECTIONS OF THE SKIN; THEIR PREVENTION AND TREATMENT; WITH AN ACCOUNT OF THE TRADE PROCESSES AND AGENTS WHICH GIVE RISE TO THEM. By R. PROSSER WHITE, M.D.Ed., M.R.C.S.Lond. Second Edition. With 24 plates; pp. xiv + 360. Price 25s. net. London: H. K. Lewis, 1920.

This book has grown considerably since the first edition. It contains 360 pages instead of 165 and is embellished with twenty-seven photographs instead of two. It also includes a terminal quatrain of verse and is much more expensive than it was in its youth. It contains a mine of information on all industrial diseases of the skin, and there is also a chapter on "Zoëtic Dermopathy," which means the cutaneous manifestations of microbic and fungoid infections contracted by those who have to do with animals of various kinds, the parasites of sponges and rose thorns. Nor are war conditions omitted. Great industry has been displayed by the author in compiling this work, which covers ground of great importance. In addition to his own observations references will be found to practically all the papers bearing on the subject, collected from all sorts of different, oftentimes obscure, sources. It will be found valuable, not only by dermatologists, but by all those who deal with industrial diseases and their consequences.

PSYCHOLOGY FROM THE STANDPOINT OF A BEHAVIORIST. By JOHN B. WATSON, Professor of Psychology, the Johns Hopkins University. Pp. xii + 429. Price 10s. 6d. net. Philadelphia and London: J. B. Lippincott, 1919.

Though the term "behaviorist" would probably have made him writhe and he would have been surprised to find the study of behaviour as an index of mental processes described as "purely an American production," the late Charles Mercier would have been the proper person to review this book, for none could better have appreciated the attitude of a writer who defines psychology as "that division of natural science which takes human activity and conduct as its subject-matter." Nor would Mercier's expression of his views have been lacking in "pep." The author's method aims at being biological and a large part of the book is devoted to the anatomy and physiology of the nervous system and of the organs concerned in the reception of stimuli and the activities resulting from stimulation. It is probably this fact which enables him to "get along without" the terms "sensation, perception, attention, will, image and the like," of which, in his preface, he says "I frankly do not know what they mean." Situations which would make the use of such terms convenient can of course be evaded or talked round, but, after all, is there any special virtue in the pose of calling a spade an agricultural implement? Misgivings as to the author's competence to deal with biological problems may well be aroused by perusal of the short paragraph devoted to the recapitulation theory which he describes as "a rather persistent but nevertheless pernicious theory" which has grown up "under the influence of several non-biologically trained psychologists." Chapters are devoted to the emotions, instincts, habits and personality, and these sections of the book have their utility though there is nothing particularly novel about them. Some interesting accounts of observations on and experiments with children will add to the common stock of knowledge and it is not the fault of Professor Watson if the results obtained are largely negative. Perhaps the most valuable chapter is that on "The Organism at Work" in which modern applications of psychological methods are dealt with and in which there is a very necessary "caution on all efficiency experimentation."

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SUPPLEMENT

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NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

La Tension Arterielle en Clinique, sa mesure, sa valeur séméiologique. Par le Dr. L. Gallavardin. Deuxième edition; avec 200 figures; pp. xii + 717. Price 30f. net. Paris: Masson et Cie, 1920.

The first edition of this work was published in 1910, and although the present volume is styled a second edition, it is so much larger that it is practically a new book. The first 230 pages are devoted to a consideration of the various methods of recording blood-pressure, and as a result of the author's extensive experience, he comes to the conclusion that the Riva-Rocci is the best method for recording the systolic or maximal, and the vibro-auscultatory for the diastolic or minimal pressure. He considers there should be a standard method for recording pressure and advises that the method adopted should always be stated, and the notation employed described in fractions in millimetres Hg. He insists on the importance of recording the residual systolic pressure. The full notation would therefore read: Tension (palpo-vibratory), 175-160/90, the first figures representing the systolic pressure including the residual, and the latter the diastolic. The physiological modifications are then considered in a section containing 20 pages, and 300 pages are devoted to pathological modifications. Hypertension, permanent and transitory, and hypotension on the same lines, receive complete consideration as well as modifications in various regions of the body. Those diseases which have not found a place in this part of the work are described in a chapter of 50 pages dealing with arterial tension in disease. A chapter of 50 pages on treatment ends this most excellent work which is one of the most authoritative pronouncements to be found in any language and is indispensable for anyone desirous of being acquainted with the subject with which it deals. The table of contents might with advantage be placed at the beginning and the index might be enlarged. We do not subscribe to the author's method of translating the titles of all the papers in the excellent bibliography to be found at the end of each chapter into his own language; it is also much easier for anyone desirous of looking up a reference to be given the year, volume, and page, rather than the year and date of the month.

On Gunshot Injuries to the Blood-vessels: Founded on Experience gained in France during the Great War, 1914-1918. By George Henry Makins, G.C.M.G., C.B. With 4 plates and 60 illustrations in text; pp. xii + 251. Price 21s. Bristol and London: John Wright and Sons, Ltd., 1919.

More than half this book is devoted to a study of wounds of individual vessels; to "documents," case-records, with the necessary elucidation, which will prove a storehouse of information for all future writers upon the subject. Many of the author's views were already known from his previous papers, but here, and in the War Museum, are the material facts for everybody's consideration. It has long been supposed that injury to the intima, or inner coats of large vessels, probably played some part in the production of certain "idiopathic" aneurysms, and colour is lent to the view by the revelation of important results of trivial defects produced at a distance from the main vascular injury. The clear distinction of arterial hæmatomata brought about by studies of gunshot wounds in the Russo-Japanese war has been further illuminated by a description of the mode of formation of the sac therefrom. Greater exactitude is introduced into the relations of vessels entering into aneurysmal varices and allied lesions. Great attention is paid to the symptoms and signs of lesions of the blood-vessels; to the discrimination of the signs of hæmorrhage and of interference with peripheral circulation; to the effects upon the circulation as a whole; to the production of murmurs; and to the elucidation of the late effects of occlusion upon the vitality of the parts supplied. The respective shares of simple deprivation of freely-circulating blood and of disturbed trophic innervation, due either to the same cause acting upon the nerve trunks or upon associated injury to them by the original trauma, cannot yet be conclusively separated. The reasons for disappointment with the high promise of angiorrhaphy at the outset of the war—mainly sepsis—are disclosed and the hopeful state in which vascular-suture remained at the end, when better methods of controlling infection of wounds had been generalized, are clearly stated. Temporary re-establishment of the circulation is shown to have a distinct though restricted field. Whether from the clinical, the histological, the operative point of view, this book is not only of great interest, but what is more, it is transparently honest and intensely practical. That there may be no present opportunity of turning its conclusions to account on a great scale in no way detracts from the value of observations which have their bearing also upon everyday problems of the circulation.

AN INTRODUCTION TO THE THEORY OF STATISTICS. By G. UDNY YULE, C.B.E., M.A. Fifth edition. With 53 figures and diagrams; pp. xv + 398. Price 12s. 6d. net. London: Charles Griffin and Co., 1919.

It is not necessary to discuss Mr. Yule's theory of statistics in detail. It is now so well known and appreciated that the call for a fifth edition is not at all surprising. The chief change is the addition of two supplements by Mr. Greenwood, the first on the law of small chances and the second on tests of goodness of fit. Both of these subjects are somewhat difficult, but the treatment of them by Mr. Greenwood should be intelligible even to those who do not possess extensive mathematical knowledge. The literature has also been brought up to date and the list of the most important papers that have been published in the last few years should be of very considerable use.

ON FACIAL NEURALGIA AND ITS TREATMENT, WITH ESPECIAL REFERENCE TO THE SURGERY OF THE FIFTH NERVE AND THE GASSERIAN GANGLION. By J. HUTCHINSON, F.R.C.S. With 37 illustrations; pp. xiii + 216. Price 15s. London: John Bale, Sons and Danielsson, 1919.

This volume forms the second edition of the author's monograph, on the same subject, published many years ago, and includes an amplification of the Jacksonian Prize Essay, awarded him in 1915. The work incorporates his latest views and practice based upon further experience. The fact may be conceded that trigeminal neuralgia is one of the most distressing forms that a surgeon is called upon to treat, while the author admits that a successful operation for its relief—one which will effect a lasting cure—is probably the most delicate, difficult and arduous procedure throughout the domain of surgery. The subject from the operative point of view forms the major part of the volume, and the operative results are compared with the alternative method of obtaining relief by means of injections of alcohol But while the reasons are given for the belief that this method is of great value, the author is of opinion that it will not displace the operation on the Gasserian ganglion as a permanent curative procedure. In support of this, cases are related in which temporary relief was obtained by injections of alcohol, and in which the ganglion operation was required for permanent cure. By means of the temporal route in operating, the flap of skin is made inside the hairy scalp, so that the scar is subsequently concealed by the growth of the hair. The author describes the ganglion operation he performs in full and precise detail, insisting upon the importance of avoiding all injury to the ophthalmic division. In discussing the various theories which have been advanced, in accounting for epileptiform neuralgia—tic douloureux, he affirms that "there is not the smallest vestige of proof" that the disease depends upon a neuritis, spreading from an inflamed dental nerve to the Gasserian ganglion, and so to other branches of the fifth. Despite this statement his notes of cases record "as usual" the previous removal of teeth—with no good effect whatever. A complete bibliography of the subject is added at the end of the volume, from 1896 to 1914, included in three sections, followed by an index.

A MANUAL OF X-RAY TECHNIC. By ARTHUR CHRISTIE. Second edition; with 48 illustrations; pp. x + 152. Price 6s. net. Philadelphia and London: J. B. Lippincott Co., 1917.

This short manual, the first edition of which was published in 1913, was written with a view to the needs of the medical service of the United States Army, and of those physicians and surgeons in private practice who might want to do their own X-ray work for the purpose of diagnosis. In this, the second edition, the manual has been revised in accordance with the important improvements in apparatus, and the increased knowledge gained from experience during the past few years. In addition to some minor changes and additions made in this revised edition a description of the Coolidge tube and a chapter on X-ray therapeutics have been added, the chapter on gastro-intestinal examination has been re-written and newly illustrated, and the chapter on diseases and injuries of the bones has been made more complete. The terminology which has been adopted by the American Röntgen Ray Society is used throughout this book. For the benefit of those readers who are not conversant with it this terminology is explained in the preface. The type is clear and there is a good index.

THE PHYSIOLOGY OF VISION, WITH SPECIAL REFERENCE TO COLOUR BLINDNESS. By F. W. EDRIDGE-GREEN, C.B.E., M.D., F.R.C.S. Illustrated: pp. xii + 280. Price 12s. net. London: G. Bell and Sons, 1920.

Naturally, being a monograph, this book takes a wide view of the subject, and, at the same time, affords the author the opportunity of reiterating the conclusions, especially on colour vision, at which he has arrived, after a series of investigations prolonged over a period of thirty years. Based upon his book "Colour Blindness and Colour Vision," published in the International Scientific Series, in 1890, and upon the large number of communications which he has since contributed to various scientific societies and journals, such as the Royal Society, the Ophthalmological Society, the British Association, the *Journal of Physiology*, and many others, the volume forms a concise reproduction of the advancements with which the author's name is so widely identified. The book consists of thirty chapters; part of these deal mostly with the purely physiological aspects of vision, and the remainder with the questions concerned in colour vision, and the description of the tests associated with the detection of colour blindness. The final chapter discusses the objections to other theories of vision and colour vision formerly in vogue, with which the author's views are at variance. In this regard he critically applies his views to the Young-Helmholtz and Hering theories and shows that the latter do not explain the phenomena of colour vision in a satisfactory manner. The soundness of Dr. Edridge-Green's work seems to appeal for acceptance, inasmuch as in no instance has it ever been successfully controverted.

TUBERCULOSIS AND PUBLIC HEALTH. By H. HYSLOP THOMSON, M.D., D.P.H. Pp. xi + 104. Price 5s. London: Longmans, Green and Co., 1920.

In this little volume Dr. Thomson has done well to call attention to the study of the problem of tuberculosis in relation to public health. The more one studies tuberculosis in a broad and comprehensive spirit the more one is compelled to agree with the author when he says that it is intimately connected with the social and economic conditions of the people, and that its death-rate constitutes a fairly accurate index of the health standard of the community. Academic and experimental knowledge of tuberculosis cannot help us very much to elucidate its complex problems. When the physician descends from the laboratory to the arena of life and sees face to face the conditions under which people live, their poverty and insanitation, their slums and overcrowding, their squalor and misery, their worry and anxiety, he comes to see the importance of the social aspect of tuberculosis rather than its specific nature. The universal prevalence of tuberculosis, and its commencement in childhood, rob a great deal of the value and significance of the infection theory. As to milk infection, on which Dr. Thomson lays such stress, one must remember that many children who drink tuberculous milk remain perfectly healthy, while in such countries as Japan, China, India, Siam, &c., where little or no milk is taken, and where tuberculous cattle are more or less absent, tuberculosis is widely and increasingly prevalent at the present day. The problems of tuberculosis are really social problems, hence their close connexion with public health; and the only sure means of eradicating the disease lie in making every effort to improve the social and economic condition of the people. Therefore all those schemes that aim at abolishing poverty, slums, insanitation, and in promoting better housing, village settlements, open-air schools, &c., many of which the author briefly describes in this volume, are measures directed not only towards the treatment but towards the prevention of the disease.

THE SEXUAL DISABILITIES OF MAN AND THEIR TREATMENT AND PREVENTION. By ARTHUR COOPER. Fourth edition. Pp. viii + 266. Price 7s. 6d. net. London: H. K. Lewis, 1920.

This new edition of the author's book contains two new chapters on "Hygiene" and "Continence" respectively, and a short account is added on the subject of "Sexuality and War," including the views of some of those whose war experience had brought sexual conditions in the troops under their observation. The fact, however, is noticeable that this experience was very variable. Sir Frederick Mott, for example, affirms that in cases of war neurasthenia, sexual desire was largely in abeyance owing to the preponderating influence of the emotions concerned with the instinct of self-preservation. Gonorrhœa, it is stated, was the most common disease of the malingerer. Five cases came under observation in which the men had paid a comrade to allow them to contract it, so that they should not be sent back to the firing line. The work has been thoroughly revised, parts have been re-written, and some parts in the former edition omitted. An index of authors, and a general index of the contents, conclude the volume.

ON LONGEVITY AND MEANS FOR THE PROLONGATION OF LIFE. By Sir HERMANN WEBER, M.D., F.R.C.P. Edited by F. PARKES WEBER, M.D., F.R.C.P. With a preface by Sir CLIFFORD ALLBUTT, K.C.B., F.R.S. Fifth edition. Pp. xxii + 291. Price 12s. 6d. net. London: Macmillan and Co., 1919.

This fifth edition of the late Sir Hermann Weber's book was already in type at the time of his death. Dr. Parkes Weber consequently was called upon to edit it—a duty which practically consisted in correcting the proofs, while Sir Clifford Allbutt contributed the preface. The author's long life confirmed the teaching laid down in his book: he practised what he taught, and, judging from the repeated editions of his volume which have been called for, it is probable that many, especially of the laity, have been influenced by his advice to follow his example in ordering their lives.

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SUPPLEMENT

(Vol. XIII, No. 9, July, 1920).

NOTES ON BOOKS.

[*The purpose of these "Notes" is not so much to praise or to blame as to draw attention to and describe some of the new books and new editions which have been added to the Society's Library.*—Ed.]

Modern Medicine and Some Modern Remedies: Practical Notes for the General Practitioner. By Thomas Bodley Scott. With a Preface by Sir Lauder Brunton, Bt., F.R.S. Second edition. Pp. xi + 198. Price 6s. 6d. net. London: H. K. Lewis and Co., Ltd., 1919.

No preface introductory to this the second edition of the author's work accompanies the volume, and the repeated description of Sir James Mackenzie as "Dr." James Mackenzie would suggest that the edition is a reprint of the first. The main features of the work include chapters on disorders of the heart, arteriosclerosis, therapeutic speculations and doubts, chronic bronchitis, and bronchial asthma. The essays, we learn, were written at odd times and in odd places, and embody many practical details gathered from the author's experience. The most suggestive of the series is that on therapeutic speculations and doubts, in which some interesting details are related of endocrine treatment. The author might be reminded to add to the usefulness of his work by including an index in the next edition.

Inguinal Hernia: the Imperfectly Descended Testicle and Varicocele. By Philip Turner, M.S., F.R.C.S. With 22 illustrations; pp. viii + 104. Price 9s. 6d. net. London: J. and A. Churchill, 1919.

In this interesting and well illustrated little book the author describes the operations he performs for the three conditions mentioned in the title. He considers that Hamilton Russell's theory of the congenital origin of the sac in the vast majority of cases has been conclusively proved. In children or young adults, when secondary changes have not occurred in the walls of the canal, he therefore simply removes the sac. When such changes have occurred or the musculature is deficient he performs Bassini's operation. For imperfectly descended testis, Mr. Turner has devised an entirely new operation in which, after freeing the testis, he passes it through a small hole in the septum into the opposite side of the scrotum, so that in unilateral cases both testicles lie together in the same half of the scrotum, and in bilateral cases each occupies the opposite side. In varicocele, after ligature and excision of the veins, he sutures the vertical incision in the sheath of the cord horizontally, thus shortening the cord and supporting the testicle.

CAVERNES PULMONAIRES ET PHENOMÈNES CAVERNEUX. Par le Dr. CH. SABOURIN. Pp. 147. Price 3 fr. Paris: Masson et Cie, 1919.

This small monograph deals with the subject of pulmonary excavation. The first part is devoted to the general symptoms of excavation revealed by percussion, palpitation, ascultation, radioscopy and radiography, fever and expectoration. The second part of this work is concerned first with cavernous sounds, existing in the absence of cavities, and next with cavities which have cavernous symptoms. The conditions in which these phenomena arise are dealt with at some length, and it is pointed out that in true excavation loud cavernous sounds are apt to disappear and re-appear. An explanation is sought as follows: That the cavity itself causes the auscultatory phenomena, but that these are often too faint to be transmitted to the ear, and that for this to take place there must be a reinforcing and transmitting apparatus formed by consolidation of a pulmonary lobe, which would explain why these sounds fade away when the surrounding lung has recovered its permeability and become apparent, and when the lung again becomes a very condensed tissue. Dr. Sabourin believes that the problem should be solved by comparative investigation of tuberculous foci without cavernous signs, of tuberculous foci, with permanent or temporary cavernous signs, and of results given by necropsies under favourable circumstances.

THE TRANSMUTATION OF BACTERIA. By S. GURNEY-DIXON, M.A., M.D.Cantab., M.R.C.S. Eng., L.R.C.P.Lond. Pp. xviii + 179. Price 10s. net. Cambridge: University Press, 1919.

The author points out in his preface that the essay on which this book is founded was compiled from notes and observations collected previous to 1913, and that the completion of the work was interrupted by war service in France, where means did not exist for bringing the references up to date. The book opens with a discussion on the scope of the inquiry, in which the definitions of terms, such as transmutation, spontaneous and impressed variation are examined, together with the difficult questions of classification of bacteria. Separate chapters are devoted to variations in morphology, fermenting power, virulence, and pathogenicity. Considerable detailed attention is also given to questions of changes in environment and the possibilities of contamination. The author concludes that (1) variation in every character of bacteria may occur; (2) that these variations may be spontaneous or impressed by conditions of environment; (3) that transmutation differs from evolution and variation merely in degree; and (4) that the occurrence of transmutation in the human body is not capable of proof, though it is suggested by circumstantial evidence. A synopsis and a list of references are given, but there is no subject index.

WHEELER'S HANDBOOK OF MEDICINE. By WILLIAM R. JACK, B.Sc., M.D., F.R.F.P.S.G. Sixth edition. Pp. xv + 561. Price 12s. net. Edinburgh: E. and S. Livingstone, 1920.

This useful handbook has now reached its sixth edition, and deserves the wide circulation that it has attained. Whilst the space allotted to each malady is necessarily compressed, a survey of the book fails to discover any noteworthy omissions. Every article has been carefully revised and brought as nearly as possible up to date. The chief alterations occur in the sections devoted to nervous diseases and the specific infections, acute poliomyelitis having been transferred from the former to the latter group. The article on tumours of the cord has been amplified, and a brief description of myasthenia gravis added to the diseases of muscles. New articles appear on Landry's paralysis, compression paraplegia and caisson disease. The final section devoted to medical diseases of the war constitutes an exceedingly apt summary. In a new edition encephalitis lethargica might perhaps receive more adequate mention, and the treatment of pulmonary tuberculosis, rheumatoid arthritis and other infective processes by intensive iodine administration might be included. The more liberal use of small type has kept the size of the book within reasonable and handy compass.

THE SYMPATHETIC NERVOUS SYSTEM IN DISEASE. By W. LANGDON BROWN, M.A., M.D.Cantab., F.R.C.P.Lond. Pp. xi + 161. Price 10s. 6d. net. London: Frowde, Hodder and Stoughton, 1920.

The inter-action of the endocrine glands with the autonomic nervous system is a fascinating problem which is now occupying a very prominent place in the world of medicine, not only for its physiological interest, but for the therapeutic possibilities connoted. The present volume—an amplification of the Croonian lectures of 1918—emphasizes the main plan of the nervous element, with particular reference to clinical considerations, and it is elaborated with the clearness of exposition and felicity of expression one expects from a clinician who is also a physiologist. The first chapter is devoted to the anatomical plan of the autonomic nervous system and the physiological effects which result from influencing the component parts—the sympathetic and the parasympathetic fibres—with the action of drugs in stimulating or paralysing these fibres. From this the author proceeds to point out that the adrenals, the thyroid and the pituitary have in common the three characteristics: that the secretion of each is stimulated by the sympathetic; that all lower carbohydrate tolerance, and that all act and re-act with the reproductive organs. Again, the reciprocal action is emphasized in the statement that the sympathetic nervous system stimulates secretion of these glands, the secretion of which in turn increases sympathetic response. Glycosuria receives very detailed consideration, and its differentiation from hyperglycæmia enables the author to include some pertinent remarks, dealing with the ventilation and disposal of misconceptions relating to diabetes mellitus. Thus the fact that sugar appears in the urine does not imply that no more can be utilized, but merely that the method of utilization cannot be brought quickly enough into play to prevent some escape, thus explaining why in the normal individual glycosuria *ex amylo* cannot occur. It explains, too, some therapeutic considerations employed hitherto empirically. Diabetes insipidus is analysed as resulting from four causes, disorders of the pituitary body, looming largely in its production. When one comes to the organs of digestion many conditions of "nervous dyspepsia" become clear when we assume increased susceptibility of the reflex nervous mechanism and the existence of lesions external to the stomach. The sympathetic nervous system in diseases of the circulatory system has important bearings upon the blood pressure, such considerations as advisability of its reduction by venesection, drugs, or lumbar puncture in cases of cerebral hæmorrhage; the meaning of œdema of the lungs; the consideration in general of hypertension and hypotension; and the real meaning of "shock."

The subject culminates in the now familiar vagotonia of Eppinger and Hess. The author analyses the opinions of the writers quoted with impartial but strict criticism. The general idea of vagotonia is acceptable to him, some of the details he is unable to assimilate. The vagotonic is a person who is unduly sensitive to a drug which stimulates the vagus, and, on account of the exaggerated tone of the centre, unduly insusceptible to the action of a paralysing drug. In other words he is very responsive to pilocarpine and relatively resistent to atropine. Increased vagus action will explain spasm of bronchial muscles and vaso-dilatation of bronchial mucosa (asthma), laryngeal spasm, bradycardia, hyperchlorhydria, low blood-pressure, respiratory arrhythmia, anginal attacks, tenesmus. Similarly sympathetic over-action causes the familiar symptom of sweating in Graves's disease (although Eppinger and Hess appear to regard this as a parasympathetic phenomenon). Another objection to the theory of Eppinger and Hess advanced by Dr. Langdon Brown is that pyloric spasm is described as a feature of vagotonia, although the sphincter is innervated solely by the sympathetic.

If, as the author suggests, one formulates a hypothetical hormone—perhaps pancreatic—which exerts a continuously stimulating action on the whole parasympathetic system as adrenalin acts on the sympathetic, unbounded therapeutic possibilities occur for many obscure and obstinate neuroses. Throughout the book the emotional element is explained in terms of sympathetic stimulation, e.g., emotional glycosuria, inhibition of digestion, the effect of pain, fear, rage, intense excitement—the freedom of katabolic activities which are primarily defensive in origin. And the subject is aptly summarized in the final chapter, which explains the familiar psychoneuroses in terms of development of an exaggerated response of an originally defensive mechanism. "Designed as an intensive preparation for action or defence, the sympathetic response may be so dissociated, perverted or prolonged as to produce through the thyroid, Graves's disease . . . . . through the pituitary body, diabetes insipidus . . . . . through the pancreas, diabetes mellitus: it may disorganize digestion, it may keep blood-pressure at a level which is inappropriate for the task of the heart and arteries."

THE PRINCIPLES OF ANTE-NATAL AND POST-NATAL CHILD PHYSIOLOGY, PURE AND APPLIED. By W. M. FELDMAN, M.B., B.S.Lond. With 6 plates and 129 illustrations; pp. xxvii + 694. Price 30s. net. London: Longmans, Green and Co., 1920.

The author explains very fully in the preface what is the plan of the book, and the different classes of readers to whom it may appeal. The ordinary physiological student will find all the phenomena of child life described with great fullness of detail. Pædiatric physicians and all persons interested in the scientific study of childhood will find much that is informative and useful. The specialist in child physiology will find a detailed account, with full references to the literature, of what has been done practically up to the date of publication in any particular line of research. As a precise knowledge of the anatomy, macroscopic as well as microscopic, of the organs in early life is necessary for a thorough understanding of their physiology, this also is supplied. We may add that the principles of mathematics, of engineering, and of electrical action are applied with a familiarity which breeds respect in the ordinary reader. In carrying out these ideas Dr. Feldman has produced not a book but a cyclopædia of information bearing on the period of life dealt with. This entails a consideration of the ante-conceptional and conceptional germinal stages, the post-conceptional or intra-uterine stage, the natal stage, and the post-natal stage up to adolescence. Facts and observations on all the phases of early life are poured out in overwhelming abundance. These are derived largely from foreign writers, and the author states that but little work bearing on the subject has been done in this country, with the exception of Dr. Ballantyne's original contributions. Dr. Feldman has been charting for most of us an unknown sea. The chart is not yet absolute and final, for much of physiological teaching has to be scrapped periodically, but this chart will serve for the present. To physicians generally we commend the book as a standard work of reference. To those dealing more particularly with children, and desirous of instructive and stimulating reading, we promise a full return. The author has not only mastered his facts, anatomical and physiological, but he discusses them critically from his own point of view. Where so much is obscure he aids the reader by pointing out where truth or probability lies, and also the bearing of the facts on clinical medicine.

THE ENGINES OF THE HUMAN BODY: BEING THE SUBSTANCE OF CHRISTMAS LECTURES GIVEN AT THE ROYAL INSTITUTION OF GREAT BRITAIN, CHRISTMAS, 1916-1917. By ARTHUR KEITH, M.D., LL.D.Aberd., F.R.S. With 2 plates and 47 figures in text; pp. xii + 284. Price 12s. 6d. London: Williams and Norgate, 1919.

The method adopted by the author of this extraordinarily convincing book is to compare the systems of the human body to mechanical engines. Thus, muscles acting upon bones are levers, so planned that either great "lift" or extreme speed in reaction is obtained according to the requirements of the body. The motor bicycle with its complex arrangements for rapidity of motion, its control and its system of "feed," is taken as an exemplar of mechanical transport. Bone, muscles, joints with their mechanical properties are explained by likening them to the parts of the motor. Every detail finds a place and the descriptions are so clear and accurate, that he who runs may read. The machine may fail to work through lack or excess of heat, but the body by nice adjustments of its temperature prevents undue cooling or overheating. The circulation, the emunctories and their nerve control are explained in their rôle as heat regulators. The machine must cease working or it wears out; the human engines toil day in and day out, and by perfect adjustment the working wear is repaired even while the travail goes on. The machine must be fed with petrol, but the body not only needs food—it must transform the raw material into the requisite pabulum. It needs further to be able to cleanse away the products of its work and dispose of excess of food metabolism. Digestion—the "workshops" of the body—elaborate the finished product for each engine supply, be it bone, muscle, nerve or the special senses. Their industry is unstinted, unrestricted by hours of labour, and their system of transport may not break down. The renovation of the tissues, their adaptation to the needs of growth or decay is the concern of countless "workers," the artificers of bone, of muscle, of gland, or of brain cells. Then there are the fighters, the phagocytes, which are always under arms, and float in the great stream of life to contest against invasion by alien hosts of disease. What the motor lacks is initiative control, but man has his elaborate systems of a central control, the brain and a telephone system which in health never breaks down. The needs of every tissue is communicated to the "G.H.Q."—the brain, and it issues instant orders for, it may be, a better blood supply, or increased activity of some section of the body builders. Into this fascinating story come flashes of biographical incidents; we are told of Fabricius of Padua, Servetus, the grave Harvey, of John Hunter and Stephen Hales and others of the giants which were in bygone days.

HANDBOOK OF PHYSIOLOGY. By W. D. HALLIBURTON, M.D., LL.D., F.R.C.P., F.R.S. Fifteenth edition (being the twenty-eighth edition of Kirkes' Physiology). With nearly 600 illustrations, some coloured; pp. xix + 936. Price 18s. net. London: John Murray, 1920.

In a foreword is given the interesting story of the evolution of Dr. Kirkes' "Manual," with the comprehensive system of physiology as Professor Halliburton has made it. It recalls to the older generation what the teaching of physiology was and what the needs of science now demand that it should be. The book comprises a brief general introduction explicative of the relations of morphology, biology and physiology. The minute anatomy of the tissues with an account of their life attributes follow and are succeeded by clear descriptions of the more highly developed structures. The functions of circulation, respiration, digestion, metabolism, and of the ductless and other glands are brought before the reader with extreme clearness. Indeed the method pursued and the happy mode of expressing the writer's meaning render the whole book vividly realistic and readable. The chemical aspect of the subject, for example, is often difficult to grasp and still more difficult to retain in the memory; but in this book there is a great deal of chemistry yet its expression is interesting and easily mastered. The exposition of the central and peripheral nervous system covers many pages and it, together with the cognate subject of the special senses, is thorough and clear. The manual, although primarily dealing with physiology, contains much extraneous information of value to medical men. There is a useful chapter on war diet, and the subject of vitamines receives attention. The illustrations, which are numerous, certainly elucidate the text.

ESSENTIALS OF TROPICAL MEDICINE. By WALTER E. MASTERS, M.D.Brux., M.R.C.S., L.R.C.P.Lond. With illustrations; pp. vi + 702. Price 42s. net. London: John Bale, Sons and Danielsson, Ltd., 1920.

In the words of the author, this volume is "a digest of our knowledge of Tropical Medicine . . . in a form handy for ready reference: the vade-mecum of the student and busy tropical practitioner." It bears the somewhat ambitious title of "Essentials," from which one might naturally assume that it contains, generally speaking, only such information as it is really necessary to acquire. This of course must always be a matter of personal opinion, but we certainly feel that in this respect the publication is open to considerable improvement. Neither can we commend the principle of attempting to satisfy the requirements both of the student and the practitioner in a single volume. The student needs precise information, in a handy and readable form, on the subjects of his curriculum, without the necessity for selecting portions from a large book of reference. The practitioner requires chiefly a practical guide to the *details* with which he must be acquainted in the actual treatment of tropical diseases. The book is divided into sections, the first five of which deal with the distribution, pathology, symptomatology, treatment, &c., of the usual diseases of tropical climates; these are classified, so far as possible, on an ætiological basis: i.e., whether due to protozoa, bacteria, worms, &c. Then follow sections on diseases of the skin, diseases of the eyes, tropical hygiene and sanitation, and laboratory hints, together with an Appendix on some minor matters. There are several errors in the text which will doubtless be remedied in future editions of the work. The book is profusely illustrated.

L'INFECTION BACILLAIRE ET LA TUBERCULOSE CHEZ L'HOMME ET CHEZ LES ANIMAUX: PROCESSUS D'INFECTION ET DE DÉFENCE; ÉTUDE BIOLOGIQUE ET EXPÉRIMENTALE. Par A. CALMETTE. Avec 46 figures; pp. vi + 619. Price 55 f. Paris: Masson et Cie, 1920.

This large work by this well-known authority on tuberculosis is a biological and experimental study on tuberculosis. The morphology of the bacillus of Koch, the various methods of staining it, its culture and chemical composition receive full consideration, and tuberculins, toxins—both endo- and exo-toxins and the anatomo-pathological conditions—found in man and animals—are described in detail. The methods of infection are succinctly stated, the author being one of those who considers the digestive tract the most frequent method of infection. Tuberculosis in children has a chapter to itself and the writer believes no tuberculosis in children exists without tracheo-bronchial adenopathy. It is generally recognized that heredity plays a much more important part after birth than before birth, by the transmission of the tendency; but hereditary transmission before birth is not denied and in some circumstances can occur from infection of the placenta or at the moment of birth. Bovine tuberculosis and its appearance in various animals such as dogs, birds, &c., the reactions of defence of the organism against tuberculosis with a full description of the diagnosis by means of tuberculin reactions, serotherapy, vaccination, all are fully dealt with; in fact, nothing of value to the laboratory worker has been omitted. There are numerous references to authorities and there are a number of plates, some excellently coloured. The work is authoritative, as was only to be expected from a writer so well known, to whom, of course, we are indebted for the ophthalmic reaction.

HALF A CENTURY OF SMALL-POX AND VACCINATION. By JOHN C. MCVAIL, M.D., LL.D. Being the Milroy Lectures delivered before the Royal College of Physicians of London on March 13, 18 and 20, 1919. Pp. viii + 87. Price 5s. 6d. net. Edinburgh: E. and S. Livingstone (1919).

In the first lecture Dr. McVail provides numerous data to prove that natural small-pox attained its maximum of infectivity and mortality in this country in the pandemic of 1870-73, in which the case mortality reached as high as 45 per cent.. but that since that time it has greatly diminished in fatality as well as in infectivity and prevalence. This diminished fatality was evident in the epidemic of 1902-05, in which the unvaccinated rate was 19·3 per cent. and the general rate in the provinces, including vaccinated and unvaccinated, was 6·9 per cent. In London in 1901-02 there was a temporary reversion to a higher general fatality-rate, viz., 16·8 per cent, but the rate in the metropolitan area in 1903 was only 3 per cent. The 1902-05 epidemic is the last occasion on which small-pox was extensively prevalent in Britain, but data available for succeeding years points to the decline in fatality still being continued. Dr. McVail indicates that there have been two types of small-pox occurring simultaneously in Britain in epidemic years, more especially since about 1900, a severe type, the European or African; and a mild type, the American. He does not necessarily believe that the disease is dying out in this country, but ascribes the increasing mildness in the type of disease found in Britain to the gradual replacement of the severe or African type by the attenuated or American type. He points out that small-pox is still very prevalent in some European countries and in North Africa and that there is still a grave risk of a re-introduction into Britain of a severe type of the disease. The second lecture gives an account of vaccination past and present. Routine infantile vaccination has declined to such an extent that in the year 1912, which is the most recent year for which returns are available, only about half the children born in England were being vaccinated, while in Scotland, in 1916, of those who reached the age of 6 months 41 per cent. remained unvaccinated. As a result of vaccination in the Army being obligatory the adult population is, however, better protected by recent vaccination than in former years. Dr. McVail disposes of the argument against infantile vacination that it often makes subsequent small-pox so mild as to be unrecognizable with consequent spread of infection by missed cases, by pointing out that it is most desirable that small-pox should assume a milder and modified form in those cases in which the disease is not actually prevented, not only because it is less fatal but also because it is less infective, and he concludes that to discourage vaccination in order that the "unvaccinated individual may have an easily diagnosable (therefore possibly fatal) attack of small-pox appears to be contrary to the principles of medical ethics and to the interests of the public health." In the last lecture there is a detailed account of the measures of prevention and control of small-pox which, in Dr. McVail's opinion, in the absence of systematic vaccination and revaccination should be adopted in this country on the appearance of small-pox at the present day. He is convinced that the measures at our disposal should enable us to deal with any invasion by the disease from other countries in which small-pox may be prevalent, despite the fact that there has been such a decrease in systematic vaccination in infancy. Vaccination of contacts supplemented by isolation, disinfection and the other measures for controlling epidemics named above proving insufficient there would remain but one method which could be effective, namely general vaccination.

Common Diseases of the Skin, with Notes on Diagnosis and Treatment. By G. Gordon Campbell, B.Sc., M.D., C.M. Illustrated; pp. viii + 229. Price 21s. net. New York: The Macmillan Company, 1920.

That the author is experienced in both dermatology and therapeutics is shown by the contents of the book as well as by the appointments he holds. The inexperienced prescribe many remedies for each disease, but the author recommends the same remedy for many diseases. He does, however, vary the names: Ung. hyd. amm. and white precipitate ointment; ung. hyd. nit. and citrine ointment; green soap and soft. The lines of treatment, as simple as they are carefully selected, should serve as models for teachers in all branches of medicine. The illustrations are so numerous and typical that even those who have paid scant attention to diseases of the skin can acquire a sound basis of knowledge.

Mechanical Dentistry: A Practical Treatise on the Construction of the Various Kinds of Artificial Dentures, comprising also useful Formulæ, Tables, and Receipts for Gold Plate, Clasps, Solders, &c. By Charles Hunter. Seventh edition. With 102 illustrations; pp. xv + 266. Price 5s. net. London: Crosby Lockwood and Son, 1920.

The author of this work has carefully explained the technique employed by dentists during the last fifty years. The innovations referred to, viz., celluloid and continuous gum-work, occupy chapters which might almost be omitted, as they have had their day. Newer methods such as bridge-work and gold crowns seem to deserve notice in such a work. The chapter on pivoting is archaic and the illustrations extremely weak. The chapters on gold-work, the striking up of plates and the mounting of tube teeth are sound and complete, though nothing is said about casting plates, a method almost universally adopted for certain cases! Every student should read these chapters carefully, and are we not all students? The chapter on vulcanite sets forth everything it is necessary to know. Emphasis is laid on *time* in vulcanizing if the results are to be good; vulcanite needs no strengtheners if properly treated. The orthodox methods of constructing "obturators" are concisely explained and well illustrated. The notes on metals and materials used in dental work are valuable and reliable, but as it is not likely that dentists will melt down sovereigns again, many of the formulæ become obsolete. To-day refining and preparing of gold is in the hands of experts, who supply any carat or alloy to order. The notes on acids, &c., are always useful for reference. The book is well written, set up in good type, and may be regarded as a standard work.

ANATOMY, DESCRIPTIVE AND APPLIED. By HENRY GRAY, F.R.S., F.R.C.S. Twenty-first edition, edited by ROBERT HOWDEN, M.A., M.B., D.Sc. With 1,215 illustrations; pp. xvi + 1366. Price 42s. net. London: Longmans, Green and Co., 1920.

Within two years another edition, the twenty-first, of "Gray" has been called for. It contains forty-four more pages, and more than eighty new illustrations, than the previous edition, and a little calculation will show that upon an average, an illustration is incorporated with almost every page of the text. In the preface, the editor, Professor Howden, discusses his preference for the Basle nomenclature, which he has adopted. He sums up the position as follows: "A careful examination of the 'Basle' terminology shows that for the most part it does not consist of new terms but of a selection of old ones, together with such modifications and additions as make for accuracy and brevity in description. It would seem, therefore, reasonable to utilize the labour that has already gone to its construction, and to adopt this terminology as a basis for future revision, as suggested by American anatomists."

ANIMAL EXPERIMENTS AND SURGERY. HUNTERIAN LECTURES DELIVERED AT THE ROYAL COLLEGE OF SURGEONS OF ENGLAND ON FEBRUARY 13, 16 AND 18, 1920. By WALTER G. SPENCER, M.S., F.R.C.S. Pp. viii + 179. Price 6s. net. London: University of London Press, 1920.

This book contains the three Hunterian Lectures delivered by the author at the Royal College of Surgeons of England during February of this year. His theme is based upon the effort to show the definite association of the advance of surgery with experiments upon animals. The first lecture is devoted to historical details, the second discusses the application of surgery to organs and organized structures, the third to improvements in surgery. The fact is pointed out that some discoveries to which experiments upon animals have led, have resulted in improvements in medical treatment, and have rendered unnecessary much of what was indifferent surgical treatment. A full list of references to the subjects discussed is added at the end of the work, but there is no index.

MICROSCOPY: THE CONSTRUCTION, THEORY AND USE OF THE MICROSCOPE. By EDMUND J. SPITTA, L.R.C.P.Lond., M.R.C.S.Eng., F.R.G.S., F.R.M.S. Third edition. With 338 illustrations; pp. xxviii + 537. Price 25s. net. London: John Murray, 1920.

The size of this third edition of the author's work is a remarkable testimony to the development in the process of years, of the science of microscopy. So essentially has it now become a special branch of science, that it is possible to conceive of anybody, especially the medical student, being somewhat appalled at finding himself confronted with the task of acquiring a workable knowledge of it. However that may be, this text-book provides him with the means for undertaking the task, and in this new edition various improvements have been effected with the object of facilitating his mental assimilation. The work has been fully revised, and contains several new features. Mention may be made of the description of a comparatively new type of low power combination for studying and reproducing objects, such as whole flies, fleas, lice and suchlike creatures of large dimensions, at different magnifications. The absence of a satisfactory method of accomplishing this in the war created a difficulty, which has now been overcome. Again special attention is directed to the employment of some new short wave-length gelatine fibres to assist revolution when used in conjunction with high-power illuminants. The index has been enlarged by numerous new entries, in order to facilitate quick reference. The admirable plates are a noteworthy feature of the volume.

THE PROBLEM OF NERVOUS BREAKDOWN. By EDWIN LANCELOT ASH. Pp. xii + 299. Price 10s. 6d. net. London: Mills and Boon, 1919.

This work is intended to appeal to all those whose vocation in life is such that some knowledge of the manifold factors connected with nervous disorders should prove of use. In Part I the ætiology is discussed under the headings of the emotions, nervous temperament, and factors predisposing to and determining breakdown. In the second part, the varieties of nervous breakdown are dealt with, such as the neurasthenic state, morbid fears and doubts, nervous indigestion, hysteria, and multiple personality. The various hygienic measures for the correction of nervous illness are given at some length in Part III. The rest cure, diet, sleep, rest and recreation have special chapters devoted to them, while the special malfunctionings of childhood and youth also receive attention. The breakdowns of war conclude the book. It may be noted that the author is not a follower of the more advanced school of neurologists.

THE MAMMARY APPARATUS OF THE MAMMALIA IN THE LIGHT OF ONTOGENESIS AND PHYLOGENESIS. By ERNST BRESSLAU, M.D. With a Note by JAMES P. HILL, D.Sc., F.R.S. Pp. vii + 145. Price 7s. 6d. net. London: Methuen and Co., Ltd., 1920.

This is an extraordinarily interesting book, for which we are indebted to Professor Ernst Bresslau. It deals with the evolution of the mammary apparatus. The first lecture or chapter describes the origin of the mammary apparatus of the mammalia and its structure and development in the monotremata. In the second lecture the Professor describes the structure and development of the mammary apparatus in the marsupialia, and in the third the development of the mammary apparatus in the placentalia. These lectures, dealing as they do with the origin and development of an essential part of the higher orders of the animal creation are full of interest to students whether undergraduate or graduate, and those who turn to them for information cannot but be grateful for the clearness of the diction, the wealth of information, and for the many diagrams and pictures with which the book is enriched. The Professor remarks that "we have no longer to suppose that the mammary apparatus of the mammals arose *de novo* in this highest class of vertebrates, but we now know that it resulted from primitive structures developed in their oviparous ancestors for brooding purposes," and from the next paragraph we learn that the recognition of the structure and function of the brooding organs of lower forms is able to explain the possession of mammary organs by the male sex, in which, however perfect the structure may be, it does not normally function. The fact that many birds, and probably some beasts, take a share in the brooding and incubation of the young, explains the similarity of structure in the two sexes. As the Professor says—"the mammary apparatus then was not exclusively acquired by the female but arose quite naturally in both sexes alike, on the ground of inherited disposition, and not until the disappearance of its function in the male did it undergo a retrograde metamorphosis in that sex." It is, however, useless to attempt quotations from such a book; it ought to be read by those who are interested in the subject with which it deals.

FEVER NURSING, DESIGNED FOR THE USE OF PROFESSIONAL AND OTHER NURSES, AND ESPECIALLY AS A TEXT-BOOK FOR NURSES IN TRAINING. By J. C. WILSON, A.M., M.D. Ninth edition. Pp. 272. Price 7s. 6d. Philadelphia and London: J. P. Lippincott Co., 1919.

This book is one of a series of Nursing Manuals, admirably clear, well printed, and well got up in all respects. The first chapter deals with fever nursing in general, and is perhaps the most valuable part of the book. It defines the terms—fever, pyrexia, apyrexia, and hyperpyrexia, it dwells on the qualifications of a good fever nurse, and gives advice as to the preparation of a sick room, the avoidance of infection, and the other duties common to all fever nursing. In Chapter II there is a classification of fevers into continued, periodical, eruptive, and fevers with marked local manifestations. All this is interesting information which tends to make the nurse's work more intelligent, and therefore pleasanter to herself, more useful to the patient, and more acceptable to the doctor. In Chapter III nursing details are described, and in Chapters IV, V and VI the continued, periodical, and the eruptive fevers are considered, and the necessary variations of nursing treatment are passed under review. Chapter VII, the last in the book, deals with fevers of widely different significance, although it is true that all of them have marked local manifestations. This no doubt accounts for the juxtaposition of rheumatic fever, pneumonia, cerebro-spinal fever, and the plague!

OPERATIVE SURGERY : A COMPANION VOLUME TO A "MANUAL OF SURGERY." Vols. I and II. By ALEXIS THOMSON, F.R.C.S.Ed., and ALEXANDER MILES, F.R.C.S.Ed. Third edition, with 279 illustrations ; pp. xviii + 619. Price 16s. net. London : Henry Frowde, Hodder and Stoughton, 1920.

This book is admirably calculated to enable students to appreciate lectures on surgery and the course of surgical operations, and to systematize their knowledge so as to get it into operation form. The authors have been compelled by considerations of space to compress their information, but thanks to their own clearness and to the admirable diagrams with which it is furnished there is no confusion, and the purpose of the book is well served. Taken in conjunction with its companion volume, "A Manual of Surgery," by the same authors, it is a clear and ready guide through the enormous field of modern surgery. Enough anatomy is introduced to afford good landmarks, but the student is rightly supposed to have an adequate knowledge of this subject before he commences the study of surgical operations. It is not likely that Thomson and Miles' "Operative Surgery" will displace the larger volumes which are equally correct and lucid ; but not everyone has the means wherewith to purchase the larger books, nor is everyone able to luxuriate in reading them before taking his qualifying examination. To the majority of students this volume, clear, accurate, and well written, with its admirable diagrams, will be of inestimable service.

THE NEW PSYCHOLOGY AND ITS RELATION TO LIFE. By A. G. TANSLEY. Pp. 283. Price 10s. 6d. net. London : George Allen and Unwin, 1920.

Though this work is intended for the general reader, it should be read by all medical practitioners to whom a knowledge of the contents would prove a valuable adjunct in enlarging their conception of humanity on a scientific basis. Ignorance of the psychological and biological factors in deviations from the normal functioning in somatic disorders must be a great handicap to all medical men, while a true insight into the mental processes which may bring about unhappiness, anti-social attitudes, nervous and mental breakdowns, will not only give the family physician the opportunity of recognizing the importance of such in their early stages and aborting them—thus benefiting society, but will give him an interest in many patients who previously, perhaps, have only bored him. The author does not deal with ordinary text-book psychology, but here presents the knowledge that has been gained by the modern study of psychopathology. Here the psycho-analytic doctrines are mainly taken as established and the work of Freud, Jung and Trotter as accepted. Thus the unconscious, psychic determinism, the workings of the libido, repression, mental conflict, dreams, rationalization, the various instincts and complexes are dilated upon. The writer is evidently an enthusiast, and puts his matter in a clear and highly interesting way. No serious exception can be taken to anything the author says, though psychologists of an older school would regard much as debatable. The title correctly describes the contents, which can be highly recommended without any reservations.

PASTEUR : THE HISTORY OF A MIND. By EMILE DUCLAUX. Translated by ERWIN F. SMITH and FLORENCE HEDGES. Illustrated ; pp. xxxii + 363. Price 21s. net. Philadelphia and London : W. B. Saunders Company, 1920.

First published in 1896, in France, the senior translator, in the introduction to the present book, said that when he read it for the first time he wrote on its margin : "The most useful book I have read in a long time." Ten years later he sees no reason for altering his judgment ; hence this illuminating sketch of the intellectual life of one of the greatest sons of France is now available for English readers. Duclaux is a capital Boswell, and possesses himself a genius of his own. Of him Madame Duclaux concludes her biography with these words, in which many will concur : "L'âme la plus modeste, la plus désintéressée, et une des plus justes de ce temps." Throughout the book we are fascinated by the manner in which our guide leads us on through the many-chambered temple of the mind of the great French physicist, chemist, microscopist, and pathologist, who little foresaw that his researches in crystallography would end in his discovery of the means for preventing rabies.

His life work is presented in eight sections, each representing connected steps in the evolution of his mind. Thus we see that the idea that guided Pasteur in his work on fermentation arose from an observation he had made during the study of the rotary power of certain crystals. He was not a physiologist, but he, nevertheless, undertook to study silkworm disease in Southern France, a work that occupied six years, incidentally placing the great questions of contagion and heredity upon an experimental basis. His studies of the cause of fermentation in beer, wine, and vinegar, enabled Pasteur to assert with assurance that the anthrax bacillus was the sole cause of anthrax in the note of April 30, 1877, though the proof which it set forth had been previously made. From this time forward his wonderful insight divined that he possessed a weapon for combating microbic diseases which his predecessors had not. His was the observation that the inoculation into an animal of a filtered culture of the organism of a disease produces symptoms similar, though less profound, than the disease of which that particular organism is the cause. From his triumphs in connexion with anthrax vaccination, in 1881, it was but a short step towards the final goal, the discovery of the prophylaxis of rabies. There is an annotated list of persons mentioned in the book, and several interesting photographs.

An Atlas of the Primary and Cutaneous Lesions of Acquired Syphilis in the Male. By Charles F. White, O.B.E., M.B., and W. Herbert Brown, M.D. With a foreword by Lieutenant-General Sir John Goodwin, K.C.B., C.M.G., D.S.O. With 4 coloured plates and 31 illustrations; p. vii + 32. Price 27s. 6d. net. London: John Bale, Sons and Danielsson, 1920.

Owing to the hold which the pathological aspect of venereal diseases has obtained on the mind both of the profession and of the public, a circumstance which must lead to the lack of support the profession will get from the public when any gaps in knowledge become evident, we cordially welcome a volume dealing almost exclusively with the clinical side. This book is a brief analysis of 19,000 cases, those most typical being illustrated with coloured plates and photographs. As chief attention is given to the primary lesion, this atlas should prove of the greatest value to the beginner, since the surest and quickest way to diagnose a sore is with the naked eye. The photographs are excellent, but the coloured plates are not up to the standard generally reached nowadays.

A Manual of Venereal Diseases for Students. By L. W. Harrison, D.S.O, M.B., Ch.B., M.R.C.P.E. With 333 illustrations in the text and 21 plates; pp. xv + 360. Price 16s. net. London: Henry Frowde, Hodder and Stoughton. Ltd., 1920.

This book will scarcely fulfill its purpose, as students require food for thought or inquiry, and not simply mere statements of current opinion. Greater attention is devoted to the pathological than to the clinical aspect; this is especially noticeable in the pages devoted to venereal diseases in women and children. As the present day treatment is new and is falling far short of our expectations, it would have been wiser to warn the student of the increase of nervous syphilis, and to have instructed him as to how to handle cases of metallic intoxication and those severe recurrences which resist all treatment, and which are now so common. The coloured illustrations do not reach the standard of present-day requirements, and we think that as few instruments as possible should be depicted, as chronic urethritis to-day is more often of instrumental than of gonococcal origin. What the student of to-day mainly requires to know is how the handling of venereal diseases now compares with the treatment in vogue before the salvarsan era. The important clinical work he can pick up in a venereal hospital only.

Orthopædics for Practitioners: an Introduction to the Practical Treatment of the Commoner Deformities. By Paul Bernard Roth, M.B., Ch.B Aberd., F.R.C.S.Eng. With 57 illustrations; pp. xii + 195. Price 10s. 6d. net. London: Edward Arnold, 1920.

This book does not pretend to be a complete treatise or text-book on orthopædic surgery. It represents, however, certain aspects of the treatment of deformities, and expresses the personal opinions of the author. These appear to be lacking neither in decision nor definition, and some of them differ strongly from those held by orthopædic surgeons of great experience. We refer, as examples, to Mr. Roth's sweeping condemnation of the practice at orthopædic hospitals of applying walking instruments in the treatment of congenital club foot, and of the practice of allowing some weight to be borne on the end of the stump in the fitting of artificial limbs. The book is written in a colloquial style, which, however, leaves no doubt as to the author's meaning.

LECTURES ON VENEREAL DISEASES. By LEONARD MYER, F.R.C.S. Pp. 88. Price 6s. net. London: The Albany Press, 1919.

This book is designed, according to the author, to meet the requirements of nurses, midwives and masseuses, and those members of the general public interested in the subject. The subject matter, therefore, is simply written and clearly put, but we think it is too curtailed to be of any real value. A little knowledge is a dangerous thing—in venereal diseases perhaps more than in any others. Treatment is omitted, because by law it must not be undertaken by any person not legally qualified to practise medicine. The book is dedicated to St. Paul's Hospital.

THE X-RAY ATLAS OF THE SYSTEMIC ARTERIES OF THE BODY. By H. C. ORRIN, O.B.E., F.R.C.S.Ed. With 21 plates, pp. 91. Price 12s. 6d. net. London: Baillière, Tindall and Cox, 1920.

It is so difficult, at the present time, to obtain bodies for dissection or for operative-surgery classes that any means by which an accurate knowledge of anatomical details can be obtained is sure of a welcome. More especially is this the case when a visual memory can be cultivated. An X-ray atlas of the arteries of the body should prove of great value, and the present volume, as a first attempt, will be examined with great interest. Unfortunately the "subject" available was only a full-time fœtus, and the author says nothing about the similarities, or comparative relations, of adult vessels. Moreover he has been able to give only six stereoscopic plates and they are necessarily small, yet it is by stereoscopic vision alone that the relations of parts can be appreciated. To read single plates requires no little practice and great clarity of anatomical memory, so that perhaps the most striking feature at a first glance, is the number of unnamed vessels. It will, it seems, require most painstaking individual instruction to enable the teacher to pass on his knowledge to a new student. The author, in times of peace, will certainly be able to produce a larger and better book on similar lines.

DIATHERMY IN MEDICAL AND SURGICAL PRACTICE. By CLAUDE SABERTON, M.D. Illustrated; pp. xii + 138. Price 7s. 6d. net. London: Cassell and Co., Ltd., 1920.

In his preface the author states: "The aim of this little book is to serve as a guide to students and practitioners who wish to master the technique of diathermy, and to appraise its place in the treatment of disease." In point of fact, this little volume is the only English complete monograph that has hitherto been published on this now important method of treatment.

First demonstrated in England by Professor Nagelschmidt of Berlin at St. Bartholomew's Hospital in 1910, the potentialities of diathermy for good were soon recognized, with the result that its employment has been more and more extended for a variety of morbid conditions both medical and surgical. With the exception, however, of Nagelschmidt's "Lehrbuch der Diathermie," published in 1913, the literature of the subject has been restricted to various articles in medical and scientific periodicals, not always accessible, and to sundry chapters in certain books on medical electricity. Accordingly this volume comes at an opportune moment to meet a decided want.

The book is divided into three parts. Part I, Technique, deals with the production of high frequency currents, and describes the various models of diathermic apparatus, the physical properties and physiological effects of diathermic currents, and the methods of application. Part II explains Medical Applications of Diathermy, and, in Part III, Surgical

Diathermy, the author gives his own experiences and quotes largely from those of others. He also touches upon ionization and other methods of treatment which are sometimes combined with diathermy.

The book contains a great deal of useful information, but some of the illustrations are reproduced on too small a scale, and some of the descriptions are slightly lacking in clearness.

At the end of his preface, the author remarks that this therapeutic method "should not be given by unqualified operators," and he utters a note of warning against the administration of electrical treatment of any kind whatever by the untrained. Anyone acquainted with diathermy must be aware that if successful results are to be obtained, strict attention to correct technique must be rigidly observed.

The section devoted to bibliography is very full and will be found extremely useful.

RHEUMATISM AND ARTHRITIS. By RALPH STOCKMAN, M.D. Pp. vii + 132. Price 15s. net. Edinburgh: W. Green and Son, 1920.

The title of this book hardly does justice to its scope. It is really a complete, though concise, treatise on all the medical forms of arthritis—with the exception of gout—as well as on those conditions which have been included vaguely under the term "rheumatism.' Acute rheumatism is dealt with first and then fibrositis in its different manifestations. The author admits the difficulty of constructing any satisfactory classification of the chronic forms of arthritis, but the divisions which he adopts are clinically convenient. A chapter is devoted to chronic non-suppurative arthritis (which he subsequently divides into rheumatoid arthritis and chronic infectious arthritis) and one to arthritis of known causation (scarlatinal rheumatism, syphilitic arthritis, &c.). Chronic osteo-arthritis is described separately and the historical sections of the book are of particular interest and, as might be expected from the author, treatment is dealt with very fully and clearly. The photographic illustrations, both clinical and pathological, are numerous and excellent.

GRANDES ET PETITES OBÉSITÉS, CURE RADICALE. Par Dr. FRANCIS HECKEL. Second edition. With 12 plates and 60 figures. Pp. ii + 537. Price 15 fr. net. Paris: Masson et Cie, 1920.

The second edition of Dr. Francis Heckel's work on obesity has been thoroughly revised and brought up to date since the appearance of the first edition in 1911. Although this volume is smaller than its predecessor, owing to condensation in certain parts, it deals more fully with others, the sections on which have been entirely re-written, notably the chapters devoted to pathology and treatment. These are based upon a study of 4,000 cases. In the first chapter normal and pathological stoutness are defined and contrasted, and the importance of the co-efficient, fat to muscular tissue, in the classification is insisted on. In the next chapter the ætiology of obesity is discussed, and this is followed by sections dealing with the symptoms and complications. The pathogenesis of various forms of excessive stoutness is then considered and leads to a discussion of the evolution, diagnosis, and prognosis. In the chapter on treatment the methods employed in the past are first described and criticized, then the author deals with the means he now adopts for reducing fat in different types of case. An appendix is devoted to the description of characteristic cases and to tables of food materials, &c. The text is illustrated by photographic reproductions of different types of obesity and the effect of treatment upon them.

DR. G. HERSCHELL'S TEXT-BOOK OF INDIGESTION. Revised and rewritten by ADOLPHE ABRAHAMS, O.B.E., M.D.Camb., M.R.C.P.Lond. Fourth edition. With 8 plates; pp. vii + 228. Price 10s. 6d. London: Edward Arnold, 1920.

Dr. George Herschell's Text-book on Indigestion appears to have filled a want, for it passed through three editions, the last of which appeared in 1905. A fourth edition was contemplated, but its publication was delayed and eventually prevented by the death of the author. On taking up Dr. Herschell's work the present author found that such radical changes had taken place in our conception of functional diseases of the alimentary tract, and so much new work had been done, especially on radiographical lines, since the last edition, that it was necessary to re-write the whole book. In doing so he departed somewhat from the idea of former editions, and decided to include organic diseases of the stomach as well as functional disorders. After considering the processes of normal digestion, he discusses the nature and causes of indigestion, and then proceeds to describe the investigation of a case, dealing in turn with the clinical symptoms and history, the physical examination of the patient, intragastric methods and technique, and the examination of the stools. The symptoms, diagnosis, and treatment of acute indigestion. chronic gastritis, gastric and duodenal ulcer, malignant disease of the stomach, dilatation of the stomach, pancreatic indigestion, reflex dyspepsia, dyspeptic symptoms associated with non-gastric organic affections, and nervous indigestion are dealt with in successive chapters. In an appendix an account of the preparation of foods for dyspeptics by the late Dr. Herschell is reproduced substantially unaltered from previous editions.

THE BLIND: THEIR CONDITION AND THE WORK BEING DONE FOR THEM IN THE UNITED STATES. By HARRY BEST, Ph.D. Pp. xxviii + 763. Price $4. New York: The Macmillan Co., 1919.

This book of nearly 800 pages discusses the question of the blind as it is presented in the United States. The subject is divided into seven parts: (1) General condition of the blind, (2) blindness and the possibilities of its prevention, (3) provision for the education of blind children, (4) intellectual provision for the adult blind, (5) material provision for the blind, (6) organizations interested in the blind, (7) conclusions with respect to the work for the blind. From lists shown in three appendices, the United States contains forty-six schools for educating the blind, in institutions, exclusive of nine day schools, fourteen homes for the adult blind, seven homes for blind children, and thirty-nine industrial establishments for the blind. Much attention in America is now being directed to preventive measures against blindness, namely the prevention of purulent conjunctivitis in infancy, the prevention of trachoma, the prevention of loss of sight occurring from injuries and accidents, and from industrial conditions. A special organization, of wide co-operation, has been formed to deal with these questions, and it is engaged in a campaign to educate the public and to secure the necessary measures for improvement. From the evidence he has been able to collect the author is in a position to announce that, taking the situation as a whole, there is little question that headway is being made in the prevention of blindness, and that blindness in the United States is tending to decrease, though perhaps very slowly as yet.

PHYSIOLOGIE NORMALE ET PATHOLOGIQUE DES REINS. Par L. AMBARD. Deuxième edition. Pp. 368. Price 18 fr. net. Paris: Masson et Cie, 1920.

In this work the author almost entirely confines himself to the views of the French school. In dealing with the normal kidney he intentionally omits what he describes as "the classsical physiology of the kidney," and limits himself to an elaborate study of the concentrating powers of the organ. This capacity for concentrating substances which occur in minimal quantities in the blood-stream, and eliminating them in much higher concentrations in the urine, he regards as the most important expression of renal function. After describing methods by which the maximum concentration of urea in the urine can be evoked in man and in the dog, and discussing the significance of the test, he proceeds to describe his well-known constant of urea excretion. He demonstrates the identity of the urea constant with the constants of other substances which are excreted by the kidney without attaining a threshold value in the blood, such as iodine, ammonia and sulphates. Working on the assumption that the urea constant holds also for substances which must attain a certain threshold value in the blood before they can be eliminated by the kidney, he is able to calculate the threshold for these substances in individuals in whom the urea constant has been determined. The thresholds for glucose and chlorides are exhaustively dealt with. It may be pointed out that the fixity of the constant in the individual has recently been questioned by various authors, and that it has been suggested that its variations in the normal are considerable. In the second half of the book he deals with the diseased kidney from the standpoint of function. Cases of nephritis are differentiated into azotæmic, œdematous, and mixed groups. In the œdematous group the excretion of the threshold substances is impaired, while the non-threshold substances are excreted in a normal manner. In the azotæmic group the power of eliminating the non-threshold substances is diminished, and, according to the author, the efficient excretion of the threshold substances is due to a lowered threshold. Such changes in the threshold, if Ambard's theory of the identity of the constant in the two classes of substances is correct, are obviously the only mechanism by which threshold substances could still be apparently normally eliminated, when the power of excreting non-threshold bodies has been largely lost. The book concludes with chapters on the general symptoms of nephritis, prognosis in nephritis, and on the testing of renal function in surgery No attempt is made to correlate functional defects with the findings of morbid anatomy.

A TEXT-BOOK OF OPHTHALMIC OPERATIONS. By HAROLD GRIMSDALE, M.B., F.R.C.S., and ELMORE BREWERTON, F.R.C.S. Second edition. With 129 figures; pp. vii + 438. Price 18s. net. London: Ballière, Tindall and Cox, 1920.

Since the first edition of this work was published, about thirteen years ago, many changes have taken place in the operative treatment of diseases of the eye. It has therefore been the first object of the authors to bring their work up to date, while preserving, as far as possible, the original lines upon which it was first designed. In keeping with this object certain operative procedures, formerly described, have been omitted, while a chapter has been added on operations on the conjunctiva, as well as one on the treatment of retained foreign bodies. Again the developments which have taken place in the operative treatment of glaucoma, during the past few years, have necessitated the chapter on this subject being re-written. The feature of the first edition, namely, diagrammatic illustrations, in preference to photographic reproductions, has been retained throughout. The useful plan has been followed of adding to each of the more important chapters a bibliography of the papers referred to in the text. At present this work is the only one on the subject in our language, thus placing it in a position which will commend it to those for whom it has been compiled.

HENRY QUIN, M.D., PRESIDENT AND FELLOW OF THE KING AND QUEEN'S COLLEGE OF PHYSICIANS IN IRELAND, AND KING'S PROFESSOR OF THE PRACTICE OF PHYSIC (1718-1891). By T. PERCY C. KIRKPATRICK, M.D., M.R.I.A. Illustrated; pp. 66. Price 10s. 6d. net. Dublin: The University Press, 1919.

Henry Quin was the son of Thomas Quin, a Dublin apothecary of some note in the early part of the eighteenth century, who himself is presumed to be a kinsman of Mark Quin, apothecary, who was Lord Mayor of Dublin in 1667. Born in 1717 or 1718, Henry Quin entered Trinity College from a private school in 1733, taking his bachelor's degree in arts four years later. He graduated in medicine in the University of Dublin in 1743, becoming M.D. Padua in 1749. He was elected, after examination, King's Professor of Physic in the medical school of Trinity College in the same year, and was soon afterwards elected a Fellow of the King and Queen's College of Physicians in Ireland. He embarked upon a fashionable practice in Dublin, and married in 1753 Miss Anne Monck, whose family was "considered as belonging to the most exclusive social circle." In 1762 he purchased the lease of No. 101, St. Stephen's Green, which is now the "St. Patrick's Nurses' Home." He was elected Registrar of the College of Physicians in 1754, and in 1757 he was chosen Censor. He was at this time Inspector of the Apothecaries' Shops. After only four years as Fellow, he was first elected President on St. Luke's Day, 1758, and he was subsequently chosen six times to fill that office. Henry Quin was an accomplished musician, and was also greatly interested in the collection of cameos and intaglios, and afterwards taught and encouraged James Tassie in the art of making pastes. He amassed a considerable fortune, and in 1771 he purchased the property of Borleigh, in Co. Wexford. He died in 1791, leaving three sons and three daughters. There is much in the book to interest the antiquarian and historian.

FRACTURES, COMPOUND FRACTURES, DISLOCATIONS AND THEIR TREATMENT: WITH A SECTION ON AMPUTATIONS AND ARTIFCIAL LIMBS. By JOHN A. C. MACEWEN, M.B., C.M.; B.Sc. With 43 plates and 61 diagrams; pp. xii + 285. Price 12s. 6d. net. Glasgow; Maclehose, Jackson and Co., 1919.

In the first section of this book the author deals with fractures in general—their varieties, the natural processes of repair and complications. The next section is devoted to individual fractures—those of the skull, vertebræ, face, clavicle, ribs, upper limb, and lower limb, being considered in turn and their treatment described and clearly illustrated. Dislocations are next dealt with in order corresponding to that of the different fractures; congenital dislocations being briefly noticed. Pathological dislocations are also considered. The last section deals with the treatment of shock and the choice of anæsthetic and the question of operating in cases in which shock is present. Amputations and artificial limbs are described with many illustrations, including one of the author's machine for making wooden sockets from plaster casts of stumps. The plates are taken from a collection of photographs by Sir Wm. Macewen and the author, and, like the diagrams, are all very clear. The experience of the war as applied to fractures is fully reflected in this work.

PHYSIOLOGICAL PRINCIPLES IN TREATMENT. By W. LANGDON BROWN, M.A., M.D.Cantab., F.R.C.P. Fourth edition. Pp. vii + 427. Price 8s. 6d. net. London: Baillière, Tindall aud Cox, 1919.

The new edition of this popular book has been revised in the light of the latest knowledge, and the chapters on diabetes and acid intoxications have been almost entirely re-written, but there has been no great addition of new matter.

PROSTATECTOMIE SOUS ANESTHÉSIE LOCALE. Par VICTOR PAUCHET. COMPLICATIONS, SOINS CONSÉCUTIFS. Par R. DE BUTLER D'ORMOND. With 20 illustrations; pp. 25. Price 5 fr. Paris: Maloine, 1919.

In this book a short and very lucid description is given of the author's method of operation in cases of enlarged prostate. He employs local or regional anæsthesia, and the first part of the book deals with this. The special points brought out are first the determination of the renal function by the methylene blue test, the removal of sufficient mucous membrane of the floor of the bladder to avoid an obstructing flap after the operation and the employment of gauze packing and Carrel's tubes for insertion in the cavity left after removal of the prostate in septic cases. The illustrations are excellent, and the book can be confidently recommended to those interested in the subject.

TRAITEMENT CHIRURGICAL DES AFFECTIONS DE L'ESTOMAC. Par VICTOR PAUCHET. With 90 illustrations; pp. 72. Price 12 fr. Paris: Maloine, 1919.

This book, or more properly atlas, for barely one quarter of the space is given to letterpress, deals essentially with the operative surgery of gastric affections. In it the author describes, with the aid of most admirable drawings, his own particular technique and methods of dealing with gastric and duodenal ulcer and gastric carcinoma. He is a firm believer in local and regional anæsthesia for all gastric operations, and gives a clear description of the technique he employs. His method of obtaining access to the posterior wall of the stomach by reflecting upwards the whole of the great omentum from the colon is one which commends itself, and is perhaps the feature of the book. This book can be strongly recommended to all who are interested in gastric surgery, for in its way it is a masterpiece of terse and graphic exposition.

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